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Pharmacotherapeutic Challenges of Vascular Endothelial Growth Factor Inhibitors in Retinal Vein Occlusion

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ABSTRACT:

Currently intravitreal antiangiogenics are undoubtedly efficacious in retinal vein occlusion (RVO), however prevailing safety concerns continue to be a key reason for continued high-quality clinical studies in the real world, taken into account that the patients with comorbidities are excluded from the randomized controlled trials. Recent studies substantiate the relevance of optimizing pharmacotherapy of RVO. Hopefully a noninvasive, multitarget, affordable, time- and cost-saving pharmacotherapy of RVO may offer a more efficient alternative to antiangiogenics monotherapy in the foreseeable future.

Keywords: Vascular Endothelial Growth Factors, Vascular Endothelial Growth Factor Inhibitors, Therapeutic Uses, Ophthalmopharmacotherapy, Injections, Intraocular; Retinal Vein Occlusion

INTRODUCTION:

Retinal vein occlusion (RVO) as a vasooclusive disorder of the retinal vein is the most common visually disabling disease affecting the retina after diabetic retinopathy in which arterial risk factors are much more relevant than venous factors, and is a major cause of vision loss and even blindness[1]. The incidence of retinal vein occlusion is growing due to aging population globally, which represents a healthcare challenge[2]. In a recent analysis of pooled data from population studies worldwide, the overall RVO prevalence was 0.77% (0.64% branch retinal vein occlusion (BRVO), 0.13% central retinal vein occlusion (CRVO), translating to more than 28 million individuals worldwide affected by RVO[3]. The first case of central retinal vein occlusion was reported by Richard Liebreich in 1855[4]. The first case of branch retinal vein occlusion was reported by Theodor Leber in 1877[5]. Despite being recognized in the 19th century there are still gaps in understanding the etiology and pathogenesis of vasooclusive disorders of the central retinal vein and its branches. Although it is more common in the middle-aged and elderly population, no age group is immune to it. The pathogenesis of RVO has varied systemic and local implications. Many case-control studies have examined the clinical features and risk factors in this disorder[6-10].

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Known risk factors for RVO include systemic vascular disease, hypertension, diabetes mellitus, hyperlipidemia and glaucoma. The strongest risk factor for any type of RVO is hypertension[3]. Hypercoagulable states are associated with RVO. These include primary hypercoagulable states with a defect in the physiological anticoagulant mechanism and secondary hypercoagulable states, which are conditions, associated with an increased risk of thrombosis [11].

In central retinal vein occlusion (CRVO) the obstruction is located in the central vein, at the level of the optic nerve, so most of the retina is affected. Anatomic features make the central retinal vein vulnerable to occlusion at this location. As the optic nerve and the accompanying central retinal artery and vein pass through the sieve-like connective tissue of the lamina cribrosa, the central retinal vein normally narrows, and the dense connective tissue of the lamina cribrosa limits any expansion of the traversing optic nerve and vessels within. Any thickening of the central retinal artery, which shares a common fibrous tissue sheath with the vein, might easily compress the lumen of the adjacent central retinal vein and start in motion the sequence of events that lead to thrombus formation[2].

In branch retinal vein occlusion (BRVO), the obstruction is located in one of the branches of the central vein, affecting only part of the posterior pole and the portion of the peripheral retina drained by occluded branch[2].

Sclerosis of the retinal artery which is associated with systemic hypertension or arteriosclerosis may result in further compression of the vein, when the increased rigidity of arterial wall and contraction of the adventitial sheath shared by artery and vein occur. Mechanical obstruction of the vein through the rigid artery in the A/V crossing may result in turbulent blood flow producing damage to venous endothelium and intima media and the sequence of events leading to occlusion of the vein[12,13]. Hemi-retinal vein occlusion affects two branches of central retinal vein in the superior or inferior retinal hemisphere[2].

The aim of this review is to evaluate the evidence on the recent pharmacotherapy of retinal vein occlusion worldwide, based on the currently available findings.

METHODOLOGY:

For this review, a literature search was conducted using PubMed®/Medline® and Google Scholar for studies published up to December 2025. The following keywords were used in various combinations: “retinal vein occlusion”, “pharmacotherapy of retinal vein occlusion”, “antiangiogenics in retinal vein occlusion”, “intravitreal pharmacotherapy of retinal vein occlusion by anti-VEGF”. Articles with high or medium clinical relevance were selected for this review.

The past two decades have seen important developments in the therapy of retinal diseases. The emerged class of agents targeting vascular endothelial growth factor (VEGF) offered the paradigm-shifting possibility of treatment strategy, particularly in RVO.

Three VEGF inhibitors have been evaluated for pharmacotherapy in patients with CRVO and BRVO: ranibizumab, aflibercept and bevacizumab. The last one widely used in ophthalmology as an off-label. At present the standard of care for macular edema (ME) secondary to CRVO or BRVO consisted of intravitreal injections of anti-VEGF agents[13-16], demonstrated the efficacy of this treatment approach[17-22].

There has been an increase in studies examining the long-term efficacy of anti-VEGF in CRVO and BRVO[21,22]. Accordingly, a multicenter, real-world observational study of 24-month treatment by ranibizumab has indicated a need for continued injections in order to maintain an improved visual acuity[23]. Similarly, Hunt et al.[24] evidenced that more than 50% of patients were still receiving treatment after 3 years. Another multicenter, international, observational study evaluating 2-year outcome concluded that frequent anti-VEGF injections are more effective for a visual improvement[25]. Three-Year Outcomes study of antiangiogenic therapy in BRVO demonstrated similar visual results for aflibercept, ranibizumab and bevacizumab[26].

There have been attempts to evaluate the long-term outcomes up to ninth year after aflibercept injections corresponding to treat-and-extend protocol in CRVO [27]. Researchers have recognized the efficacy of this treatment regimen, but mentioned the absence of comparative findings with other treatment protocols. A recent meta-analysis by Wu et al.[28], after systematically reviewing and meta-analysing the randomized controlled trials and real-world studies on the efficacy of aflibercept 2 mg or ranibizumab in RVO, concluded that there was no notable difference, however future clinical studies are required to provide new insights into the optimal treatment approach. Besides, the question when to initiate anti-VEGF therapy is still left unanswered[29].

Si et al.[30] attempted to provide the most valuable indicator for unfavorable outcomes after antiangiogenic therapy in RVO and postulated that visual acuity at one month after the first anti-VEGF injection is the key indicator.

Recent study have evidenced a significantly deteriorated response to intravitreal VEGF inhibitors in patients with RVO and carotid artery disease comorbidity comparing to patients suffering only from RVO[31]. So, while current intravitreal antiangiogenics are undoubtedly efficacious, prevailing safety concerns, especially systemic safety, continue to be a key reason for continued high-quality clinical studies in the real world, taken into account that the patients with comorbidities are excluded from the randomized controlled trials. There has been an increase in studies examining cardiovascular safety of locally delivered anti-VEGF agents in retinal vascular diseases[23,32,33]. Risk of anti-VEGF related cardiovascular adverse events (AEs) in patients with CRVO and BRVO was evaluated in case-control study conducted by Albrecht et al.[32]. Researchers have recognised an increased risk of death and ischemic stroke in patients with CRVO and BRVO respectively, however pointed the need for further validation.

Glacet-Bernard et al.[23] documented the transient ischemic attack in CRVO cases and hypertension, myocardial ischemia, cerebral infarction in BRVO, cases possibly related to therapy by ranibizumab**.**

Recent meta-analyses as those of authors like Zhong et al.[34], Ngo Ntjam et al.[35], Reibaldi et al.[36], Jhaveri et al.[37] cover a significant amount of works that raise awareness about cardiovascular AEs associated with antiangiogenic therapy of retinal diseases, such as RVO, age-related macular degeneration and diabetic retinopathy.

A cross-sectional study in the US Food and Drug Administration Adverse Event Reporting System (FAERS) database[38] indicated that ranibizumab, aflibercept and bevacizumab use correlates with an increased risk of cardio- and cerebrovascular events. Further study[39] explored the similar causal association for all VEGF inhibitors, however a higher frequency of cardiac and central nervous AEs were evidenced after ranibizumab injections. Anti-VEGF drugs cardiovascular safety real-world concern was highlighted by Li and coworkers[40].

Researchers have also recognised that ranibizumab is associated with cardiac disorders, hypothesing that the drug inhibits VEGF-A, responsible for cardiomyocites protection and survival. Recent studies reconfirmed a cardiotoxicity of VEGF inhibitors[41,42]. It is therefore crucial to explore the cardiovascular impact of intraocular antiangiogenic therapy[43]. Furthermore, a recent meta-analysis[44], after systematically reviewing and meta-analysing the studies on the relation between RVO and acute myocardial infarction (MI) published before, concluded that " RVO is linked to a 32.4% elevated risk of MI" and emphasized a need of cardiovascular monitoring for RVO patients. The latest research on the matter also evidenced an increased risk of MI in CRVO patients with high-intensity treatment[45]. There is a growing recognition that real-world concerns with antiangiogenic ocular therapy in RVO include the potential for cardiovascular side effects, a particular worry in patients with multimorbidity[46] , and the need for frequent repeated intraocular injections with long-term monitoring[47], with the cumulative potential of ocular side effects and resistancy[13]. From the other side, the economic burden of intraocular antiangiogenic injections on the healthcare system is another current concern[2,48]. Besides, retinal vein occlusion, especially CRVO, significantly deteriorates not only a vision, mental health, but also the quality of life[49]. Accordingly, all the reports described above substantiate the relevance of optimizing pharmacotherapy of RVO. There is still room for improvement. Unlocking pathological mechanisms in RVO will help to find potential therapeutic targets[50]. The development of innovative approaches continues[51-53]. The recent one is presented by Ma et al.[54]. The efforts have been made to develop a multifunctional nanodrug with synergistic activity containing small interfering RNA (siRNA) targeting VEGF and dexamethasone. Researchers evidenced antioxidative, anti-inflammatory and anti-angiogenic effects of this nanodrug and stated that "simultaneous delivery of all components to the target site, enhanced their combined therapeutic potential while reducing systemic exposure and adverse effects" with "potential as a versatile and effective treatment for multiple ocular conditions".

Hopefully a noninvasive, multitarget, affordable, time-and cost-saving pharmacotherapy of RVO may offer a more efficient alternative to antiangiogenics monotherapy in the foreseeable future.

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