

# NEOVASCULAR GLAUCOMA, A LATE COMPLICATION OF CENTRAL RETINAL VEIN OCCLUSION

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**Keywords:** central retinal vein occlusion (CRVO), neovascular glaucoma (NVG), macular edema, Avastin®(Bevacizumab), Triamcetonolone acetamid

**Abstract:** The purpose of this paper is to present the evolution of central retinal vein occlusion (CRVO) in a young patient with risk factors (thrombophilia and hypercholesterolemia) and the therapeutic management of the condition. The complications of the CRVO are macular edema and neovascular glaucoma. Treatment of the complications is medical, intravitreal injection with vascular endothelial growth factors (VEGF) inhibitors (Avastin) and steroidal anti inflammatory drugs (triamcinolone acenonide), and surgical, trabeculectomy with iridectomy and postoperative subconjunctival 5-fluorouracil (5-FU) injections. The management of CRVO summarises two major objectives, identifying risk factors with their management and recognising and treating sight threatening complications that can be encountered. The particularity of this case is the patients' early age, NVG being a late secondary complication of CRVO in the elderly.

## INTRODUCTION

Neovascular glaucoma, as a typical secondary glaucoma, is due to ocular or (earlier) systemic diseases characterised by ischemia (retinal hypoxia) with the release of proangiogenic factors.(1) It is one of the most devastating forms of glaucoma, with loss of sight, and in lack of rapid and aggressive treatment, it can lead to eye loss. Frequent causes of NVG are: occlusion of the central retinal vein, proliferative diabetic retinopathy and carotid occlusion definition.(2) Less frequent causes are: retinal venous occlusion, central retinal artery occlusion (CRAO), intraocular tumours, endofthalmitis, premature retinopathy, sickle cell anemia, radiation retinopathy, Eales' disease, Coats' disease, ocular ischemic syndrome, Takayasu's disease, Horton's disease, and trauma.(3) Clinically, NVG can be divided into the following 2 stages that generally follow each other in progression, and the early stage is subdivided further into rubeosis iridis secondary open-angle glaucoma, the advanced stage comprising of closed-angle glaucoma:

Early stage, (rubeosis iridis) biomicroscopy exam:

- Presence of tiny, neovascular, dilated capillary tufts at the pupillary margin,
- Poorly reactive pupil,
- Iris and angle neovascularisation.(4)

Early stage, open-angle glaucoma biomicroscopy exam:

- Proliferation of neovascular tissue over the angle,
- Ectropion uvea.(5)

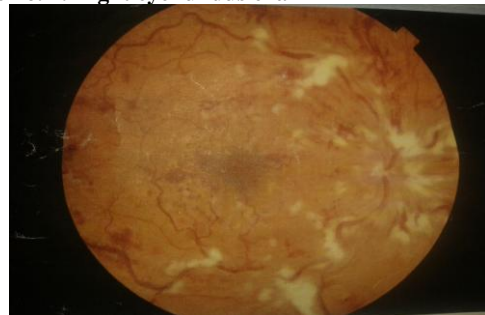
Advanced stage, angle-closure glaucoma biomicroscopy exam: conjunctival injection, corneal edema, possibly hyphema, synechial angle closure, severe rubeosis, fixed mydriasis, retinal and nerve neovascularisation and/or hemorrhage, optic nerve cupping.(6) From the speciality literature, it is known that NVG is a late complication of CRVO in the elderly or those suffering from diabetes mellitus,

but apparently rare cases that affect the young with systemic pathologies exist.(7,12,13,14)

## CASE REPORT

Patient C.C. a 35-year old urban taxi driver, presented to the emergency room of the Drobeta Turnu Severin Hospital and after triage, he was sent to the ophthalmology ward. He presented painless, acute loss of visual acuity. The ophthalmological exam showed: both eye visual acuity (VA) , VA=0.1 fc, right eye intraocular pressure (RIOP) of 16 mmHg and left eye intraocular pressure (LIOP) of 17 mmHg, the presence of the juvenile arch (cardiovascular disease risk factor). The Right eye fundus exam showed papilledema, dot hemorrhage, numerous hard exudates, diffuse edema especially central edema, sinuous turgescient veins. Left eye fundus exam was normal.

Figure no. 1. Right eye fundus exam



The diagnosis was established - central retinal vein occlusion, and the following treatment was administered: acenocumarol three tablets/day, 75 mg of aspirin one tablet/day, Enoxiparine 6000 IU every 12 hours for three days, ceftriaxone two grams and dexktoprofenum one tablet per day for seven days.

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## CLINICAL ASPECTS

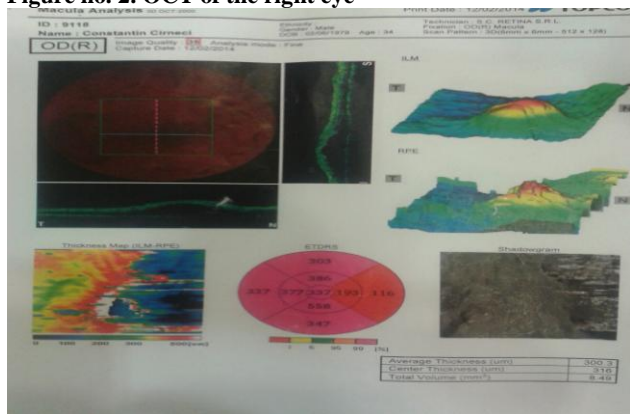
**Table no. 1. Differential diagnosis**

Retinal ischemic syndrome	<ul style="list-style-type: none"> <li>(-) papilledema</li> <li>(-) juxtapapillary hemorrhage</li> </ul>
Hypertensive retinopathy (15,16,17)	<ul style="list-style-type: none"> <li>bilateral modifications</li> <li>insignificant visual loss</li> </ul>
Diabetic retinopathy	<ul style="list-style-type: none"> <li>bilateral changes</li> <li>dot or spot hemorrhage</li> <li>microaneurysm</li> <li>hard exudates</li> </ul>

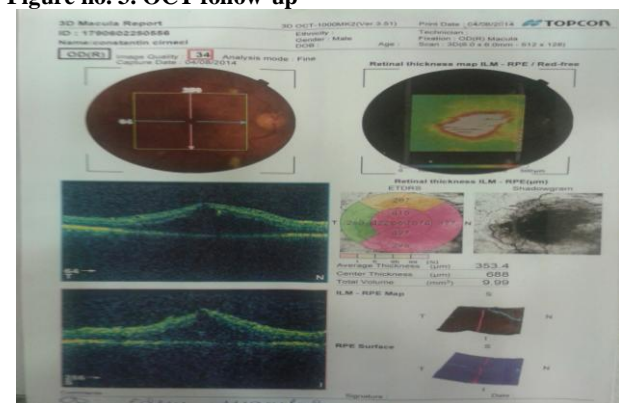
*Patient history:*

A personal pathological history of biliary dyskinesia and hiatal hernia, gastroesophageal reflux disease and dyslipidemia, with no family history of other diseases.(8,9,10) A series of clinical laboratory and imagistic tests were performed: Complete blood count with biochemistry that was normal. The diagnosis and the young age of the patient meant it mandatory to search for other pathologies, such as thrombophilia (complete blood count, C reactive protein, S protein, antithrombin III) antiphospholipid syndrome (Ro antibodies, La immunoglobulin G and M) lipid panel (cholesterol and lipid fractions). After six days, the patient presented himself at the Emergency Clinical Ophthalmology Hospital in Bucharest with right eye Visual Acuity (RVA)= 0,05 fc, left eye Visual Acuity (LVA)=1 fc, RIOP=18 mmhg, LIOP=17 mmhg. Positive diagnostic of sechelelar CRVO and macular edema was established (figure no. 2) and treatment with Avastin® and triamcetonol acetonid was initiated. Re-evaluation at the Emergency Clinical Ophthalmology Hospital in Bucharest with ophthalmological exam showed RVA=1/2 fc, LVA= 1fc, RIOP=18 mmHg, LIOP=17 mmHg. The diagnosis and treatment remain the same (figure no. 3).

**Figure no. 2. OCT of the right eye**



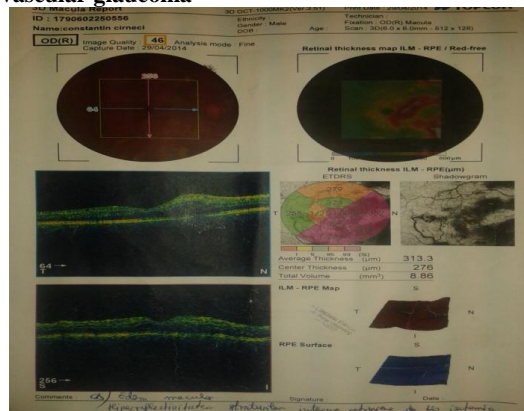
**Figure no. 3. OCT follow-up**



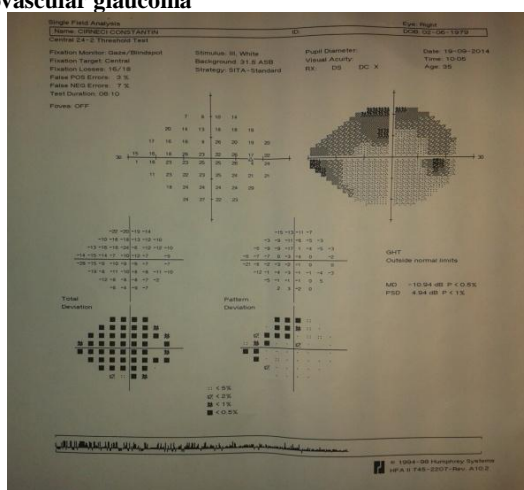
A series of two visits followed on 04.08.2014 and

18.09.2014. On the first one, the diagnosis was sequelae of CRVO and macular edema with RVA=1/2 fc, LVA=1 fc, RIOP=18 mmHg (COSOPT®), LIOP=17 mmHg, but on the second visit, RVA=0.8 fc, LVA=1 fc, RIOP=40 mmHg (COSOPT®+ SifiTan®), LIOP=17 mmHg and the diagnosis was open angle with the absence of neovascularisation (figures no. 4, 5).

**Figure no. 4. Positive diagnosis: sequelae of CRVO and Neovascular glaucoma**



**Figure no. 5. Positive diagnosis: sequelae of CRVO and Neovascular glaucoma**



Administered treatment: scleral flap trabeculectomy, iridectomy at 12 o'clock position, 5 fluorouracil subconjunctival injection, with a favourable post-op evolution, and with RVA=1/2, RIOP - digitally hypotone, after the first week follow-up.(11)

### CONCLUSIONS

The management of CRVO has two urgent objectives: risk factors identification and their treatment (hypercholesterolemia, thrombophilia), and the identification and treatment of the complications that can impair sight (macular edema, neovascular glaucoma).

*Case particularity*

Young, 35-year-old male, with thrombophilia, CRVO and neovascular glaucoma, and the irreversible loss of sight.

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## CLINICAL ASPECTS

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