LETTER TO THE EDITOR

Perifoveal Microcirculation in Macular Oedema with Retinal Vein Occlusion

Hidetaka Noma*,1, Hideharu Funatsu1, Tatsuya Mimura2 and Katsunori Shimada3

1Department of Ophthalmology, Yachiyo Medical Center, Tokyo Women’s Medical University, 477-96, Owada-shinden, Yachiyo, Chiba 276-8524, Japan
2Department of Ophthalmology, University of Tokyo Graduate School of Medicine, 7-3-1, Hongo, Bunkyo-ku, Tokyo 113-8655, Japan
3Department of Hygiene and Public Health II, Tokyo Women’s Medical University, 8-1, Kawada-cho, Shinjuku-ku, Tokyo 162-8666, Japan

Keywords: Macular oedema, perifoveal capillary blood flow velocity, retinal vein occlusion.

TO THE EDITOR

Central retinal vein occlusion (CRVO) is a common retinal vascular disease that often results in macular oedema, which is the most frequent cause of visual impairment in these patients [1]. Therefore, it is important to understand the vascular and haemodynamic abnormalities that underlie the progression of macular oedema in CRVO. Remky et al. [2] found that the perifoveal blood flow velocity (BFV) was significantly reduced in CRVO patients compared with healthy subjects by using scanning laser ophthalmoscopy with fluorescein angiography (FA) to measure BFV in the perifoveal capillaries based on the movement of fluorescent dots. However, the relationship between perifoveal BFV and macular oedema in CRVO patients remains unclear. Accordingly, we investigated the relation between perifoveal capillary BFV and retinal thickness at the central fovea in CRVO patients with macular oedema, healthy volunteers, and patients who had branch retinal vein occlusion (BRVO) with macular oedema [3].

Seven CRVO patients with macular oedema were studied, including 4 men and 3 women with a mean age of 65.0 ± 4.9 years (Table 1). The fundus findings were confirmed by standardized fundus color photography and FA, which was performed with a Topcon TRC-50EX fundus camera, an image-net system (Tokyo Optical Co. Ltd., Japan), and a preset lens with a slit-lamp. Perifoveal BFV was measured by FA combined with scanning laser ophthalmoscopy and the tracing method [3]. Retinal thickness at the central fovea was measured by optical coherence tomography with the Zeiss-Humphrey apparatus (Zeiss-Humphrey Ophthalmic Systems, Dublin, California, USA). Each subject underwent complete ophthalmic evaluation. The data obtained were compared with findings previously reported for patients with BRVO and healthy volunteers [3]. Results are expressed as the mean ± standard error and the range. To compare BFV data among the groups, ANOVA and a Tukey-Kramer post hoc test were performed. To examine correlations between parameters, Spearman’s rank correlation coefficients were calculated by linear regression analysis. A two-tailed P value of less than 0.05 was considered to indicate statistical significance. This study was conducted according to the tenets of the Declaration of Helsinki, and was approved by the institutional review board. All subjects provided informed consent.

As shown in the Fig. (1), the mean perifoveal capillary BFV decreased significantly across the three groups of subjects from healthy controls (1.49 ± 0.11 mm/sec) to BRVO patients (1.08 ± 0.28 mm/sec) and then CRVO patients (0.78 ± 0.19 mm/sec) (p<0.001). There was a significant negative correlation between perifoveal capillary BFV and retinal thickness at the central fovea in the CRVO patients (r=−0.85, p=0.014).

This study showed that the perifoveal capillary BFV was significantly lower in patients with CRVO than in healthy persons and BRVO patients. This difference may have been detected because functional and/or anatomic capillary closure is more advanced in CRVO. It is possible that a reduction of BFV in CRVO leads to functional vascular obstruction and relative retinal ischaemia, resulting in the increased production of molecules like vascular endothelial growth factor (VEGF), which potently increases vascular permeability [4]. VEGF also increases the expression of intercellular adhesion molecule-1 (ICAM-1) by cultured endothelial cells, leading to the rolling, adhesion, and migration of leukocytes [5]. Accordingly, it can be suggested that chronic trapping of leukocytes in the capillaries of CRVO patients may lead to impaired perfusion, resulting in the overexpression of VEGF and ICAM-1 along with the development/progression of macular oedema. Thus, VEGF...
and ICAM-1 may both play a role in reducing the BFV of CRVO patients with macular oedema.

In conclusion, a decrease of perifoveal capillary BFV may have a role in the pathogenesis of macular oedema in patients with CRVO.

ACKNOWLEDGEMENT

Declared none.

CONFLICT OF INTEREST

Declared none.

PROPRIETARY INTEREST

The authors do not have any proprietary interest in this manuscript.

GRANTS AND FUNDS

The authors have not received any financial support.

REFERENCES

[1] Gutman FA, Zegarra H. Macular edema secondary to occlusion of the retinal veins. Surv Ophthalmol 1984; 28(Suppl): 462-70.
[2] Remky A, Wolf S, Knabben H, Arend O, Reim M. Perifoveal capillary network in patients with acute central retinal vein occlusion. Ophthalmology 1997; 104: 33-7.
[3] Noma H, Funatsu H, Sakata K, et al. Macular microcirculation and macular oedema in branch retinal vein occlusion. Br J Ophthalmol 2009; 93: 630-3.
[4] Dvorak HF, Brown LF, Detmar M, Dvorak AM. Vascular permeability factor/vascular endothelial growth factor, microvascular hyperpermeability, and angiogenesis. Am J Pathol 1995; 146: 1029-39.
[5] Lu M, Perez VL, Ma N, et al. VEGF increases retinal vascular ICAM-1 expression in vivo. Invest Ophthalmol Vis Sci 1999; 40: 1808-12.