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Review Article

Clinical-Therapeutic Orientation in Retinal Venous Obstruction

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Abstract

Retinal vein occlusion (RVO) is a retinal vascular disease that can affect the central retinal vein in central retinal vein occlusion (CRVO) or branch retinal vein occlusion (BRVO), which causes decreased vision (the second leading cause of blindness after diabetic retinopathy).

CRVO is accompanied by retinomacular edema and retinal / peripapillary / iris neovascularization that cause serious complications: absolute neovascular secondary glaucoma, vitreous hemorrhage, retinal traction detachment, possibly present in both forms of CRVO.

Branch retinal vein occlusion (BRVO) is often asymptomatic and can be diagnosed accidentally or by retinal control, and is 5 times more common than CRVO.

CRVO prophylaxis is done by identifying and appropriate treatment of risk factors (multiple): hypertension, diabetes, smoking, obesity, Primary Open Angle Glaucoma (POAG), hypercoagulability.

RVO treatment, the non-ischemic form, requires the treatment of macular edema with: intravitreal AntiVEGF - Ranibizumab, Aflibercept, Bevacizumab, repeatedly, cortisone therapy with intravitreal Triamcinolone or Dexamethasone implant, focal / grid laser photocoagulation and / or panretinal photocoagulation.

In all cases of RVO, the non-ischemic form, the following are required: clinical surveillance, fluorescein angiography (FA), OCT for immediate detection of progression to the ischemic form.

The treatment of RVO, ischemic form, is the treatment of macular edema with repeated intravitreal antiVEGF treatment, corticosteroids, focal laser photocoagulation, grid, pan-photocoagulation indicated in the treatment of exudative ischemic areas with neovessels proliferation. Panretinal photocoagulation (PRP) is an effective treatment for iris peripapillary retinal neovascularization and secondary complications.

The current prognosis of RVO is improved by regular examination of OCT-SD, antiVEGF medication, intravitreal cortisone and laser photocoagulation that provide prophylactic and curative treatment of RVO and complications: vitreous hemorrhage, neovascular glaucoma, retinal traction detachment.

Key words: branch retinal vein occlusion; cystoid macular edema; Bevacizumab; retinal vein occlusion; vascular endothelial growth factor; neovascular glaucoma NVG

Introduction

- Retinal vein occlusion - RVO - caused by venous thrombotic blockage (clot, atheroma fragment), or by compression of the artery vein at the intersection of the venous artery, is a relatively common retinal vascular disease.
- The prognosis of RVO has now changed with the introduction of new diagnostic methods - spectral domain optical coherence tomography - OCT - SD, and the use of new drugs administered intravenously, antiVEGF (vascular endothelial grow factor inhibitors), steroid implants [8].

The risk factors should be identified and their treatment can ensure RVO prevention:

- age, sex, obesity

- cardiovascular diseases: hypertension 50-60%, atherosclerosis, carotid stenosis
- biological factors: diabetes 15-34%, hyperlipidemia, hyperviscosity (multiple myeloma, polycythemia), hypercoagulability
- ocular factors - 25% chronic open-angle glaucoma
- inflammatory factors: retinal vasculitis - Behcet, sarcoidosis, SLE, syphilis, autoimmune diseases - more common in young people
- other factors: smoking, oral contraceptives, diuretics, sympathomimetics
- other diseases: cavernous sinus thrombosis, Coats disease, von Hippel Lindau, Eales, retinal phlebitis
- vascular genetics

RVO physiopathology

Retinal venous occlusion causes venous blood to stagnate above obstructive blockage (venous stasis) with increased pressure at this level and altered endovascular structure, endothelial proliferation and capillary occlusion in the secondary ischemic area after obstruction and blood hypercoagulability.

- venous retinal ischemia is a factor generating edematous maculopathy and proliferative neovascularization with its complications - vitreous hemorrhage, retinal traction detachment, absolute neovascular secondary glaucoma.[5]
- *the evolution of RVO depends on the location and extent of retinal vascular ischemia with its consequences- edematous maculopathy and neovascularization.*

RVO clinical overview

According to localization, the retinal venous obstruction may be:

- Central retinal vein occlusion - CRVO

- Branch retinal vein occlusion – BRVO, which is often asymptomatic and its diagnosis can be accidental or by routine check-up / both sometimes late.

  - BRVO is 5 times more common than CRVO.

In BRVO, the vision loss is NOT high, so the ocular disease can be diagnosed late by complications.

Decreased vision in BRVO is present in both forms of the disease – CRVO and RVO - but to varying degrees

- vision loss is progressive for days, weeks, small, medium, sometimes severe

RVO has two clinical forms:

- NON-ISCHEMIC–EDEMATOUS – perfused – 75-80% - Retinopathy of venous stasis, very rarely with neovessels, but it is possible to convert the non-ischemic form to the ischemic form - 16%

- ISCHEMIC – non-perfused hemorrhagic with retinal edema and peripapillary retinal iris neovascularisation. (1,3,4)

Clinical forms in RVO

RVO presents 2 clinical forms: non-ischemic CRVO and ischemică CRVO and BRVO

NON-ISCHEMIC CENTRAL RETINAL VEIN OCCLUSION

NON-ISCHEMC CRVO

- slight visual impairment, sometimes not perceived by the patient (or neglected)

-minimal deficiencies of the visual field with blind spot enlargement, central scotoma

- relative afferent pupillary defect (RAPD) - absent

-Early Eye Fundus

- dilated, tortuous veins
- normal arteries
- superficial hemorrhages, up to the periphery of the retina, deep hemorrhages rarely
- diffuse papillary and retinal edema (rarely), without macular edema
- *Neovessels - absent*

-Late Eye Fundus

- retinal hemorrhage, partially resorbed
- *possible CME*
- vein ensheathing
- *cilio-retinal collaterals around the optical disc*

- favourable evolution, rarely complications

-functional and organic recovery in 50% of cases; complete in 6-12 months

- interdisciplinary monitoring of the patient because *conversion to ischemic form is possible*

- preserved VA

Ischemic Crvo

- severely decreased VA in 60% of the cases

-Visual field – central deficits, absolute scotoma

-RAPD – present

- Iris neovessels - 33% complicated by ***secondary neovascular glaucoma***

-Eye Fundus – the same signs as in non-ischemic RVO but more pronounced

-Early Eye Fundus

- tortuous retinal veins, less dilated
- filiform arteries
- numerous massive, burning, round retinal hemorrhages
- *dense, numerous, confluent "cotton wool exudates" - major sign of significant retinal ischemia*
- papillary edema with peripapillary exudates and hemorrhages
- macular edema
- iris and retinal neovessels – rubeozis iridis

-Late Eye Fundus

- vein ensheathing
- collaterals surrounding the optical disc
- *extensive disc and peripheral neovascularization*
- *chronic macular edema, CME*
- *evolution with sometimes serious complications*, accompanied by irreversible vision loss due to macular edema and retinochoroidal and iris neovascularization

- -*complications* - vitreous hemorrhage 60% due to proliferative retinopathy and neovascularization, retinal traction detachment, neovascular glaucoma
- reserved prognosis by macular ischemia and extensive neovascularization
- evolution - partial resorption of retinal papillary edema
- macular lesions secondary to edema with macular epiretinal gliosis and pigmentation disorders
- complications of retinal and/or disc neovascularization with proliferative retinopathy and diabetic retinopathy
- Signs of gravity in CRVO [5]
- VA – n.d.
- Eye Fundus – interpapilllary-macular numerous, confluent cotton nodules
- deep bleeding (sign of major ischemia)
- *macular edema, cystoid macular edema (CME)*
- *extended retinal neovascularization*
- rubeosis iridis at the edge of the pupil and the iridocorneal angle with absolute neovascular secondary glaucoma

Branch Retinal Vein Occlusion Brvo

It affects one of the 4 retinal vein branches at the level of the arteriovenous junctions by compressing the venous branch by the thickened and rigid artery inside the common sheath.[4]

  - if the venous obstruction is on a branch in the vicinity of the optical disc, retinal ischemia comprises the entire vascularized retinal quadrant of the obstructed vein
  - if the obstruction is at the periphery of the retina on a small venous branch, the symptoms are minimal
  - branch retinal vein obstruction is more frequently located in the upper temporal sector 63%.
- Symptoms of branch retinal vein obstruction depend on the location of the obstruction and the presence of vision loss only when the macular area is involved.
- Clinical signs:
  - there are changes of the Eye Fundus located in the quadrant, in the obstructed venous territory: superficial flame-shaped hemorrhages, deep, cottony exudates, rarely CME, retinal papillary edema in the sector with preservation of the median raphe.
  - late (6-12 months) - venous sclerosis, collateral circulation, hard exudates, residual hemorrhages, microaneurysms
- Complications
  - Chronic macular edema, CME (rarely)
  - Retinal neovascularization (one in 3 cases)
  - Macular gliosis
  - Changes to the EPR

Hemiretinal Vein Occlusion (Hrvo)

- it occurs at the level of the arterio / venous crossings located in the vicinity of the optical disc
- altitudinal amputation of the visual field
- variable decrease of the visual acuity
- eye fundus changes in the upper or lower hemiretina corresponding to the site of the obstruction
- prognosis - dependent on macular ischemia and neovascularization

Venous Stasis Retinopathy

- There are retinal vascular obstructions associated with occlusive disorders of the carotid artery
- Risk factors – diabetes, hypertension, carotid diseases, oral contraceptives (estrogenic)
  …-young people - 30-40 years old
- Symptoms
- Visual acuity decreases if macula is damaged
- Visual field: scotoma, blind spot enlargement
- Eye Fundus
- Dilated, tortuous retinal veins, superficial retinal hemorrhages
- Papillary edema, sometimes discrete macular

Papillophlebitis

- It is CRVO in young people under 40 years
- Onset with sudden, significant decrease in vision, more pronounced on waking (2/10)
- It is caused by a congenital anomaly of the optical disc that causes central retinal vein (CRV) compression in the lamina cribosa
- Clinically - moderate CRVO with papillary edema
- Prognosis - good for young people.

Complementary examinations in RVO

- Laboratory tests: glycemia, glycosylated Hb, CRP, urea, electrophoresis, lipid profile, coagulability check, blood count, Leiden factor, clinical tests according to associated pathology (1)

- FA identifies areas of unperfused capillaries, extent and presence of macular ischemia

- ERG - reduced b-wave amplitude is associated with increased risk of ischemia

- OCT (optical coherence tomography) - monitor of macular and intraretinal edema (determining the thickness of the retina) and by regular examination, it highlights the evolution of edema under treatment

- complete clinical evaluation of cardiovascular disease, diabetes

Treatment objectives in RVO

Reduction of retinal edema and macular edema

- the incidence of macular edema is more frequent after venous branch obstruction

- macular edema is individualized in OCT-SD, an extremely important investigation in the management of RVO for its identification and quantification, but also for the detection of the conversion of venous obstruction from non-ischemic to ischemic form [5,6].

The treatment used for this purpose is:

- ***antiangiogenic agents*** - antivascular endothelial growth factor - antiVEGF: ranibizumab, bevacizumab, aflibercept injected intravitreally

  - reduce / stop the development of neovessels favoured by postocclusive retinal ischemia and prevent the formation of macular edema
  - prevent the repetition of RVO in 9/10 cases

- ***corticosteroids*** - intravitreal Triamcinolone and Dexamethasone biodegradable implant with slow diffusion of corticosteroid 3-4 months

- have anti-inflammatory effect with reduced macular edema, but also ocular side effects - cataracts, glaucoma - and systemic effects [7,8].

- ***photocoagulation laser (2)***
  - photocoagulation laser in the treatment of RVO destroys ischemic territories, fights and prevents the preretinal, prepapillary and iris neovascular proliferation in ischemic CRVO. Photocoagulation laser in the treatment of CRVO (depending on FA) improves the visual prognosis and is an effective prophylactic and curative treatment of complications of ischemic capillarophaty disease
  - *Focal laser photocoagulation* is a useful procedure in the treatment of retinal macular edema and retinal neovascularization, but is less effective than antiVEGF or dexamethasone [2]
  - it is indicated in branch retinal vein obstruction no later than 3 months complicated with persistent macular edema
  - it allows resorption of cystoid macular edema (treatment after 3 months because there may be spontaneous resorption)
  - *Laser grid photocoagulation* is indicated in CRVO when VA <20/40 and macular edema is persistent, but also in branch obstruction with macular edema.
    - combined laser grid treatment with antiVEGF treatment increases therapeutic efficacy
  - *Panretinal photocoagulation* is used to limit the growth of neovessels and to reduce the risk of vision loss
    - Panretinal photocoagulation reduces retinal ischemia, may promote resolution of macular edema, and may sometimes reduce the need for antiVEGF therapy
    - Panretinal photocoagulation indicated in ischemic CRVO requires extensive treatment in the periphery of the retina (without touching the macula) in the entire area of the unperfused retina
    - if iris neovessels develop secondarily due to obstruction, panretinal photocoagulation should be performed quickly to prevent neovascular glaucoma (NVG)
    - for severe forms of RVO (NVO) the combination of antiVEGF with panretinal photocoagulation is beneficial
    - **Reducing the risk to repeat RVO in the same eye or congener through:**

  - treatment of risk factors: antihypertensive treatment, control of diabetes, treatment of blood dyscrasia, diagnosis and treatment of glaucoma
  - correct and timely treatment of macular edema (major cause of vision loss) and treatment of neovascularization

- **Treatment of complications:** persistent macular edema, ischemic maculopathy, neovascularization, neovascular glaucoma, vitreous hemorrhage, retinal traction detachment
  - *laser*, focal, grid, *photocoagulation*, pan-photocoagulation to prevent the proliferation of neovessels and to prevent the aggravation of the disease. (2)
- Pan-photocoagulation reduces retinal ischemia and promotes the resorption of edema.
  - *Intravitreal injection of corticosteroids triamcinolone* or dexamethasone implant in vitrectomized or pseudophakic patients to combat the inflammatory component and to reduce macular edema.
- can be used when an effective response to antiVEGF treatment is not obtained
  - efficient antiVEGF treatment for reducing intraretinal edema and improving vision: Bevacizumab (Avastin), Ranibizumab (Lucentis), Aflibercept (VEGF - Trap-Eylea)
    - antiVEGF treatment at 4 weeks blocks the growth of neovessels and prevents the formation of macular edema
    - stabilizes VA
    - requires FA, OCT control to assess the evolution of the disease under treatment
    - the therapeutic associations, depending on the clinical aspect, have a higher therapeutic efficacy compared to monotherapy; can be used when an effective response to monotherapy is NOT obtained [6].

Medical treatment in RVO

- ***Prophylactic treatment*** of diseases associated with hypertension, diabetes, atherosclerosis, blood diseases
- ***Systemic medical treatment*** with interdisciplinary control for the detection of RVO-generating pathology with the control and treatment of underlying disease and risk factors.
- ***Ophthalmic treatment***
  - treatment of primary open-angle glaucoma
  - prevention (with early detection) of NVG (100 days) by systematic examination with slit lamp and gonioscopy to highlight neovessels
  - emergency retinal panophotocoagulation in rubeosis iridis and angle neovessels.
- ***Anticoagulants*** – they are NOT effective and are NOT indicated unless very rarely in coagulation abnormalities.
- ***Fibrinolytics*** – have been used by systemic or direct injection into the ophthalmic artery but the approach is difficult and there is a risk of complications - NOT used in current practice.
- ***Isovolemic hemodilution*** - with a reduction in hematocrit to 30-35%, decreases plasma viscosity and can improve microcirculation and retinal perfusion; but side effects may occur (fainting, shortness of breath). It has questionable effectiveness.
- ***Platelet anticoagulants*** - have NOT been shown to be effective.
- ***Tissue plasminogen activator*** - *tPA*
  - converts plasminogen to plasma and destabilizes intravascular thrombi
  - reduces the size of the clot in the occluded vessel, with the dislocation of the thrombus and the repermeabilization of the occluded vein[1]
  - it is indicated in severe forms of systemic CRVO, intravitreally, or by endovascular cannula in the retinal vein occluded after vitrectomy in pars plana (risk of vitreous hemorrhage)

Intravitreal anti-VEGF agents

- *Bevacizumab* – Avastin – is a human monoclonal antibody with action against VEGF isomers (stimulators of endothelial proliferation with the appearance of neovessels, increased capillary permeability and edema)
  - intravitreal administration reduces macular edema after 7-10 days (OCT documented) and improves visual function

  - repeated intravitreal dose of 1.25 mg (0.05 ml) every 1 month
  - indicated in: NVG, macular edema with edema reduction and VA amelioration [1,5]
- *Ranibizumab* – Lucentis – is a non-selective VEGF inhibitor
  - Indicated for the treatment of macular edema associated with RVO in a dose of 0.5 mg in 0.05 ml
  - Aflibercept - VEGF Trap, Eylea - administered intravenously binds multiple isoforms of VEGF and placental growth factor - PIGF

- ***Corticosteroids***
  - reduce the mediators of inflammation in the production of edema - prostaglandins and leukotrienes
  - reduce capillary permeability and inhibit the expression of VEGF genes in their metabolic field
  - intravitreal triamcinolone acetonide improves VA by reducing macular edema; after 7-10 days and lasts 3-6 months (increased risk of intraocular pressure); possible local side effects: retinal detachment, cataracts, endophthalmitis

Laser photocoagulation

Laser photocoagulation effects are produced by:

- selective destruction of the external retina and partially of the photoreceptors with increasing oxygen diffusion in the choroidal vessels in the internal retina

- reduction of the production of neovascular factors - VEGF that improves hypoxia

- reduction of neovascularization and amelioration of retinal ischemia with decrease in cytokines that favour the regression of neovessels [9,2]

Laser photocoagulation is used to treat retinal macular edema and retinal neovascularization.

Laser photocoagulation is guided by FA exploration.

Laser photocoagulation treatment can limit retinal edema, can promote its extension and by scars between EP and neuroepithelium it limits serous detachment of the neuroepithelium [11].

Laser photocoagulation in CRVO is focal, grid, panretinal.

- Focal laser photocoagulation is applied in the area of edema and promotes its resorption
  - can be used in CRVO secondary edema, but is less effective than antiVEGF medication or dexamethasone implant [10]
  - is indicated in branch retinal vein obstruction no more than 3 months complicated by persistent macular edema

- Laser grid photocoagulation

- Panretinal photocoagulation (scatter PRP) destroys ischemic territories and prevents preretinal, prepapillary and iris neovascularization in ischemic CRVO.
  - Panretinal photocoagulation allows regression of cystoid macular edema and stabilizes VA, but should NOT be performed before three months as spontaneous regression of CME is possible during this time.

Treatment recommendations in CRVO / RVO:

- Antiplatelet agents: they do not prevent neovascularization, do not improve vision
- Fibrinolytics limited indication with intravenous tPA injection and concomitant heparinization in the early stage – not indicated
- Isovolemic hemodilution could prevent neovascularization, would improve visual acuity (VA), have questionable indication
- intravitreal antiVEGF optimal, promising treatment, improves VA and it is a reserve therapeutic option in case of unfavourable evolution
- Intravitreal steroids can improve vision, can increase intraocular pressure, indicated in patients with macular edema, not superior to photocoagulation treatment, triamcinolone can improve VA
- Grid photocoagulation In CRVO with evolution over time and neovessels
  - indicated in macular ischemia
- Panretinal photocoagulation
  - Prophylactic treatment in patients without neovascularization – debatable
  - Treatment in patients with neovascularization to limit secondary complications
  - Only if the retina or optical disc has neovessels
- Pars plana vitrectomy Not routinely recommended

Surgical treatment in RVO aims at the treatment of venous occlusion and secondary macular edema [3].

- Endovascular retinal surgery *with injection of tissue plasminogen activator.*

- *Radial optic neurotomy* by incision in the nasal part of the optical disc radially and parallelly to the nerve fibers - partially controversial, can be performed in selected cases [12].

- *Chorioretinal venous anastomoses* - by creating a shunt between a retinal and choroidal vein (bypassing the occluded vein), (with the improvement of retinal venous drainage), with the help of laser or by vitreoretinal surgery [13,14]

- Yag laser transluminal embolization [14]

- Adventiceal dissection at the arteriovenous junction - *Arteriovenous sheatotomy* with posterior vitrectomy.

- *Posterior vitrectomy* with or without peeling of the inner limiting membrane would rapidly resolve macular edema; could be beneficial by improving retinal oxygenation and removing vitreous retinal traction

Therapeutic efficacy in various clinical forms of RVO

CRVO treatment, non-ischemic form[1,9]

- Anticoagulants - are not effective

- Fibrinolytics in the ophthalmic artery difficult to apply with risks and complications (Not used)

- Isovolemic hemodilution in the first months after the onset of the disease with decreased blood viscosity and improved retinal circulation (controversial)
  - May be indicated in CRVO without extensive peripheral ischemia, in the absence of complications

- **Macular edema treatment in CRVO**
  - Intravitreal antiVEGF[9]: Ranibizumab (lucentis) – 0,5 mg, Aflibercept (Eylea) 2 mg, Bevacizumab (Avastin) – 1,25 mg monthly for the first 6 months

- Corticosteroid therapy: Intravitreal Triamcinolone 1-4 mg, Dexamethasone (Ozurdex) implant– 0,7 mg
- Focal/grid laser photocoagulation

➢ Spot 50-100 µm when macular edema is > 3 months and VA < 20/40
- Panretinal laser photocoagulation in quadrat spot 500 µm when there is

➢ Rubeosis iridis > 2 clock hours of neovascularization on the iris or angle; retinal and optical disc neovascularization or neovascular glaucoma is developed [2] .

➢ *In all* cases of RVO, the non-ischemic form, clinical surveillance and FA, OCT *are required* for several *months, for the immediate detection* of the evolution to the ischemic form [1,5].

CRVO treatment, ischemic form[1,9]

- Partially benefits from the treatment of non-ischemic form of CRVO
  - medical treatment is ineffective
  - hemodilution is less effective
- **Treatment of macular edema in CRVO**

- *intravitreal antiVEGF*: Ranibizumab (Lucentis) – 0,5 mg, Aflibercept (Eylea) – 2 mg, Bevacizumab (Avastin) – 1,25 mg monthly for 6 months. *Intravitreal injection with repeated antiVEGF agents, is the basic treatment of macular edema in CRVO with VA <6/10.*

- *corticosteroids:* intravitreal triamcinolone acetonide - 4 mg, Biodegradable dexamethasone implant - Ozurdex

- *Laser photocoagulation*

➢ Focal - decreases macular edema but it does NOT improve VA

➢ Panphotocoagulation - spot 50 µm when it is present: rubeosis≥2 clock hours neovascularization on *iris or angle, retinal/disc neovascularization, neovascular glaucoma is present.*

- Laser photocoagulation is indicated in the treatment of exudative ischemic areas with proliferation of neovessels [10].

- photocoagulatory *treatment* does NOT restore vision, but prevents NVG
- *laser treatment* should be preceded by *intravitreal injection* with antiVEGF *and allow the regression* of neovessels and *retinal edema.*

➢ *Focal photocoagulation* - may be useful in some macular edema and is indicated in branch retinal vein obstruction more than 3 months, complicated by persistent macular edema (with OCT evaluation).

➢ *Grid photocoagulation* as the first therapeutic line is NOT indicated and does NOT help the beneficial change of VA, but reduces macular edema
- The current indication for grid photocoagulation is represented by Non- or partially responsive patients after repeated injections with antiVEGF

➢ *Panretinal photocoagulation* is indicated in neovascularization of the retina and anterior segment in areas of nonperfusion extended at FA, when panretinal photocoagulation should be initiated rapidly to reduce intravitreal hemorrhage and prevent neovascular glaucoma.
- Panretinal photocoagulation is indicated as a matter of urgency if NVG has been installed.
- Panretinal photocoagulation should be performed after the occurrence of neovascularization.
- Panretinal photocoagulation does not appear to be useful for prophylactic purposes.
- Panretinal photocoagulation has a beneficial effect combined with antiVEGF medication in forms with significant neovascularization and GNV.

• Pascal laser photocoagulation through numerous impacts, in fractions of a second, with short duration of pulses with high power through cumulative energy induces harmless thermal effect, decreases treatment time, improves patient comfort.

• The prognosis of CRVO is clearly improved by laser photocoagulation indicated according to FA, as an effective treatment of complications of ischemic capillary disease.

• *Laser photocoagulation in the treatment of CRVO provides prophylactic and curative therapy for eye complications.*

• Laser photocoagulation is effective in the treatment of CRVO, but should be indicated and applied with discernment.

Visual prognosis in ischemic CRVO is more reserved

- Initial low VA value and extension of retinal ischemia are correlated with reduced final VA.

Treatment of branch retinal vein obstruction

- treatment of risk factors
- sectoral photocoagulation for the treatment of ischemia in the obstructed territory to prevent the development of vitreous hemorrhage by preretinal neovascularization
- macular photocoagulation is indicated in branch obstruction complicated with persistent macular edema with VA less than 5/10 for VA stabilization and amelioration - RVO prognosis is generally good, 50-60% have final VA 20/40 or better even without treatment

Ophthalmic antiglaucomatous treatment for decreasing high intraocular pressure

- Prevention of secondary neovascular glaucoma by systematic ophthalmologic examination by slit lamp and gonioscopy

- Emergency retinal panophotocoagulation if rubeosis iridis and angular neovascularization are present.

Other drugs with questionable effects: urokinase (through microcatheter, it would improve VA), oral or infused pentoxifylline (seems to improve VA in some RVO patients) ticlopidine.

Conclusions

- Retinal venous occlusion (central or branch retinal vein) is accompanied by retinal ischemia, a factor causing edematous maculopathy and proliferative neovascularization with its complications: vitreous hemorrhage, retinal traction detachment, secondary neovascular glaucoma.
- RVO has two clinical forms: non-ischemic edematous 75-80% with possible conversion to ischemic form - 2% and uninfused ischemic form with retinal edema and neovascularization with individualized clinical appearance for non-ischemic, ischemic CRVO and RVO.

- RVO treatment should be adapted to the clinical form of RVO to reduce retinal and macular edema by:

➢ antiangiogenic agents - antiVEGF: ranibizumab, bevacizumab, aflibercept intravitreal therapy which limits the development of neovessels favoured by postocclusive retinal ischemia, prevent the

development of macular edema, stabilize and can significantly improve VA; are an effective treatment for RVO.

- ➢ corticosteroids - intravitreal triamcinolone and dexamethasone - implant with anti-inflammatory effect are indicated in the treatment of macular edema, when an effective therapeutic response to antiVEGF treatment or in combined treatment (corticoids, antiVEGF, laser photocoagulation) with higher therapeutic efficiency is NOT obtained.
- ➢ laser photocoagulation destroys the ischemic territory, prevents and combats the preretinal / prepapillary / iris neovascular proliferation in ischemic CRVO.
  - ✓ Laser photocoagulation (depending on FA) improves the visual prognosis and is an effective treatment for complications of ischemic capillaropathy.

- Surgical treatment in RVO aims to treat venous occlusion and secondary macular edema (not generally recommended for possible complications and moderate visual results)
- Treatment of the non-ischemic form of RVO is the treatment of macular edema with: antiVEGF, corticosteroids, laser photocoagulation, but in all cases of non-ischemic RVO, clinical surveillance OCT, FA are required to detect possible progression to ischemic form.
- Treatment of the ischemic form benefits from the treatment of the edema with: antiVEGF, intravitreal corticosteroids, focal laser photocoagulation, panphotocoagulation, tissue plaminogen activator, surgical treatment if necessary.
- The current prognosis of RVO is clearly improved by the introduction of OCR-SD and the use of intravenous drugs - antiVEGF, steroids and photocoagulation laser treatment applied with discernment.
- Prophylactic and curative treatment of risk factors (hypertension, diabetes, coagulation disorders) is important.

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