International Ophthalmology **16**: 273–276, 1992.

Color Doppler imaging in evaluation of optic nerve blood supply in normal and glaucomatous subjects

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Key words: color-Doppler imaging, glaucoma, ophthalmic artery, central retinal artery, ciliary arteries

Abstract

The authors evaluate by means of Color Doppler Imaging (CDI) the blood flow in ophthalmic artery, posterior ciliary arteries and central retinal artery in a group of glaucomatous patients. The data obtained in this study show a statistically significant reduction of the mean systolic peak flow velocity in ophthalmic artery in glaucomas in comparison with controls. In Glaucomatous patients with uncontrolled IOP there was a reduction of end-diastolic flow velocities and a rise of resistivity index in ciliary arteries and central retinal artery.

Introduction

Doppler ultrasonography can be succesfully used to study the blood supply of the ocular tissues in glaucoma [1–4, 13]. A recent development of ultrasonography is represented by Color Doppler Imaging (CDI) [16] in which the informations about the blood flow are superimposed in color on the standard B-Mode echographic image.

One of the advantages of this technique is the possibility of localizing and following small vessels which cannot be resolved on the grey scale image.

In some recent works, the CDI was used to visualize the vascular system of the eye and the orbit particularly considering the ophthalmic artery and some of its branches, evaluating the main features of their blood flow both in normal and in some vascular disorders [5, 6, 8, 11, 12].

Aim of our work was to investigate by means of this technology a group of patients suffering from chronic simple glaucoma, considering the velocity-time spectra of the blood flow in the ophthalmic artery, in the central retinal artery and in the posterior ciliary arteries in order to evaluate the local circulatory condition in glaucomatous optic nerve disease [9, 10].

Subjects and methods

In our study we examined 40 eyes of 20 patients (11 women and 9 men) suffering from chronic simple glaucoma; their mean age was 63 years (range 39–78).

Four eyes were operated on with trabeculectomy according to Cairns; the other eyes were treated with instillation of Beta-blockers alone (22 eyes) or associated with parasympathomimetics (14 eyes).

At the time of the CDI examination, 32 eyes were in good tonometric compensation (mean IOP = 16 mmHg, range 10–20 mmHg), 8 eyes had a therapy-resistant abnormal IOP (mean IOP = 31 mmHg, range: 27–36 mmHg).

Thirty six eyes showed mild or moderate alterations of the visual field (blind spot enlargement, relatives or little absolute defects in the paracentral

Table 1. Flow velocities (cm/s) and R.I.

		Ophthalmic artery			Ciliary artery			Central retinal artery		
		Syst.	Diast.	R.I.	Syst.	Diast.	R.I.	Syst.	Diast.	R.I.
Glaucomas	Mean	36,9	9,6	0,75	12,2	2,9	0,75	12,3	1,5	0,89
	St.Dev.	4,5	2,5	0,07	2,6	0,9	0,1	3,2	1,9	0,1
Normals	Mean	41,3	9,5	0,76	12,5	3,1	0,71	13,4	2,0	0,85
	St.Dev.	3,4	2,0	0,04	1,7	0,4	0,07	2,4	1,8	0,08
	p	0,001	n.s.	n.s.	n.s.	n.s.	0,1	n.s.	n.s.	0,1

area), while only 4 eyes showed severe damage (wide absolute defects), measured on a computerized perimeter Octopus 2000.

The control group consisted of 40 normal eyes belonging to 20 normal, age-comparable, subjects (mean age = 62 years, range 38–76 years), 13 women and 7 men.

None of the subjects we studied had a history of ocular or systemic vascular diseases that could interfere with our study.

We obtained informed consent from every subject. After measurement of IOP, the eyes were examined by CDI to evaluate ophthalmic artery, central retinal artery and posterior ciliary arteries.

All examinations were performed with a color Doppler unit (QUAD 1, Quantum Medical Systems Inc, Issaquah, Wash) supplied with a 7,5 MHz linear-phased array transducer, using an high-medium flow setting that allows the detection of high and medium velocity flows (from 3,3 m/s to 1,5 cm/s).

In every patient we also performed a CDI evaluation of the carotid arteries to exclude the presence of hemodynamically significant stenosis that could influence the ophthalmic artery flow.

Furthermore, patients with refractive errors higher then 3 spheric diopters and 1.5 astigmatic diopters were excluded from our series.

For every vessel we measured peak systolic (S.V.), end-diastolic (D.V.) flow velocities and the resistivity index of Pourcelot (R.I.) [15], i.e. (S.V. – D.V.) / S.V.

As one can easily realize when observing this formula, the R.I. mainly depends upon the end-diastolic flow velocity, which in turn is inversely proportional to peripheral resistances.

The statistical significance of the average values between the studied groups was evaluated by means of Student's t-test.

Results

We could identify and study the ophthalmic artery in all our patients. Using our technology, the ciliary arteries and the central retinal artery could be found respectively in 36 and 37 eyes in normals and in 32 and 36 eyes in glaucomas.

In Table 1 the mean values of systolic and diastolic velocities and of Pourcelot resistivity index of the ophthalmic artery, the ciliary arteries and the cen-

Table 2. Flow velocities (cm/s) and R.I.

Glaucomas		Ophthalmic artery			Ciliary artery			Central retinal artery		
		Syst.	Diast.	R.I.	Syst.	Diast.	R.I.	Syst.	Diast.	R.I.
Controlled	Mean	36,8	9,5	0,74	12,4	3,5	0,71	12,2	1,7	0,87
	St.Dev.	4,7	2,8	0,08	2,5	1,0	0,07	2,1	1,3	0,09
Uncontrolled	Mean	35,9	9,4	0,75	11,9	1,2	0,86	12,7	0,9	0,94
	St.Dev.	4,9	3,1	0,06	2,7	1,1	0,09	1,2	0,6	0,08
	p	n.s.	n.s.	n.s.	n.s.	0,001	0,001	n.s.	0,05	0,05

tral retinal artery for normals and glaucomas are shown.

In Table 2 the average values of the same parameters, obtained in the glaucomatous population, comparing the patients with well controlled IOP levels and those who were therapy-resistant with abnormal IOP are shown.

Finally, in glaucomas, the comparison of our results with the amount of visual field loss did not show any significant correlation with the parameters used.

Discussion

Comparing the controls with the whole group of glaucomatous patients, we could not find any significant difference for the blood flow velocity of the ciliary arteries and the central retinal artery, while the peak systolic velocity of the ophthalmic artery was significantly lower ($p < 0.001$) in glaucomas.

The average decrease of systolic velocity in ophthalmic arteries in glaucomas is not associated with a similar reduction of the diastolic velocity, as clearly demonstrated by the normal values of the resistivity index in these vessels.

On the contrary, in ciliary and central retinal arteries the resistivity index is slightly higher in glaucomas then in controls, albeit with very poor statistical significance. The comparison of well controlled glaucomatous patients with those with abnormal IOP provided some further useful informations. In fact, in the 8 eyes with therapy-resistant glaucoma and elevated IOP, we observed in the ciliary and central retinal arteries a statistically significant decrease of the mean end-diastolic flow velocities and a significant increase of the mean resistivity index values.

In these patients we could constantly observe a decrease of the flow which often disappeared in the end-diastolic phase; this phenomenon can be assumed as an expression of an increase of the peripheral resistance in the vascular district supplied by the ciliary and central retinal arteries.

In our study the eyes with uncontrolled glaucoma are few; for this reason these data scarcely influence the results of the whole glaucomatous group. In conclusion, we would like to point out the most interesting results arising from our work:

1. The average decrease of the systolic velocity in the ophthalmic artery in the group of patients suffering from chronic simple glaucoma: this result cannot be easily interpreted but it might represent a generic vascular 'risk factor' for the onset of glaucomatous optic nerve disease.

2. The decrease of the end-diastolic velocity in central retinal and ciliary arteries with the parallel increase of the resistivity indices in the glaucomas with abnormal IOP: this phenomenon might depend on a direct or indirect mechanical effect of ocular hypertension on the vascular walls with a consequent slowing of the blood flow. This might have particularly serious haemodynamic implications on the ciliary circulation which is a low-resistance vascular district and shows a poor autoregulation [10].

As the ciliary system is the main source of optic nerve blood supply, our data could suggest that the abnormal increase of IOP in glaucomas could lead to the glaucomatous atrophy through an ischemic mechanism with the reduction of optic nerve blood perfusion.

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