Sriwijaya Journal of Ophthalmology(SJO)
http://sriwijayaopthalmology.com
Vol 1 Issue 1 2018

Branch Retinal Vein Occlusion with Complications of Vitreus Bleeding Performed by Vitrectomy and Endolaser

Faika Novadianaz[1*]

[1]Department of Ophthalmology, Faculty of Medicine, Universitas Sriwijaya, Indonesia

*correspondence author email : faika.novadianaz@gmail.com

Abstract

Introduction. Retinal vein occlusion is the largest group of retinal blood vessel abnormalities after diabetic retinopathy. Macular edema and neovascularization are major complications in BRVO that require therapy**.** Vitrectomy is indicated in cases of vitreous hemorrhage that fail spontaneous resolution after 6 weeks to 3 months. The time to do vitrectomy depends on the tendency of the ophtalmologist and the patient's visual needs.

Case Presentation. A woman, 59 years old, housewife, came to the Eye Clinic on July 2016. The main complaint history is that the right eye has become more blurred since ± 2 months ago. On examination of the posterior segment of the right eye the detail is difficult to assess. In this patient suspected turbidity of the vitreous cavity so that an ultrasound examination was performed. Vitreous bleeding is an indication for vitrectomy. Intraoperatively after the vitrectomy, bleeding and ghost vessels were found in the super-temporal region.

Conclusion. The main goals of therapy in BRVO are to improve hemodynamics, overcome macular edema, and prevent neovascularization.

Keywords: branch retinal vein occlusion, complication, vitreus bleeding, vitrectomy, endolaser.

Introduction

Retinal vein occlusion is the largest group of retinal blood vessel abnormalities after diabetic retinopathy. Occlusion that occurs in the retinal vein is divided into central retinal vein occlusion (CRVO) and branch retinal vein occlusion (BRVO). CRVO is caused due to thrombosis in the central retinal vein as it passes through the lamina cribrosa, whereas BRVO is caused due to venous thrombosis in the artery venosus crossing where arteries and veins have the same vascular

membrane.[1,2] The Beijing Eye Study states, reported a higher incidence of BRVO than CRVO, where the 10-year incidence of BRVO was 1.6 per 100 subjects, and CRVO was only 0.3% 100 subjects.[3]

Based on the *Branch Retinal Vein Occlusion Study* (BVOS), the prognosis for BRVO is better than CRVO. Exactly 50-60% of cases of BRVO that are not treated have vision that persists at onset, even improves ≥ 6/12 after one year. So, when patients with mild visual impairment due to macular edema caused by BRVO, it is enough to observe in the first 3 months to see the development of the situation. However, in some patients the symptoms may appear late and there can be more severe vision problems. In this condition, only 18 - 41% spontaneous improvement occurs with an average vision of 6/12 so it is advisable to do treatment earlier.[4]

Macular edema and neovascularization are major complications in BRVO that require therapy. In macular edema, laser photocoagulation, intravitreal steroid injection, or intravitreal anti *vascular endothelial growth factor* (VEGF) injection can be performed. Neovascularization of the disk or retina is an indication of photocoagulation in the ischemic area, although there is evidence that photocoagulation does not affect the visual prognosis in cases of vitreous bleeding.4 Vitrectomy is indicated in cases of vitreous hemorrhage that fail spontaneous resolution after 6 weeks to 3 months. The time to do vitrectomy depends on the tendency of the eye doctor and the patient's visual needs.[1]

Reported a case of branch retinal vein occlusion with complications of vitreous haemorrhage performed by vitrectomy and endo-laser with visual repair.

Case Report

A woman, 59 years old, housewife, came to the RSMH Eye Clinic on July 25, 2016 with medical records 963115. The main complaint history is that the right eye has become more blurred since ± 2 months ago.

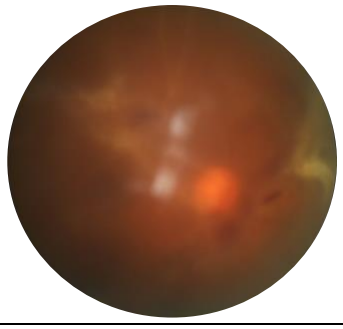
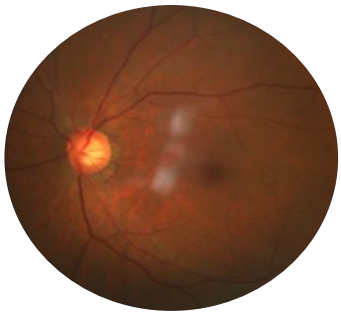
Since about 2 years ago sufferers complained of vision in the right eye blurred, blurred felt suddenly, like seeing flying objects (+). Complaints of red eye (-), pain (-), glare (-), vision like a curtain (-), vision like a rainbow (-). Patients are treated by a general practitioner, given 2 kinds of drops (Timolol□ and Catarlent□). Complaints aren't reduced, the patient does not seek treatment again.

Since about 2 months ago, sufferers complain of blurring of the right eye. Red eye (-), pain (-). Patients went to the local hospital, said there was bleeding in the right eye. Patients were referred to Mohammad Hoesin Hospital.

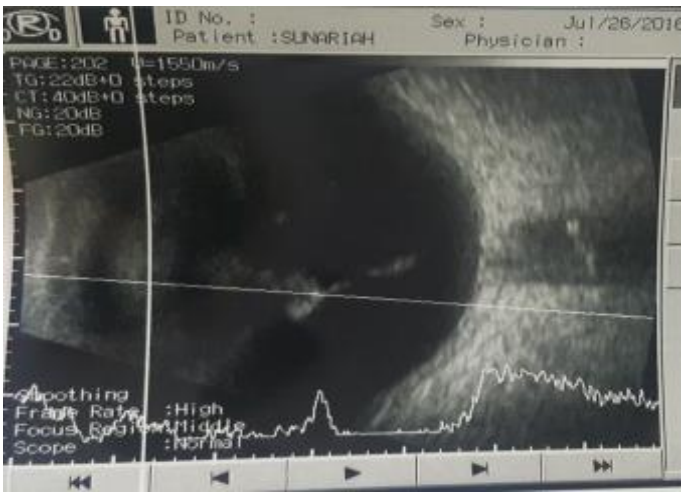
History of diabetes is denied, history of high blood pressure (+) since, 15 years ago, no regular treatment, history of wearing sunglasses is denied, history of trauma was previously denied, history of the same disease in the family is denied.

Physical examination generalist status General state: Good, Awareness: Compost mentis, Blood Pressure: 160/90 mmHg, Pulse: 82 x / minute, Respiration: 20 x / minute, Temperature: Afebris.

Ophthalmology Status Table

	OD	OS
Vision	2/60 pH (-)	6/30 pH (-)
TIO	15,6 mmHg	15,6 mmHg
KBM	Ortoforia	
GBM	Good in all directions	Good in all directions
Palpebra	Quiet	Quiet
Conjunctiva	Quiet	Quiet
Cornea	Clear	Clear
BMD	Medium	Medium
Sliced	Good Image	Good Image
Pupil	Round, Central, RC (+), Ø 3 mm	Round, Central, RC (+), Ø 3 mm
Lens	Turbid, subcapsular gr. II	Turbid, subcapsular gr. II
Posterior Segment	RFOD (+)	RFOS (+)
Papil Macula Retina	Detail difficult to be rate 	Round, firm boundary, normal red color, c / d: 0.3, a / v: 2/3 Fovea reflex (+) N Good blood vessel contour 

USG of the Right Eye Orbita



Retina: intact, Vitreus: echo-spike, choroid: not thickened. Impression: Vitreous hemorrhage OD

Diagnosis of bleeding from the vitreus OD, subcapsular cataract gr. II ODS. Management is informed consent, Pro pars plana vitrectomy + endo-laser OD, Pro laboratory check, Pro X-ray thorax PA, Pro Consultant Internal Medicine and Anesthesia. Prognosis of Quo ad vitam: Bonam, Quo ad functionam: Dubia ad bonam.

Discussion

Vitreous bleeding is one of the complications that threatens vision. The most common etiology of vitreous bleeding is diabetic retinopathy (39-54%), followed by retinal detachment without retinal detachment from RPE (12% -17%), *posterior vitreous detachment* (7.5-12.0%), *rhegmatogenous retinal detachment* (12% -17%) 7% -10%), and neovascularization that occurs in the retina due to occlusion complications from central or branch retinal veins (3.5% -10%).[2] The incidence of vitreous haemorrhage in branch retinal vein occlusion is very small, this is because in the retina branch disease the decrease in vision is quite mild and 50% of cases can be spontaneous resolution, so the patient does not seek treatment. Nevertheless, there are some cases that develop complications that threaten vision.[4]

In this case report, a 59-year-old woman with a history of 15 years of hypertension had complaints that her right eye vision had blurred in the past 2 months. Previously the patient complained that his vision had been felt like this but not too severe in the previous 2 years. The right eye vision of 2/60 cannot be corrected and the left eye vision of 6/30 cannot be corrected, with a grade 2 posterior subcapsular lens cloudy. On examination of the posterior segment of the right eye the detail is difficult to assess. In this patient suspected turbidity of the vitreous cavity so that an ultrasound examination was performed. On ultrasound examination the vitreous appeared echo-spike with the appearance of vitreous bleeding. In patients with hypertension, blood vessels will experience several pathophysiological changes in response to an increase in blood pressure. Retinal blood vessels will experience vasoconstriction in general at an early stage, which is an autoregulatory mechanism that should act as a protective function. On fundoscopic examination a narrowing of the retinal arterioles will be seen.[16-19]

Increased blood pressure for a long time causes thickening of the arteries intima, hyperplasia of the tunica wall of the media and hyaline degeneration. At this stage there will be more severe arteriolar constriction and changes in the crossing of the arteries known as arteriovenous crossing. In more severe circumstances retinal vein occlusion can occur. In branch retinal vein occlusion, occlusion occurs in the intersection of arteries and veins where arteries and veins have the same adventitial sheath. Thicker and less flexible artery walls cause compression on the veins which causes turbulent flow in the veins. Turbulent flow that occurs together with endothelial damage facilitates the formation of thrombus.[1,2] The symptoms that arise due to obstruction of the retinal branch vein, in the form of a calm eye vision dropped suddenly. These symptoms are likely experienced by patients in the previous 2 years. However, unilateral symptoms with a non-significant decrease in vision do not make patients to seek further treatment. In the case of BRVO spontaneous resolution can occur as much as 50-60%.[2,4.18]

Vitreous bleeding is an indication for vitrectomy. Intraoperatively after the vitrectomy, bleeding and ghost vessels were found in the super-temporal region. Two-thirds of BRVOs occur in the super-temporal quadrant because in this region there is an increase in the number of arteriovenous crossings where occlusion often occurs. Ghost vessel is a chronic sign of BRVO due to chronic occlusion, giving a picture of an unoccupied blood vessel.[2,19]

Bleeding in the super-temporal quadrant laser photocoagulation was performed. The basic principle of photo-coagulation is the occurrence of coagulation in tissues due to exposure to strong light rays. This laser mechanism destroys adjacent photoreceptors in the ischemic area of the retina

and is replaced by glial scar so that oxygen consumption in the outer retina is reduced. Oxygen that usually diffuses from the chorio-capillary to the retina decreases, and immediately diffuses through the laser scar in the photoreceptor layer without being consumed by the mitochondria in the photoreceptors. Furthermore, the flow of oxygen into the inner retina with the intention of alleviating hypoxia and increasing oxygen tension and vasoconstriction occurs so that arteriolar resistance increases, hydrostatic pressure in the capillaries decreases and edema formation will decrease.[2,19]

At the first day of follow-up, the patient's vision was increased to 6/60 with a posterior segment that could be assessed, a tortous vein was obtained, a small amount of bleeding and a *ghost vessel* in the super-temporal area with laser lesions. Vision improvement in these patients is due to vitreous bleeding that has been removed so as to open the visual axis.2 The prognosis of quo ad vitam is dubia ad bonam, because vitreous bleeding has not yet caused traction on the retina so that vitrectomy with the addition of lasers in the ischemic area can improve vision. However, patients are advised to keep blood pressure under control, ischemic processes in blood vessels including blood vessels in the retina can happen again.[4,19]

Conclusion

Branch retinal vein occlusion (BRVO) generally does not require therapy because most cases experience spontaneous resolution. Other cases that are not treated can experience several complications, the most common of which is macular edema and vitreous bleeding. The main goals of therapy in BRVO are to improve hemodynamics, overcome macular edema, and prevent neovascularization. In this case report, patients with BRVO with complications of vitreous bleeding undergo vitrectomy with an additional endo-laser in the ischemic area. The results of this action vision improvement in patients.

References

1. Retinal vein occlusion (RVO) guidelines. The Royal College of Ophthalmologists. London. 2015.Retina and Vitreous. American Academy of Ophthalmology. Canada. 2014-2015.
2. J.Q. Zhou, et al. The 10-year incidence and risk factor of retinal vein occlusion the Beijing eye study. Ophthalmology. 120: 803-808. 2013.

3. Branch vein occlusion study group. Argon laser scatter photocoagulation for prevention of neovascularization and vitreous hemorrhage in branch vein occlusion. A randomized clinical trial. Arch Ophtalmol. 104: 34-41. 2008.
4. Fundamentals and Principles of Ophthalmology. American Academy of Ophthalmology. Canada. 2014-2015.
5. Retinal Vascular Disease. Springer. New York, USA. 2007.
6. Rehak J, Rehak M. Branch retinal vein occlusion: pathogenesis, visual prognosis, and treatment modalities. Curr Eye Res. 33: 111–131. 2008.
7. Rogers S, et al. The prevalence of retinal vein occlusion: pooled data from population studies from the United States, Europe, Asia, and Australia. Ophthalmology. 117: 313–319. 2010.
8. Klein R, et al. The epidemiology of retinal vein occlusion: the Beaver Dam Eye Study. Trans Am Ophthalmol Soc. 98:133–141. 2010.
9. David R, et al. Epidemiology of retinal vein occlusion and its association with glaucoma and increased intraocular pressure. Ophthalmologica.197: 69–74. 2008.
10. Lim LL, et al. Prevalence and risk factors of retinal vein occlusion in an Asian population. Br J Ophthalmol. 92:1316–1319. 2008.
11. Zhao J, et al. Arteriovenous crossing patterns in branch retinal vein occlusion. The Eye Disease Case-Control Study Group. Ophthalmology. 100: 423–428. 2013.
12. Jefferies P, Clemett R, Day T. An anatomical study of retinal arteriovenous crossings and their role in the pathogenesis of retinal branch vein occlusions. Aust N Z J Ophthalmol. 21:213–217. 2003.
13. Christoffersen NL, Larsen M. Pathophysiology and hemodynamics of branch retinal vein occlusion. Ophthalmology. 106: 2054–2062. 2009.
14. Fraenkl SA, et al. Retinal vein occlusions: the potential impact of a dysregulation of the retinal veins. EPMA J. 1:253–261. 2014.
15. Petr Kolar. Risk Factors for Central and Branch Retinal Vein Occlusion: A Meta-Analysis of Published Clinical Data. Hindawi. 2014.
16. M.Rehakand, P.Wiedemann. Retinal vein thrombosis: pathogenesis and management. Journal of Thrombosis and Haemostasis. 8: 1886–1894. 2010.
17. J.W.Y.Yau, et al. Retinal vein occlusion: an approach to diagnosis, systemic risk factors and management. Internal Medicine Journal. 38: 904–910. 2008.

18. Jaulim, A. Et al. Branch retinal vein occlusion. Epidemiololgy, pathogenesis, risk factors, clinical features, diagnosis, and complications. An update of the literature. Retina. 33: 901-909. 2013.
19. Haller JA, et al. Dexamethasone intravitreal implant in patients with macular edema related to branch or central retinal vein occlusion twelve-month study results. Ophthalmology. 12: 2453 – 60. 2011.