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CLINICAL INVESTIGATION

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Visual field defect and perfusion of the juxtapapillary retina and the neuroretinal rim area in primary open-angle glaucoma

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Abstract ● Background: At this time little information is available about the relationship between glaucomatous visual field defects and impaired blood flow in the optic nerve head. The purpose of this study was to examine blood flow of the juxtapapillary retina and the rim area of the optic nerve head in primary open-angle glaucoma with a borderline visual defect. ● Methods: Juxtapapillary retinal and neuroretinal rim area blood flow was measured by scanning laser Doppler flowmetry (SLDF). The visual field was evaluated by static perimetry (Octopus-G1). The optic nerve head was assessed on 15° color stereo photographs. We examined 116 eyes of 91 patients with POAG with controlled IOP and 66 eyes of 44 healthy individuals. The POAG group was divided into eyes with a mean defect lower than 2 dB (POAG group I) and in eyes with a mean defect equal to or greater than 2 dB (POAG group II). The mean age of POAG group I and POAG group II was 55±11 years and 57±10 years, respectively. The mean age of the control group was 45±15 years. The eyes of POAG group I had an average C/D ratio of 0.71±0.18 with an average mean defect of the visual field of 0.97±0.68 dB; the eyes of POAG group II had an average C/D ratio of 0.80±0.17 with an average mean defect of the visual field of 8.2±6.0 dB. The intraocular pressure on the day of measurement in POAG group I was 18.2±3.7 mmHg, in POAG group II 17.6±4.0 mmHg, and in the control group 15.1±2.5 mmHg. For statistical analysis, age-matched groups of 32 normal eyes of 32 subjects (mean age 52±10 years) were compared to 18 glaucomatous eyes of 18 patients (POAG group I, mean age 55±11 years) and 59 glaucomatous eyes of 59 patients (POAG group II, mean age 55±10 years). ● Results: In the eyes of POAG group I and POAG group II, both juxtapapillary retinal blood flow and neuroretinal rim area blood flow were significantly decreased compared to an age-matched control group: neuroretinal rim area "flow" POAG group I –65%, POAG group II –66%; juxtapapillary retina "flow" POAG group I –52%, POAG group II –44%. All eyes of the POAG group I (MD<2 dB) and 56 of 61 eyes of the POAG group II (MD>=2 dB) showed a retinal perfusion lower than the 90% percentile of normal blood flow. We found no correlation between reduction of juxtapapillary or papillary blood flow and mean defect in POAG eyes. ● Conclusion: Glaucomatous eyes with no defects or borderline visual field defects as well as glaucomatous eyes in an advanced disease stage show significantly decreased optic nerve head and juxtapapillary retinal capillary blood flow.

Introduction

The pathogenesis of glaucoma is still unknown in detail, but there is some reason to believe that glaucoma is associated with ocular and systemic vascular changes [7, 18, 34]. Defective autoregulation of the optic nerve head [31] and the retina [11], arterial hypertension and nocturnal hypotension [13], and increased intraocular pressure may be risk factors for developing glaucomatous optic nerve atrophy. There is some evidence that glaucomatous optic nerve atrophy is associated with decreased ocular macro- and microcirculation [9, 12, 19, 22, 29, 32, 33, 36]. A quantitative correlation was described between measurements of the disc rim and measurements of visual function in glaucoma [1–5, 35]. At this time little information is available about the relationship between glaucomatous visual field defects and impaired blood flow in the optic nerve head. The purpose of this study was to examine blood flow of the juxtapapillary retina and rim area of the optic nerve head in primary open-angle glaucoma with no or borderline visual defect.

Subjects and methods

Retinal and optic nerve head blood flow was measured by scanning laser Doppler flowmetry (SLDF, Heidelberg Retina Flowmeter), which is a combination of a *laser Doppler flowmeter* and a *scanning laser device* known from laser scanning ophthalmoscopes. SLDF permits measurement and visualization of perfusion of the superficial capillaries of the juxtapapillary retina and optic nerve head. The principles of function have been described in detail elsewhere [10, 20, 21, 23–26]. The parameters for the measurements by SLDF were: laser wave length 670 mn, observation angle 10°, depth of measurment 300 µm, pixel size 10×10 µm, focus at the superficial retinal layer. Retinal and optic nerve head blood flow was quantified in sample areas 100 ×100 µm, which were located away from ophthalmoscopically visible vessels. For evaluation of the juxtapapillary retina, we chose two nasal and two temporal sample areas in the equator line, in each case 0.5 and 1 mm away from the neuroretinal rim. The blood flow of the optic nerve head was measured at the neuronal rim, locating the area of interest with a sample area 100×100 µm at the rim of the optic nerve head. Figure 1 depicts the areas of measurement by SLDF.

Criteria for the diagnosis of glaucoma were at least two intraocular pressure readings higher than 21 mmHg and glaucomatous changes of the optic nerve head [14, 16, 27, 28]. The intraocular pressure was measured in at least one 24-h pressure profile, including four intraocular pressure measurements between 5 p.m. and 7 a.m. the optic nerve head was assessed on 15° color stereo photographs, without knowledge of the results of the SLDF measurements or visual field tests. The transparencies were projected in a scale of 1–15 and the outlines of the optic disc and optic cup, determined by contour of the vessels and not by pallor, were plotted on paper and morphometrically analyzed. For the morphological classification of the optic disc, quantitative and qualitative criteria were used such as an unusually small neuroretinal rim area in relation to the optic disc size, cup-to-disc ratios that were higher vertically than horizontally, an abnormal shape of the neuroretinal rim, the appearance of splinter-shaped optic disc hemorrhages, and reduced visibility of the retinal nerve fiber bundles, including localized defects. This method has been described in detail previously [17]. Early glaucomatous damage of the disc is predominantly characterized by loss of the neuroretinal rim at the inferior and superior parts of the optic nerve head. This results in a loss of normal configuration of the neuroretinal rim, first with a equal width of the rim, followed by a configuration with the neuroretinal rim being smaller at the superior and inferior disc poles than nasally and temporarlly, or with a notch, and finally only a remnant of the rim in the nasal part of the disc. Finally, there is no rim in any part of the disc. According to this, five stages of glaucomatous optic nerve head atrophy were defined (*stage 0:* normal configuration; *stage I:* loss of the normal configuration of the neuroretinal rim, equal width of the rim; *stage II:* neuroretinal rim smaller at the superior and inferior disc poles than nasally and temporally, notch, nerve bundle defects; *stage III:* remnant of the rim in the nasal and temporal parts of the disc; *stage IV:* remnant of the rim in the nasal part of the disc; *stage V:* no visible rim).

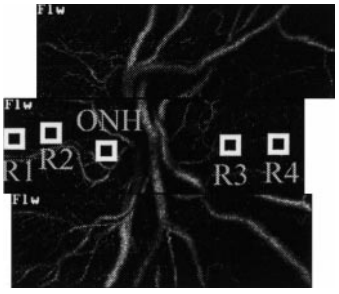

Fig. 1 Area of interest for blood flow measurements by SLDF of the nasal and temporal juxtapapillary retina and rim area. *R1* nasal retina 1, *R2* nasal retina 2, *ONH* Optic nerve head, *R3* temporal retina 3, *R4* temporal retina 4

The visual field was tested with the Octopus program G1 at least twice. The parameter „mean defect“ of this program with a normal range from +2 to −2 dB was used in calculation of the results. Visual field parameters, however, were not used in estabishing the diagnosis of glaucoma.

To examine the optic nerve head and for other measurements in patients with miosis, the pupil was dilated by tropicamid. For the measurement of retinal blood flow and visual field the diameter of the pupil was greater than 2 mm and lower than 4 mm.

We examined 116 eyes of 91 patients with POAG with controlled IOP. The POAG group was divided into eyes with a mean defect lower than 2 dB (POAG group I, n=27 eyes of 18 patients) and in eyes with a mean defect equal to or greater than 2 dB (POAG group II, n=89 eyes of 68 patients). The mean age of POAG group I and of POAG group II was 55±10 and 57±10 years, respectively. The mean disk area of POAG group I and of POAG group II was 2.85±0.85 and 2.59±0.60 mm^2, respectively. The mean rim area of POAG group I and POAG group II was 1.07±0.37 and 0.85±0.44 mm^2, respectively. The eyes of POAG group I had an average C/D ratio of 0.71±0.18 with an average mean visual field defect of 0.97±0.68 dB; the eyes of POAG group II had an average C/D ratio of 0.80±0.17 with an average mean visual field defect of 8.21±6.08 dB. Table 1 summarizes the morphological disc parameters of POAG patients.

The reported maximum intraocular pressure (IOP_{max}) in the medical history of POAG group I and POAG grup II was on average 29.3±10.3 and 31.0±9.5 mmHg, respectively. The IOP at the day of measurement in POAG group I and POAG group II was controlled (18.2±3.7 and 17.6±4.0 mmHg, respectively) for IOP-lowering topical drug therapy (β-blockers in 64 and 66.7%, respectively, α-adrenergic substances in 28 and 29.4%, respectively, cholinergic substances in 16 and 40.6%, respectively) and/or previous surgical treatment (laser trabeculoplasty in 14.8 and 35.3%, respectively, filtering operations in 11.1% and 18.8% respectively; see also Table 4). All patients were examined by slit-lamp examinations. No patients had pigmentary dispersion or lens exfoliation syndrome.

Table 1 Optic disc parameters

	POAG, MD<2 dB			POAG, MD>2 dB POAG			*P*-value
	POAG group 1			Group 2			
Rim area (mm^2)	1.08±0.37			0.85±0.45			n.s. ($P>0.05$)
Disc area (mm^2)	2.85±0.86			2.60±0.60			n.s. ($P>0.05$)
Excavation area (mm^2)	1.77±0.72			1.75±0.69			n.s. ($P>0.05$)
Morphological glaucoma stage[a]	1.75± 1.04	Stage 0	0%	1.86± 1.36	Stage 0	0%	
		Stage I	50%		Stage I	58.6%	
		Stage II	37.5%		Stage II	3.4%	
		Stage III	0%		Stage III	20.7%	
		Stage IV	12.5%		Stage IV	17.2%	
		Stage V	0%		Stage V	0%	
C/D ratio	0.71±0.18			0.81±0.17			$P<0.005$

[a] Stage 0: normal confirguration; stage I: loss of the normal configuration of the neuroretinal rim, equal width of the rim; stage II: neuroretinal rim smaller at the superior and inferior disc poles than nasally and temporally, notch, nerve bundle defects; stage III: remnant of the rim in the nasal and temporal parts of the disc; stage IV: remnant of the rim in the nasal part of the disc; stage V: no visible rim

Table 2 Ocular and systemic data

All eyes	No. of eyes	Mean blood pressure (mmHg)	IOP at the day of measurement (mmHg)	Reported maximum IOP (mmHg)	Mean defect (dB)	C/D ratio	Age (years)
POAG, <2 dB	27	120±21	18.4±3.5	29.3±10	0.94±0.65	0.71±0.18	55.2±10
POAG, >2 dB	89	121±18[a]	17.1±4.5	31.0±9.5	8.4±6.1	0.80±0.17	57.3±10
Control	66	111±19	15.1±2.2		–	0.52±0.18	44.5±14

Age-matched groups, one eye of one subjects	No. of subjects	IOP at the day of measurement	Mean defect	C/D-ratio	age
POAG, <2 dB	18	18.2±3.7	0.96±0.68	0.73±0.15	55.2±10
POAG, >2 dB	59	17.6±4.0	8.2±6.0	0.81±0.14	55.4±9.8
Control	32	15.1±2.5	–	0.50±0.17	51.7±10

[a] Significant difference from controlAge-matched

The control group consists of 66 eyes of 44 healthy individuals without any signs of optic nerve damage. The mean age of the control group was 45±15 years. The IOP at the day of measurement of the control group was 15.1±2.5 mmHg. The control group consisted of healthy subjects working in our department and patients with clear media and without any systemic disease or intraocular disease. The visual field was not tested in the control group. Table 2 summarizes the ocular and systemic findings of all groups.

To verify differences between the POAG groups and the control group non-parametric tests (Mann-Whitney-Wilcoxon test) were performed. For statistical analysis age-matched groups of 32 normal eyes of 32 subjects with 18 glaucomatous eyes of 18 patients (POAG group I) and 59 glaucomatous eyes of 59 patients (POAG group II) were compared.

Only one eye of one person was used for statistical analysis. The level of significance was reached when the *P*-value was lower than 0.05. Linear correlations between mean defect and juxtapapillary retinal and papillary perfusion were calculated by the Pearson correlation; *r*-value und *P*-value were determined.

Results

Juxtapapillary retinal and neuroretinal rim area blood flow in POAG and mean defect:

Eyes with primary open-angle glaucoma both in POAG group I and II showed significantly reduced blood flow of the juxtapapillary retina and neuroretinal rim area compared to controls (Table 3). Figure 2 showed the flow data of the five areas of measurement as box plots. No correlation was found between the juxtapapillary retinal blood flow and mean defect of the visual field. In eyes with a mean defect lower than 2 dB we observed a significant reduction of juxtapapillary retinal blood flow. Figure 3 depicts the juxtapapillary perfusion and the mean defect as a scatterplot. All eyes of POAG group I showed a retinal and rim area circulation lower than the

Table 3 Juxtapapillary and optic nerve head blood flow in POAG

All eyes	No. of eyes	Juxtapapillary retina	Optic nerve head rim area
POAG, <2 dB	27	224±30[a,b]	175±59[a]
POAG, >=2 dB	89	260±62[a]	170±78[a]
Control	66	471±89	514±126
Age-matched groups, one eye of one subjects	**No. of eyes**	**Juxtapapillary retina**	**Optic nerve head of one subject**
POAG, <2 dB	18	225±33[a]	181±66[a]
POAG, >=2 dB	59	258±63[a]	181±77[a]
Control	32	452±94	526±133

[a] Significant difference from control
[b] Significant difference from POAG group II

Table 4 IOP-lowering topical drug therapy and previous surgical treatment (in %)

	POAG, <2 dB	POAG, >=2 dB
β-Blocker substances	64%	66.7%
α-Adrenergic substances	28%	29.4%
Cholinergic substances preceded surgical treatment	16%	40.6%
Laser trabeculoplasty	14.8%	35.3%
Filtering operation	11.1%	18.8%

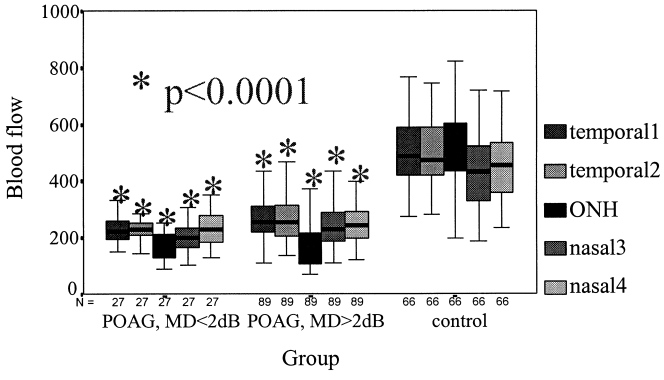

Fig. 2 Box blots of flow of the nasal, temporal juxtapapillary retina and rim area in POAG eyes and normal eyes

90% percentile of normal blood flow of the juxtapapillary retina.

Juxtapapillary retinal and neuroretinal rim area blood flow and C/D-ratio, IOP, and age:

The juxtapapillary retinal and rim area blood flow in eyes with POAG showed no correlation with the C/D ratio, with the reported maximal intraocular pressure, with the IOP at the day of measurement, or with age.

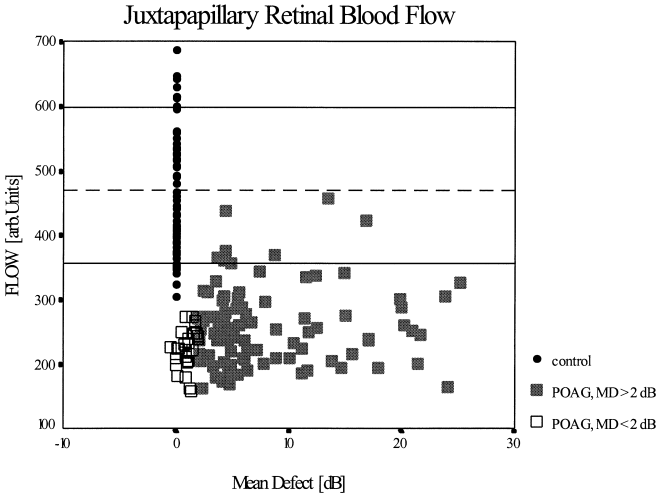

Fig. 3 Scattergram of juxtapapillary retinal blood flow versus mean defect. The reference lines show the 10% percentile, the 90% percentile, and the mean (*dotted line*) of the "Flow" of the control group

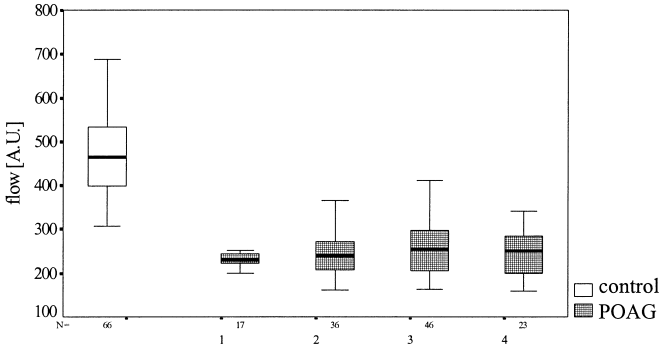

Fig. 4 Box blots of flow of the juxtapapillary retina comparing control and POAG subgroups (*1* no local therapy, *2* only β-blocker, *3* β-blocker and cholinergic drugs and/or adrenergic drugs, *4* cholinergic drugs and/or adrenergic drugs without β-blocker)

Discussion

The reliability was examined by performing five seperate perfusion measurements in ten eyes of ten healthy persons on 5 consecutive days. The blood flow was measured at the same retinal location of each eye. The coefficient of the reliability of "flow", "volume," and "velocity" were 0.82, 0.81, and 0.83, respectively.

In this cross-sectional study we correlated juxtapapillary retinal and rim area blood flow with the visual field defect. There is evidence that significant correlation exists between the optic disc rim area and the visual field index for overall loss of sensitivity (mean defect) [1–6, 8]. At this time little information is available about the relationship between glaucomatous visual field defect and impaired blood flow in the optic nerve head. As shown recently [26], we found significantly decreased capillary blood flow of the juxtapapillary retina and optic nerve head in POAG, confirming data by Wolf et al. [36] and Hamard et al. [12]. Wolf and coworkers evaluated the blood flow in retinal arterioles and venoles. They stated that eyes with POAG are associated with an increased arteriovenous passage time (+41%) and with decreased dye velocity (−11%). The blood flow in the optic nerve head was examined by Hamard and coworkers using single-point laser Doppler flowmetry. They found that eyes with POAG and LTG are associated with decreased capillary blood flow in the optic nerve head (−42% and −56%, respectively).

In this study we observed that POAG eyes with borderline visual defects were associated with significantly reduced juxtapapillary retinal and rim area perfusion. Capillary blood flow of the neuroretinal rim area and of the juxtapapillary retina in eyes with POAG showed *no* significant correlation with the mean defect of the visual field. Our measurements confirm an observation by Schweitzer and coworkers [33] that early stages of POAG are associated with a decreased concentration of oxyhemoglobin in the papillomacular bundle.

In the study presented all POAG patients in group 1 (MD<2 dB) showed glaucomatous optic nerve atrophy, diagnosed by a very experienced glaucoma specialist. POAG group 1 may reflect an early stage of glaucoma with glaucomatous optic nerve atrophy with a visual defect lower than 2 dB. The mean neuroretinal rim area in eyes with POAG with MD<2 dB was significantly smaller than the rim area of normal eyes. In a study published in 1988 the optic disc of 369 normal subjects was examined [15]. On average the rim area of normal subjects was 1.97±0.50 mm^2; the mean disc area was 2.69±0.70 mm^2. Thus, we may assume that in POAG group 1 all eyes with ocular hypertension without any glaucomatous changes of the optic nerve head were excluded. POAG group 2 reflects patients with a more progressed glaucoma stage, indicated by a visual defect of more than 2 dB.

We found no significant differences in juxtapapillary retinal and rim area blood flow with or without topical beta blocker therapy. There was no relationship between the use of adrenergic drugs, the kind of surgical therapy or blood flow reduction in eyes with POAG (Fig. 4). We found no effect of the topical antiglaucomatous drugs or previous surgical treatment on the retinal or optic nerve blood flow. Thus, the decreased juxtapapillary retinal and neuroretinal rim area blood flow found in eyes with POAG does not seem to be caused by topical drugs. We therefore assume that treatment with beta blockers or other adrenergic or cholinergic drugs has no effect on rim area or retinal capillary blood flow measured by SLDF.

We found no significant difference in mean defect (MD) in patients with or without therapy with cholinergic eye drops. Patients *with* cholinergic therapy had a mean defect of 6.6±5.2 dB, patients *without* cholinergic therapy revealed a mean defect of 6.0±6.5 dB. Thus, it is very probable that the observed difference in mean defect in groups I and II is not due to the miotic pupil.

Among the advanced cases we found seven eyes with normal flow. The cause for normal retinal blood in cases with advanced glaucoma remains unclear. One may speculate that in these cases the papillomacular bundle was less affected, leading to normal retinal blood flow readings in the equator line.

In conclusion, our observations suggest that POAG with controlled IOP is associated with impaired circulation in the juxtapapillary retina and neuroretinal rim area of the optic nerve head. Eyes with early stages of glaucoma with a mean defect lower 2 dB showed a significant reduction in juxtapapillary retinal and rim area blood flow.

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