



COLOUR DOPPLER MAPPING AND OCTO-ANGIOGRAPHY IN THE DIAGNOSIS OF GLAUCOMA

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ABSTRACT

Atherosclerosis is a systemic disease affecting arteries of different calibers. The development of atherosclerotic plaques and intima thickening contribute to the reduction of the vessel lumen that against the background of changes in the rheological properties of blood leads to perfusion disorders and ischemia of various organs including the eye.

Relevance. The involvement of vascular factors in the pathogenesis of glaucoma has been discussed in the literature for many years. A number of studies have shown that vascular factors play a crucial role in the pathophysiology of glaucoma with the claim that low ocular blood flow may cause damage to retinal ganglion cells. There is ample evidence of reduced ocular blood flow in glaucoma. However, the exact relationship between ocular haemoperfusion, visual field loss and structural damage remains controversial. One of the main requirements in ocular haemoperfusion research is an accurate and reliable measurement of blood flow. Of the 200 publications available in the literature over the last 15 years, 140 of them report data obtained by colour Doppler mapping (CDC). This method allows visualisation of blood flow in small vessel diameters when colour is superimposed on a two-dimensional image. A recently emerged method of OCT-angiography (OCTA) based on determining

the degree of decorrelation of consecutive B-scans (split-spectrum amplitude-decorrelation angiography, SSADA) allows to measure retinal and chorioidea blood flow in both peripapillary and macular areas [5]. However, OCT angiography in glaucoma is poorly understood.

Objective: To assess the state of retinal macular hemoperfusion and retrobulbar blood flow in patients with primary open angle glaucoma using optical coherence tomography with angiography (OCT-A) and colour Doppler mapping (CDC).

Material and Methods: The study was conducted in 65 eyes of patients with primary open-angle glaucoma (POAG), including 38 eyes with primary glaucoma and 27 with advanced and advanced stages. The control group consisted of 22 eyes of healthy subjects of similar age without ophthalmopathy. Glaucoma was diagnosed on the basis of typical changes of DZN detected by ophthalmoscopy (abnormal neural rim proportions, glaucoma EDZN, peripapillary atrophy,



wedge-shaped defects in retinal nerve fiber layer adjoining the DZN edge, hemorrhages along the DZN edge). The results of standard automated perimetry were reduced. Those patients who had previously used antiglaucoma drops were advised to discontinue them for up to 3 weeks (drug washout period), the remaining patients had newly detected glaucoma. The control group consisted of individuals with no first-line relatives with glaucoma, corneally compensated intraocular pressure (IOPcc) < 22 mmHg, unchanged RGN, normal retinal nerve fiber layer and no visual field defects. The inclusion criteria included emmetropic refraction and open AOC, as confirmed by anterior segment optical coherence tomography (Visante OCT, Zeiss), with an anterior chamber angle of at least 30° being acceptable. Only patients who had not previously undergone eye surgery were included in the analysis. The examination was performed in the macular zone by spectral optical coherence tomography (SD-OCT) on the RtVue xR Avanti with

Two vascular plexuses were studied: superficial, located in the retinal nerve fiber layer starting 3 µm below the surface of the inner boundary membrane and up to 15 µm below the inner plexiform layer (IPL), and deep, localized in the retinal layer between 15 and 70 µm below the IPL. In the para- (0.6 to 2.5 mm) and perifoveolar (2.5 to 5.5 mm) of the fovea two indices were measured: vascular area (Flow Area) and blood flow index (Index) or averaged value of the amplitude decoherence value. Colour Doppler mapping (CDC) with pulse Dopplerography using multifunctional ultrasound diagnostic device Voluson 730 ProSystem

with 10 to 16 MHz linear transducers was used to assess blood flow in the eye vessels and retrobulbar space. Blood flow in the ocular artery (VA), central retinal artery (CRA), central retinal vein (CRV), medial and lateral posterior short ciliary arteries (PSCA), vorticular veins (VV), superior ocular vein (OVV) was studied. The following quantitative blood flow indices were determined: maximal systolic velocity (V syst), final diastolic velocity (V diast), mean velocity during cardiac cycle (V mean), resistance index (RI) and pulsatility index (PI). The exact two-sided Wilcoxon-Mann-Whitney rank sum test was used. Indicators with a P-value<0.05 were considered statistically significant. Statistical analysis was performed using the SPSS version 21 statistical package and the MASS library of R.

RESULTS: The structural and functional indices were significantly different between the examined groups. Mean GCC (retinal ganglion cell complex) thickness was reduced in glaucoma patients compared to normals: 71.62±10.22 and 92.59±7.49 µm, respectively (p=0.33). FLV (focal loss volume index of retinal ganglion cell complex) was higher in eyes with glaucoma: 6.39±2.03 and 0.79±1.11% (p<0.001), respectively, as well as GLV (global loss volume index of retinal ganglion cell complex): 18.31±8.23 and 5.94±5.43% respectively (p<0.001). All blood flow parameters were reduced in glaucoma compared to healthy subjects: Index superficial parafovea POUG I 0.031±0.017 compared to healthy subjects 0.044±0.011 (p=0.002), compared to POUG II-III 0.026±0.019 (p=0.160) respectively; Index superficial perifovea POAG I 0.027±0.014 compared to healthy individuals 0.042±0.012 (p<0.001),



compared to POAG II-III 0.020 ± 0.011 ($p=0.063$) respectively; Flow superficial parafovea area of POUG I 1.66 ± 0.76 and 2.53 ± 0.53 mm² ($p=0.001$) compared to POUG II-III 1.44 ± 0.94 mm² ($p=0.296$) respectively. For the deep retinal layers, the indices were as follows: Index deep parafovea POUG I 0.017 ± 0.015 and 0.031 ± 0.015 ($p<0.001$) compared to POUG II-III 0.017 ± 0.003 ($p=0.800$), Index deep perifovea POUG I 0.016 ± 0.016 and 0.030 ± 0.015 ($p<0.001$) compared to POUG II-III 0.012 ± 0.008 ($p=0.539$), Flow deep parafovea area in POAG I 1.06 ± 0.89 and 1.97 ± 0.82 mm² in healthy group ($p<0.001$) compared to POAG II-III 0.96 ± 0.92 mm² ($p=0.646$) respectively. Significant differences between OCTA parameters were obtained comparing eyes with primary glaucoma and healthy eyes, while differences between glaucoma stages (primary to advanced stages) were generally not significant.

Retrobulbar blood flow results showed less significant differences between groups except for data on diastolic blood flow velocity in the CVC and WCCA as well as in the CAC. In addition, there was a significant difference in diastolic blood flow velocity in the ocular artery between patients with initial POAG and healthy subjects.

Significant decrease of blood flow in posterior short lateral ciliary arteries (PSCA Lat.) in systole 12.76 ± 3.76 cm/s in POAG I group compared to healthy subjects 14.38 ± 1.82 cm/s ($p=0.004$), in POAG II-III 11.09 ± 2.63 cm/s ($p=0.005$) and in diastole 4.95 ± 1.96 cm/s in POAG I group and 5.17 ± 1.15 cm/s in healthy group ($p=0.005$), compared to POAG II-III 3.46 ± 1.58 cm/s ($p=0.006$) and posterior short medial ciliary arteries (PSCA med.) in systole 11.6 ± 2.86 cm/s in POAG I group

and 13.83 ± 2.23 cm/s in healthy group ($p=0.004$), respectively, compared to POAG II-III 10.43 ± 2.39 cm/s ($p=0.007$). Decreased venous blood flow velocity was also obtained: Systolic velocity (V syst) in the CVC in POUG I 8.1 ± 3.5 cm/s compared to the healthy subjects group 10.5 ± 3.6 cm/s ($p=0.009$), compared to POUG II-III 8.0 ± 3.0 cm/s ($p=0.612$), diastolic velocity (V diast) in CVS in POUG I 5.2 ± 2.1 cm/s and 7.1 ± 2.5 cm/s ($p=0.011$), compared to POUG II-III 5.1 ± 2.4 cm/s ($p=0.472$). Systolic velocity (V syst) in CAC in POAG I was 13.7 ± 2.7 cm/s ($p=0.074$) compared to healthy group 12.4 ± 1.8 cm/s ($p=0.074$), compared to POAG II-III 10.7 ± 2.6 cm/s ($p<0.001$). Diastolic velocity (V diast) in CAC in POUG I 4.2 ± 0.9 and 4.3 ± 1.1 cm/s in healthy group ($p=0.277$), compared to POUG II-III 3.2 ± 1.5 cm/s ($p=0.001$). Mean blood flow velocity (V mean) in CAC in POAG I was 7.8 ± 1.6 cm/s in healthy subjects ($p=0.037$), compared to POAG II-III 6.0 ± 1.6 cm/s ($p<0.001$).

Systolic velocity (V syst) in GA was 31.7 ± 7.7 cm/s in POUG I compared to healthy group 37.5 ± 11.1 cm/s ($p=0.011$), compared to POUG II-III 33.7 ± 11.9 cm/s ($p=0.590$). Diastolic velocity (V diast) in GA was 8.0 ± 3.2 cm/s in POUG I and 11.3 ± 5.6 cm/s ($p=0.004$), compared to POUG II-III 7.8 ± 3.7 cm/s ($p=0.957$). Mean blood flow velocity (V mean) in GA in POUG I was 14.9 ± 4.6 and 19.2 ± 7.4 cm/s ($p=0.008$), compared to POUG II-III 15.6 ± 6.1 cm/s ($p=0.536$).

Discussion: In the present study we found for the first time a decreased vascular density in the deep plexus already in primary glaucoma, with one of the parameters (index deep perifovea) being among the most informative to differentiate primary from normal



glaucoma. Our results showed a significant decrease in capillary density already at the initial stage of glaucoma not only in the superficial plexus supplying the GCS layer, but also in the deep vascular plexus as compared with healthy subjects of similar age. Notably, the difference in capillary density in both plexuses between primary and advanced glaucoma was less significant. However, the use of OCT-A due to its ability to perform segmented assessment of blood flow provided detailed information about the blood supply to the inner retinal layers in the macula. It may be

supposed that deterioration of trophicity in the aforementioned layers explains the involvement of the macula in the pathological process at early stages of glaucoma.

Conclusion. The results of this study showed a decrease of hemoperfusion in the macular zone in early stages of glaucoma. The high informative value of OCT-A method in the study of macular blood flow opens perspectives both in understanding of glaucoma pathogenesis and early diagnosis of the disease.

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