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Abstract

Branch retinal vein occlusion (BRVO) usually results from a thrombus at the arteriovenous crossings where a thickened artery compresses the underlying venous wall, resulting in elevated venous pressure and consequent macular edema (ME). Intravitreal anti-vascular endothelial growth factor (VEGF) agents can treat ME in BRVO. However, refractory ME, the mechanism of which is not well elucidated, occurs frequently. Sixty-six eyes with ME secondary to BRVO were enrolled in this retrospective observational case-control study. Twenty eyes received a sub-Tenon's capsule injection of triamcinolone acetonide (STTA), 22 eyes an intravitreal anti-VEGF injection (ranibizumab), 16 eyes were switched from STTA to ranibizumab, 4 eyes underwent vitrectomy, and 4 eyes were untreated. Multiple regression analysis and multivariate logistic regression analysis were conducted, respectively, to identify independent predictors of visual acuity (VA) prognosis and risk factors for refractory ME longer than 1 year. The mechanism of refractory ME and therapeutic approaches for identified risk factors also were investigated. Thirty-four (52%) eyes had refractory ME for over 1 year. Microaneurysms were identified as risk factors for refractory ME, leading to poor final VA. Ranibizumab suppressed microaneurysm formation and refractory ME, with early administration more effective. For already formed microaneurysms, laser photocoagulation reduced additional treatments. Microaneurysms may cause refractory ME in BRVO. Alternative therapy to suppress microaneurysms should be considered to prevent refractory ME in patients with BRVO.