

Original Article

Pattern of dyslipidemia in retinal vein occlusion (RVO) patients of Bangladesh.

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Abstract

Purpose: To investigate association between the development of RVO and dyslipidemia.

Methods:

In this retrospective study we investigated 135 patients with RVO. 48 (35.55%) with central retinal vein occlusion, 78 (57.77%) with branch retinal vein occlusion and 9 (6.66%) with hemi retinal vein occlusion.

Result:

Patient age was $54.98 \pm SD 12.86$ years. Minimum age was 31 years and maximum age was 85 years. Female was 57 (42.22%) and male was 78 (57.77%). From age 31 – 40 years there were BRVO 15, CRVO 7 and HRVO 1. From age 41 – 50 years there were BRVO 15, CRVO 15 and HRVO 2. From age 51 – 60 years there were BRVO 21, CRVO 13 and HRVO 3. From age 61 – 70 years there were BRVO 18, CRVO 4 and HRVO 2. From age 71 – 80 years there were BRVO 8, CRVO 5 and HRVO 1. From age 81 – 90 years there were BRVO 1 and CRVO 4. There were 92 patients with hypertension, 37 with diabetes and 28 were both hypertension and diabetes. In case of RVO high density lipoprotein (HDL) was mean of $31.70 \pm SD 9.54$, 110 (81.48%) patients were < 40 mg/dl. Low density lipoprotein (LDL) was mean of $136.06 \pm SD 45.62$, 83 (61.48%) patients were > 130 mg/dl. Total cholesterol (TC) was mean of $192.57 \pm SD 44.67$, 48 (35.55%) patients were > 200 mg/dl. Triglyceride (TG) meant $227.04 \pm SD 182.96$, 91 (67.40%) patients were > 150 mg/dl. In case of CRVO HDL was mean of $32.21 \pm SD 11.24$, LDL was mean of $121.17 \pm SD 46.49$, TC was mean of $191.78 \pm SD 50.57$, TG was mean of $270.17 \pm SD 262.81$. In case of BRVO HDL was mean of $31.37 \pm SD 8.58$, LDL was mean of $145.51 \pm SD 44.71$, TC was mean of $192.43 \pm SD 41.43$, TG was mean of $197.36 \pm SD 108.04$. In case of HRVO HDL was mean of $27.00 \pm SD 3.89$, LDL was mean of $129.44 \pm SD 37.90$, TC was mean of $180.56 \pm SD 45.64$, TG was mean of $143.22 \pm SD 80.98$.

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Conclusion:

In case of CRVO low HDL and high TG are risk factors. In case of BRVO low HDL, high LDL and high TG are risk factors. In case of HRVO low HDL is risk factor. Though sample size of HRVO is too small to give any comment.

Keywords:

Retinal vein occlusion, central retinal vein occlusion, branch retinal vein occlusion, hemi retinal vein occlusion, high density lipoprotein, low density lipoprotein, total cholesterol, triglyceride.

Introduction:

Retinal vein occlusion (RVO) is a potentially vision-threatening retinal vascular disorder, representing the second most frequent disease of the eye. RVO is a relatively frequent disease which has been reported to be associated with an increased risk of mortality from cardiovascular diseases. To date, the pathogenesis of RVO is not fully understood. Atherosclerosis is considered to be the most important underlying condition and several traditional risk factors (hypertension, diabetes, and smoking habit) have been identified to play a role in the pathogenesis of the disease.

Based on the exact location of the occlusion and the clinical findings, RVO is generally grouped into central RVO (CRVO), hemi RVO, and branch RVO (BRVO).[2,6] Although BRVO is the most common type, visual acuity reduction is usually more marked in CRVO, as macular edema is often more prevalent and more severe in CRVO.

Apart from macular edema, complications of RVO include neovascularization, vitreous hemorrhage, optic neuropathy, and secondary glaucoma. Over the years, treatment for RVO has developed from clinical observation to the use of laser, use of intravitreal corticosteroids, and more recently, the use of intravitreal anti-vascular endothelial growth factors (anti-VEGF). In addition, pars plana vitrectomy surgery is deployed in severe vitreous hemorrhage from RVO. These treatment modalities are often deployed to treat or stop the progression of the complications of RVO that could result in irreversible blindness.

RVO is often a disease of the elderly, most commonly occurring in patients aged 30 years and above. Details of

the pathogenesis are unclear, and when proposed, complex. However, atherosclerotic changes in the retinal veins are considered to be the most important pathogenic mechanism from which RVO occurs. Risk factors associated with atherosclerosis—hypertension, diabetes mellitus, hyperlipidemia, and smoking—are seen to be associated with RVO, further buttressing the importance of atherosclerosis in the development of RVO. In addition, the role of thrombophilic risk factors, myeloproliferative disorders, and other causes of hypercoagulation of blood are gaining prominence in the etiopathogenesis of RVO. Besides systemic predisposing factors for RVO, glaucoma (most commonly primary open angle) is the main ocular predisposing factor.

RVO causes retinal hypoxia leading to an up-regulation of the production of VEGF, causing neovascularization and macular edema by stimulating vascular endothelial cell growth and increased vascular permeability.

Materials And Methods:

It was a hospital-based retrospective observational study. All patients diagnosed with RVO and who met the criteria from January 2023 to July 2024 were included in the study. The inclusion criterion was all patients diagnosed with RVO in the hospital within the study period. Using the inclusion and exclusion criteria, 135 case files of 135 patients were reviewed for data collection. The main outcome variables to be studied were demographic characteristics, types of RVO, and risk factors for RVO. Records of all patients diagnosed with RVO within the period of study were reviewed only by the principal investigator to ensure uniformity in the reporting. Case files containing the medical records of the patients were collected from the Medical Records Department. A detailed review of the case files was done. Bio-data, ophthalmic and systemic history, and ophthalmic findings were collated. Details of ophthalmic findings collated include visual acuity assessment with the Snellen visual acuity chart, anterior segments findings from the slit-lamp biomicroscopy, dilated funduscopy findings, optical coherence findings and serum lipid profile findings.

Result:

Mean patient age was $54.98 \pm SD 12.86$ years. Minimum age was 31 years and maximum age was 85 years. Female was 57 (42.22%) and male was 78 (57.77%). From age 31 – 40 years there were BRVO 15, CRVO 7 and HRVO 1. From age 41 – 50 years there were BRVO 15, CRVO 15 and HRVO 2. From age 51 – 60 years there were BRVO 21, CRVO 13 and HRVO 3. From age 61 – 70 years there were BRVO 18, CRVO 4 and HRVO 2. From age 71 – 80 years there were BRVO 8, CRVO 5 and HRVO 1. From age 81 – 90 years there were BRVO 1 and CRVO 4.

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BRVO high density lipoprotein (HDL) was mean of $31.70 \pm SD 9.54$, 110 (81.48%) patients were < 40 mg/dl. Low density lipoprotein (LDL) was mean of $136.06 \pm SD 45.62$, 83 (61.48%) patients were > 130 mg/dl.

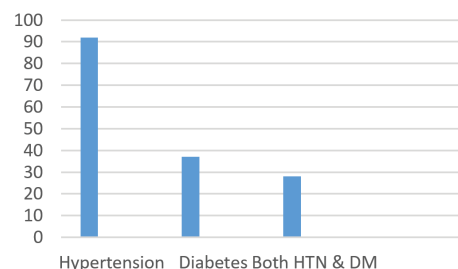
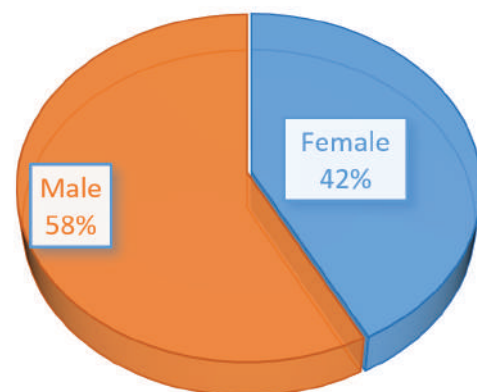
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In case of HRVO HDL was mean of $27.00 \pm SD 3.89$, LDL was mean of $129.44 \pm SD 37.90$, TC was mean of $180.56 \pm SD 45.64$, TG was mean of $143.22 \pm SD 80.98$.

Age distribution:

Age	Population	BRVO	CRVO	HRVO
31-40	23	15	7	1
41-50	32	15	15	2
51-60	37	21	13	3
61-70	24	18	4	2
71-80	14	8	5	1
81-90	5	1	4	0

Risk Factors:



Lipid profile distribution:

	Mean HDL	Mean LDL	Mean TC	Mean TG
CRVO	32.21 ± SD 11.24	121.17 ± SD 46.49	191.78 ± SD 50.57	270.17 ± SD 262.81
BRVO	31.37 ± SD 8.58	145.51 ± SD 44.71	192.43 ± SD 41.43	197.36 ± SD 108.04
HRVO	27.00 ± SD 3.89	129.44 ± SD 37.90	180.56 ± SD 45.64	143.22 ± SD 80.98

Discussion

This study substantiates the conclusion that low HDL-C is a risk factor for RVO. In contrast, we found no evidence of a causal association with total cholesterol but high LDL-C and high triglycerides are also associated with RVO.

Although the exact pathogenesis of RVO remains elusive, numerous studies have reported that RVO is associated with atherosclerosis, a chronic inflammatory disease of the arteries. Hyperlipidemia is a major risk factor for atherosclerosis, and abnormal lipid metabolism is an important component of atherosclerosis. Meanwhile, LDL-C is the most abundant atherogenic lipoprotein in plasma and leads to the initiation and progressive growth of atherosclerotic plaque increases in a dose-dependent manner.

Thus, LDL-C may play an important role in the development of RVO. And the association between elevated LDL-C level and the development of RVO has been demonstrated by previous studies. GLGC results suggested that high LDL-C may be a risk factor for RVO. However, many lipid trait SNPs carry pleiotropic lipid trait effects and overlap between genetic determinants of LDL-C and HDL-C is widespread. MVMR is useful to estimate the direct causal effect of each lipid profile component, independent of the other lipid profile variables. MVMR analysis for plasma lipids suggested that high LDL-C may not be an independent risk factor for RVO.

A few studies have reported that the total cholesterol is significantly related to the risk of RVO. Total cholesterol and LDL-C are easy direct targets for medical treatments, such that baseline total cholesterol and LDL-C level may vary substantially in clinical trials, complicating the interpretation of results regarding RVO risk. Our findings suggested that total cholesterol may not be independent risk factors for RVO. Thus, more studies are required to determine the causal relationship between total cholesterol and RVO.

Unlike LDL-C, HDL-C is widely believed to exert atheroprotective effects. Despite the evidence from various previous reports indicating an association between HDL-C and the risk of RVO, this conclusion remains controversial. HDL-C directly mediates reverse cholesterol transport that hinders the accumulation of cholesterol in the arterial wall

and prevents the progression of atherosclerosis. Additionally, HDL-C has been shown to inhibit the endothelial inflammatory response and oxidation of LDL-C. Through these underlying mechanisms, increasing HDL-C level may have improved the endothelial health of the retinal vessel, and decreased the risk of RVO. Many lipid trait SNPs carry metabolic syndrome trait effects, especially diabetes, which is an important and concerning confounder. A low HDL-C level is associated with a higher risk of type 2 diabetes and studies have shown that RVO is associated with diabetes. Low HDL-C, high LDL-C and high TG are risk factor for RVO, suggesting that controlling of HDL-C, LDL-C and TG level may be effective in managing RVO.

In conclusion, the present study indicated that exposure to abnormal lipid level may increase the risk of RVO.

Conclusion: In case of CRVO low HDL and high TG are risk factors. In case of BRVO low HDL, high LDL and high TG are risk factors. In case of HRVO low HDL is risk factor. Though sample size of HRVO is too small to give any comment.

References:

1. Shin YU, Cho H, Kim JM, Bae K, Kang M, Shin JP, et al. Prevalence and associated factors of retinal vein occlusion in the Korean National Health and Nutritional Examination Survey, 2008–2012. *Medicine (Baltimore)* 2016;95: e5185.
2. Thapa R, Bajimaya S, Paudyal G, Khanal S, Tan S, Thapa SS, et al. Prevalence, pattern and risk factors of retinal vein occlusion in an elderly population in Nepal: The Bhaktapur retina study. *BMC Ophthalmol* 2017;17:1–8.
3. Ivanovska Adjievska B, Boskurt S, Orovcanec N, Dimovska-Jordanova V. The outcome of low-frequency intravitreal bevacizumab therapy for macular edema in retinal vein occlusions. *Clin Ophthalmol* 2017;11:1183–90.
4. Brown DM, Campochiaro PA, Singh RP, Li Z, Gray S, Saroj N, et al.; CRUISE Investigators. Ranibizumab for macular edema following central retinal vein occlusion: Six-month primary end point results of a phase III study. *Ophthalmology* 2010;117:1124–33.e1.
5. Rath EZ, Frank RN, Shin DH, Kim C. Risk factors for retinal vein occlusions a case-control study. *Ophthalmology* 1992;99:509–14.
6. Sharma R, Bhat MA. Risk factors in retinal vein occlusion. *IJCMR* 2016;3:979–81.
7. Jaulim A, Ahmed B, Khanam T, Ms C, Chatziralli IP. Branch retinal vein occlusion: epidemiology, pathogenesis, risk factors, clinical features, diagnosis, and complications. An update of the literature. *Retina* 2013;33:901–10.
8. Rogers S, McIntosh RL, Grad B, Journ D, Cheung N, Lim L, et al. The prevalence of retinal vein occlusion: Pooled data from population studies from the United States, Europe, Asia, and Australia. *Ophthalmology* 2011;117:1–14.

9. Wong TY, Larsen EKM, Klein R, Mitchell P, Couper DJ, Klein BEK, et al. Cardiovascular risk factors for retinal vein occlusion and arteriolar emboli. *Ophthalmology* 2005;112:540–7.
10. Oak R. Vein occlusion in Chinese subjects. *Ophthalmology* 2007;114:1795–6.
11. Sofi F, Marcucci R, Fedi S, Giambene B, Sodi A, Menchini U, et al. High lipoprotein (a) levels are associated with an increased risk of retinal vein occlusion. *Atherosclerosis* 2010;210:278–81.
12. Rehak M, Krcova V, Slavik L, Fric E, Langova K, Ulehlova J, et al. The role of thrombophilia in patients with retinal vein occlusion and no systemic risk factors. *Can J Ophthalmol* 2010;45:171–5.
13. Karia N. Retinal vein occlusion: Pathophysiology and treatment options. *Clin Ophthalmol* 2010;4:809–16.
14. Prisco D, Marcucci R. Retinal vein thrombosis: Risk factors, pathogenesis and therapeutic. *Pathophysiol Haemost Thromb* 2002;32:308–11.
15. Malayan AS, Shakhshuvaryan ML, Grigoryan GL, Melkonyan AK. Retinal vein occlusion in Armenia. *Eur J Ophthalmol* 1999;9:196–201.
16. Arnarsson A, Stefa´nsson E. Laser treatment and the mechanism of edema reduction in branch retinal vein occlusion. *Invest Ophthalmol Vis Sci* 2000;41:877–9.
17. The Central Vein Occlusion Study Group. Evaluation of grid pattern photocoagulation for macular edema in central vein. *Ophthalmology* 1995;102:1425–33.
18. Campochiaro PA, Heier JS, Feiner L, Gray S, Saroj N, Rundle AC, et al.; BRAVO Investigators. Ranibizumab for macular edema following branch retinal vein occlusion six-month primary end point results of a phase III study. *Ophthalmology* 2010;117:1102–12.e1.
19. Lee JY, Yoon YH, Kim HK, Yoon HS, Kang SW, Kim J, et al.; Korean RVO Study. Baseline characteristics and risk factors of retinal vein occlusion: A study by the Korean RVO study group. *J Korean Med Sci* 2013;28:136–44.
20. Bradshaw SE, Gala S, Nanavaty M, Shah A, Mwamburi M, Kefalas P. Systematic literature review of treatments for management of complications of ischemic central retinal vein occlusion. *BMC Ophthalmol* [Internet]. 2016;16:104.
21. Prisco D, Marcucci R, Bertini L, Gori AM. Cardiovascular and thrombophilic risk factors for central retinal vein occlusion. *Eur J Intern Med* 2002;13:163–9.
22. Aritirk N, Oge Y, Erkan D, Sullu Y, Mohajery F. Relation between retinal vein occlusions and axial length. *Br J Ophthalmol* 1996;80:633–6.
23. Feist RM, Ticho BH, Shapiro MJ, Farber M. Branch retinal vein occlusion and quadratic variation in arteriovenous crossings. *Am J Ophthalmol* 1992;113:664–8.
24. Paul M, Wayne S, Andrew C. Prevalence and associations of retinal vein occlusion in Australia. *Arch Ophthalmol* 1996;114:1243–7.
25. Hayreh SS, Zimmerman MB, Podhajsky P. Incidence of various types of retinal vein occlusion and their recurrence and demographic characteristics. *Am J Ophthalmol* 1994;117:429–41.