

Recent Updates In Retinal Vein Occlusion

An essay
Submitted for partial fulfillment of master degree in ophthalmology

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2016



Acknowledgement

First of all, all gratitude is due to Allah almighty for blessing this work, until it has reached its end, as a part of his generous help, throughout my life.

Really I can hardly find the words to express my gratitude to Prof. Dr. Magdy Mohamed El Barbary, Professor of ophthalmology, faculty of medicine, Ain Shams University, for his supervision, continuous help, encouragement throughout this work and tremendous effort he has done in the meticulous revision of the whole work. It is a great honor to work under his guidance and supervision.

I would like also to express my sincere appreciation and gratitude to **Dr. Tamer Fahmy Eliwa**, Lecturer of ophthalmology faculty of medicine, Ain Shams University, for his continuous directions and support throughout the whole work.

Last but not least, I dedicate this work to my family, whom without their sincere emotional support, pushing me forward this work would not have ever been completed.



Sara Daoud

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List of Abbreviations

AMD	Age related macular degeneration
ASNV	Anterior Segment Neovascularization
BCVA	Best Corrected Visual Acuity
BM	Bruch's membrane,
BRV	Branch Retinal vein
BRVO	Branch Retinal vein occlusion
BRVOS	Branch Vein Occlusion Study Group
CMT	Central macular thickening
CRAI	cilioretinal arteriolar insufficiency (CRAI)
CFT	central foveal thickness
CRV	Central Retinal vein
CRVO	Central Retinal vein occlusion
DEX	Dexamethasone
ELM	External limiting membrane
ERG	Electroretinography
FA	Fluorescein Angiography

FDA	United States Food and Drug Administration
FAF	Fundus Autofluorescence
GCL	Ganglion cell layer
GL	Gride laser
HRB	High Reflectance Band
INL	Inner nuclear layer
IOP	Intraocular pressure
IPL	Inner plexiform layer
IVBI	Intravitreal bevacizumab injection
IVFA	Intravenous Fluorescein Angiography
IS\OS	Inner segment/outer segment of photoreceptors
logMAR	Logarithm of the minimum angle of resolution
ME	Macular Edema
mfERG	Multifocal Electroretinogram
NFL	Nerve fiber layer
NVI	Neovascularization of the Iris
OCT	Optical coherence tomography

ONL	Outer nuclear layer
OPL	Outer plexiform layer,
PDT	Photodynamic therapy
PhNR	Photopic negative response
PIRW	Perivenular Ischemic Retinal Whitening
PRP	Panretinal photocoagulation.
RPE	Retinal pigment epithelium
PRN	When necessary
PSNV	Posterior Segment Neovascularization
RVO	Retinal vein occlusion
SD-OCT	Spectral-domain optical coherence tomography
SGL	Subthreshold Grid Laser
TA	Triamcinolone Acetonide
VEGF	Vascular Endothelial Growth Factor

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Aim of the Essay

Review the retinal vein occlusion as risk factors, etiology, classifications, investigations and presentation of different treatment options used now for treatment of the retinal vein occlusion.

Dermography

RVO is the second only to diabetic retinopathy as the most common retinal vascular disease. It is estimated that 16.4 million people in the world have RVO. In a population based study (pooled data involving approximately 50,000 participants from the United States, Europe, Asia, and Australia) RVO was the fifth most frequent cause of unilateral blindness (*Rogers et al.*, 2010).

The prevalence of RVO has shown an age and sex standardized prevalence of 5.20 per 1,000 for any form of RVO. The prevalence of branch RVO (BRVO) was higher than central RVO (CRVO), increased with age and there was no difference between the sexes. Over 50 % of cases of RVO occur in patients older than 65 years of age (*Mall et al.*, 2013).

CRVO in young adults is uncommon: only 10–15% of patients with CRVO are under 40 years of age (*Marcucci et al*, 2010).

The risk of developing a second episode of the same or different types of RVO in the same eye was 1% within 2 years and 2.5% within 4 years (*Hayreh*, 2005).

The prevalence of bilateral CRVO has been reported to be 6.3–12%. Bilaterality of CRVO seems higher than bilaterality of BRVO (*Rogers et al.*, 2010).

The risk of BRVO at each vein-posterior crossing is about 12 times higher than each vein-anterior crossing. Occlusion in inferotemporal branches occurs in 22–43% of cases, macular branches in 24%, and nasal branches in 0.5–2.6%. As nasal BRVOs are associated with fewer complications and better vision than other types, they are often under detected (*Berger et al.*, 2015).

Familial clustering of RVO (CRVO in particular) has been reported but these reports have been few in number. It is interesting that such cases are more often bilateral, with a younger age at onset than sporadic cases (*Karia*, 2010).

Etiology

The etiology of RVO is still incompletely known. An external compression of the vein wall is suggested (*Cossca et al.*, 2011).

The tract of central retinal vein passing through the lamina cribrosa is the most frequent site of occlusion as central retinal veins and arteries are present within the same adventitial sheath (Figure 1), arterial stiffness can affect neighboring veins, leading to CRVO (Marcucci, 2010).

Several reasons are responsible for this localization:

- (1) As ageing occurs, the collagen tissue of the lamina cribrosa becomes thicker and stiffer causing a compression of the vascular wall.
- (2) Degenerative processes at the expense of the central retinal artery may cause compression on the contiguous venous wall.
- (3) The consequently turbulent flow enhances the risk of endoluminal thrombus formation (*Marcucci*, 2010).

BRVO may be the result of a combination of vein compression at arteriovenous crossings, degenerative changes within venous walls, and hypercoagulability (*Jaulim et al.*, 2013).

The turbulent flow together with the preexisting endothelial vascular damage from the different conditions creates a local environment favorable to intravascular thrombus formation. Once the venous flow is compromised or interrupted, retinal ischemia ensues downstream from the occlusion site. Retinal ischemia is one of the most important up-regulators of vascular endothelial growth factor (VEGF) production (*Pe'er et al.*, 1995).