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Ocular blood flow evaluation in injured and healthy fellow eyes

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PURPOSE. *To assess if injured eyes develop ocular blood flow disturbances that may contribute to development of traumatic glaucoma.*

METHODS. *Twenty-five eyes of 25 patients hospitalized from January 1997 to July 1999 for blunt (15) or penetrating (10) eye injury and elevated intraocular pressure (IOP) (>23 mm Hg) were controlled at least 24 months after the trauma and underwent visual field examination, pulsatile ocular blood flow (pOBF), and color Doppler imaging (CDI) analysis of ophthalmic artery, central retinal artery, nasal and temporal short posterior ciliary arteries. Uninjured healthy eye was used as control.*

RESULTS. *IOP was significantly higher in injured eyes (15.1±3.3 vs 13.0±2.7 mmHg; p<0.01), but only 2 eyes (8%) were under medical treatment. pOBF values were significantly lower in injured eyes: 11.25±6.56 µL/sec in the trauma eyes and 15.40±7.29 in fellow eyes (p=0.002). Resistivity index of all investigated retrobulbar vessels was very significantly higher in injured eyes than in fellow eyes (p<0.0001). There is no significant correlation between IOP and ocular blood flow disturbance.*

CONCLUSIONS. *Long-term follow-up (mean 39±12 months) of injured eyes shows, besides a slight but significant increase of IOP, a very significant impairment of ocular blood supply to injured eyes compared to healthy fellow eyes with reduction of pulsatile ocular blood flow and marked increase of resistance to flow in all retrobulbar vessels. These anomalies may be considered an independent risk factor to develop traumatic glaucoma. (Eur J Ophthalmol 2005; 15: 48-55)*

KEY WORDS. *Ocular trauma, Ocular blood flow, Color Doppler imaging, Resistivity index*

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INTRODUCTION

Ocular trauma, either blunt or penetrating, is often associated with abnormalities of intraocular pressure (IOP): there may be hypotony due to reduction of aqueous production (after ciliary body contusion or inflammation) or excessive aqueous drainage (internally through a cyclodialysis cleft or externally through a break of ocular wall) but more often there is a raise in IOP that may lead to post-traumatic glaucoma. The raise in IOP may be early or late and may have several of the following mechanisms, most of which are related to impaired trabecular drainage:

- Intraocular and trabecular inflammation (that is generally transient) (1)
- Angular recession and trabecular damage and scarring (2, 3)
- Hyphema with blockade of trabecular meshwork by red cells (4, 5)
- "Ghost cells" coming from vitreous hemorrhage may pass in the anterior chamber and cause trabecular impairment and elevated IOP (6)
- Iris and/or ciliary body dialysis (7)
- Lenticular alterations: cataract or lens subluxation

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may cause an alteration of aqueous humor dynamics with pupillary block and/or angle closure (8)
- In cases of penetrating trauma there may be also a direct injury and subsequent scarring of angle or trabecular structures.
- Periocular tissue scarring with elevation of episcleral veins pressure.

These causes illustrate why after ocular trauma there often are IOP disturbances and there may be a traumatic glaucoma, which often has a poor response to therapy and prognosis (9, 10).

In recent years there has been growing evidence that vascular factors are important in the pathogenesis and the progression of glaucomatous damage not only in so called low tension glaucoma but also in primary open angle glaucoma (11-14).

Noninvasive methods to evaluate ocular blood flow are available that give information on blood supply to the orbit and ocular districts: pulsatile ocular blood flow (pOBF) system by Langham measures the variations in IOP induced by blood systolic pulse and derives the pulse volume and from it the blood flow (mainly to the choroid) (15, 16). Color Doppler imaging (CDI) uses Doppler effect to measure the velocity of blood cells (peak systolic velocity = PSV and the end diastolic velocity = EDV) and the resistance to flow (Pourcelot Resistivity index: RI = PSV–EDV/PSV) in different retrobulbar vessels that are strongly correlated to blood flow (17, 18); other instruments utilize scanning laser Doppler flowmetry. Many instruments are therefore available to assess and quantify the state of intraocular circulation (19, 20).

Secondary glaucomas are considered to be mainly or exclusively determined by elevated IOP, but the exact mechanism of glaucomatous damage is not clear even when IOP is strongly elevated and vascular factors may play a role in these secondary glaucomas as well.

We decided to assess the state of ocular blood flow in traumatized eyes (that had in their clinical history an elevated IOP in the early post-traumatic course) using as a control the fellow not traumatized eye. The aim of the study was to identify or exclude the presence of permanent ocular hemodynamics alterations in traumatized eyes (a long time after trauma) that could be influential on the development of traumatic glaucoma in addition to and/or independently from IOP (21, 22).

METHODS

We selected from medical records the patients hospitalized at our institution between January 1, 1997, and July 31, 1999, with a diagnosis of ocular trauma (either blunt or penetrating, Tab. I) and an elevated IOP (≥23 mmHg) during the early post-trauma phase and at least 2 years follow-up; we excluded chemical burns (chemical injuries often have corneal dense leukomas and therefore difficulties in assessing IOP) and selected only mechanical traumatisms; the fellow eye had to be healthy, without any disease other than cataract (inclusion and exclusion criteria are listed in Tab. II). Twenty-five patients responded to inclusion criteria: from the medical records we extracted demographic data, date and type of trauma, maximum IOP recorded in the post-trauma period, surgical or laser treatment when performed; the patients were then summoned and underwent a complete ocular examination (refraction, anterior segment examination, IOP measurement by Goldmann tonometry, fundus examination), visual field (HFA II, Humphrey-Zeiss, San Leandro, CA, Program Central 30-2 SITA fast), ocular blood flow assessment by means of pOBF–Langham system (OBF Labs UK Ltd, Malmesbury, UK), and CDI (ATL Apogee 800 Plus; ATL Co., Bothell, WA) evaluating peak systolic velocity, end diastolic velocity, and Pourcelot RI of ophthalmic artery, central retina artery, nasal and temporal posterior ciliary arteries.

OBF measures were obtained with a slit lamp mounted unit and the patient in sitting position after topical oxybuprocaine eyedrop instillation, while recording arterial pressure; the instrument measures IOP and automatically records and selects five complete pulse cycles and derives pulse amplitude (mmHg), pulse volume (µL), and pulsatile blood flow (µL/sec) (23).

CDI measures were obtained with the patient in supine position while recording arterial pressure, using a 7.5 MHz flat probe. The eyes are closed and a coupling gel is applied to the eyelids. The vessels investigated are as follows (24).

Ophthalmic artery (OA) is localized where it runs parallel to the optic nerve, about 40 mm from the anterior ocular surface, before the crossing of the optic nerve itself. It has a typical wave profile, similar to the internal carotid with an evident dicrotic notch.

Central retinal artery (CRA) is localized in the ante-

rior part of the optic nerve shadow, just posterior to the globe wall; often the central retinal vein is visible at the same location. The wave profile is flat and rounded as compared to the OA wave.

Short posterior ciliary arteries (SPCA, nasal and temporal) are located just posterior to the globe wall at the side of the optic nerve shadow. The SPCA are often difficult to localize as they are small vessels at the inferior resolution limit of CDI. The wave profile is round (without dicrotic notch) but more evident than CRA, probably because more than one single artery contributes to the wave.

The instrument calculates PSV, EDV, and Pourcelot RI (RI = PSV–EDV/PSV).

The parameters evaluated were maximum recorded IOP, IOP at the time of visit, visual field mean deviation (MD) and pattern standard deviation (PSD), OBF pulsatile flow (µL/sec), and CDI values (PSV, EDV, RI) for all four arteries examined (24-27). All parameters were matched with the healthy fellow eye data.

A subgroup analysis and comparison of blunt and penetrating injured eyes was performed. The statistical analysis was performed by means of two-tailed t-test and correlation between different variables with Pearson test. A p value less than 0.05 was the significance limit.

TABLE I - CAUSES AND MECHANISMS OF INJURY

Injury mechanism	Number No.	(%)
Scleral and/or corneal wound	6	(24)
Intraocular foreign body	4	(16)
Bottle cork	6	(24)
Elastic rope	1	(4)
Airbag	1	(4)
Domestic accident (fall)	1	(4)
Working accident (blunt)	6	(24)

TABLE II - INCLUSION AND EXCLUSION CRITERIA

Inclusion criteria	Exclusion criteria
IOP ≥ 23 mmHg	Chemical or electric injury
Blunt or penetrating injury	Severe corneal leukoma
Visual acuity ≥ 0.3	Visual acuity < 0.3
Follow-up ≥ 24 months	Severe structural damage to the eye or orbit
Healthy fellow eye	Fellow eye with vascular ocular disease

IOP = Intraocular pressure

RESULTS

Demographics

The 25 patients included 19 men (76%) and 6 women (24%), the mean age was 52±16 years, the side of injury was the right in 12 cases (48%) and the left in 13 cases (52%). The trauma was blunt in 15 cases (60%) and penetrating in 10 cases (40%). The mechanisms and causes of trauma are listed in Table II.

Thirteen patients (52%) underwent surgery either to repair the primary injury or for subsequent complications; three patients underwent Argon laser trabeculoplasty to reduce elevated IOP. All procedures performed are listed in Table III. Three patients in the blunt trauma group underwent surgery: two cataract extraction and one anterior chamber washing. In the penetrating injury group four patients underwent wound repair and subsequent cataract extraction, two patients underwent wound repair and simultaneous vitrectomy with foreign body extraction. The maximum IOP recorded after trauma ranged from 23 to 50 mmHg (mean 28±7 mmHg). The time elapsed from trauma to the study visit ranged from 24 to 53 months with a mean of 39±12 months.

Intraocular pressure

At the time of visit only 2 patients (8%) were still treated to reduce IOP and one of them was treated in both eyes; the remaining 23 (92%) had IOP under 21 mmHg without any therapy. Mean IOP at the visit recording was 15.1±3.3 mmHg in the injured eye and 13.0±2.7 in the fellow eye and the difference proved to be significant ($p<0.01$). The subgroup analysis comparing blunt trauma and penetrating trauma (Tab. IV) and operated and nonoperated eyes (Tab. V) showed no statistically significant difference between subgroups ($p>0.05$).

TABLE III - SURGICAL AND LASER PROCEDURES PERFORMED ON THE 25 INJURED EYES AFTER THE TRAUMA

Procedure	Patients	(%)
Foreign body extraction	3	(12)
Argon laser trabeculoplasty	3	(12)
Corneo-scleral suture	8	(32)
Anterior chamber washing	1	(4)
Cataract	6	(24)

Visual field

The mean visual field MD was –4.39±4.13 dB in the trauma eyes and –1.87±1.43 in the fellow eyes: the difference was statistically significant ($p<0.05$). The mean visual field PSD was 3.49±3.77 in the trauma group and 1.84±0.61 in the fellow eyes: the difference was not statistically significant ($p=0.085$). In a subgroup analysis comparing blunt and penetrating trauma (Tab. IV) or operated and nonoperated eyes (Tab. V) penetrated and operated eyes had worse results than blunt trauma and nonoperated eyes, but the difference was not statistically significant (p always greater than 0.05).

pOBF

The pOBF estimate gave a mean of 11.25±6.56 µL/sec in the trauma eyes and 15.40±7.29 in fellow eyes: the

TABLE IV - COMPARISON BETWEEN BLUNT AND PENETRATING INJURIES

Characteristics	Blunt trauma (n=15)	Penetrating trauma (n=10)	p
Max IOP (mmHg)	29.36± 7.49	26.00±5.01	0.23
Follow-up (months)	39.53±14.35	39.3 ±8.86	0.96
Visual acuity	0.83± 0.34	0.72±0.35	0.46
IOP (mmHg)	15.29± 3.84	14.90±2.51	0.78
pOBF (µL/sec)	13.61± 6.59	9.45±6.58	0.16
MD (dB)	-3.81± 3.51	-5.16±4.94	0.47
PSD (dB)	2.41± 1.79	4.95±5.2	0.13
OA-PSV (cm/sec)	39.38±23.51	50.85±32.76	0.27
OA-EDV (cm/sec)	10.90± 7.52	11.98±7.85	0.73
OA-RI	0.73± 0.07	0.77±0.05	0.15
CRA-PSV (cm/sec)	16.16± 6.02	18.02±7.08	0.49
CRA-EDV (cm/sec)	4.99± 3.29	4.70±1.53	0.79
CRA-RI	0.71± 0.11	0.73±0.05	0.56
NSPCA-PSV (cm/sec)	21.49±12.95	19.89±7.28	0.80
NSPCA-EDV (cm/sec)	5.54± 3.64	5.16±2.34	0.84
NSPCA-RI	0.74± 0.07	0.75±0.05	0.90
TSPCA-PSV (cm/sec)	28.57±23.46	21.64±10.05	0.32
TSPCA-EDV (cm/sec)	7.17± 5.25	6.31±3.75	0.64
TSPCA-RI	0.74± 0.06	0.72±0.07	0.50

Values are mean±SD. No differences were significant
IOP = Intraocular pressure; pOBF = Pulsatile ocular blood flow; MD = Mean deviation; PSD = Pattern standard deviation; OA = Ophthalmic artery; PSV = Peak systolic velocity; EDV = End diastolic velocity; RI = Resistivity index; CRA = Central retinal artery; NSPCA = Nasal short posterior ciliary arteries; TSPCA = Temporal short posterior ciliary arteries

TABLE V - COMPARISON BETWEEN OPERATED AND NONOPERATED EYES

Characteristics	Operated eyes	Nonoperated eyes	p
IOP (mmHg)	15.10±2.36	15.42±4.97	0.81
pOBF (ml/sec)	9.74±6.18	13.47±6.45	0.15
MD (dB)	-4.74±4.31	-3.15±4.23	0.66
PSD (dB)	4.28±4.63	2.89±1.89	0.28
OA-RI	0.76±0.05	0.68±0.19	0.08
CRA-RI	0.73±0.05	0.66±0.20	0.34
NSPCA-RI	0.75±0.05	0.69±0.21	0.67
TSPCA-RI	0.75±0.06	0.67±0.18	0.12

Values are mean±SD. No differences were significant
IOP = Intraocular pressure; pOBF = Pulsatile ocular blood flow; MD = Mean deviation; PSD = Pattern standard deviation; OA = Ophthalmic artery; RI = Resistivity index; CRA = Central retinal artery; NSPCA = Nasal short posterior ciliary arteries; TSPCA = Temporal short posterior ciliary arteries

TABLE VI - DATA OF CDI EVALUATION OF RETROBULBAR VESSELS IN INJURED AND FELLOW HEALTHY EYES

Vessel		Injured eye, mean±SD	Fellow eye, mean±SD	Difference (t-test), p
OA	PSV	44.42±28.02	37.64±22.42	0.037
	EDV	11.48± 7.63	12.97± 8.08	0.10
	RI	0.74± 0.07	0.66± 0.06	$3.32x10^{-6}$
CRA	PSV	17.10± 6.45	16.68± 8.13	0.77
	EDV	4.96± 2.71	6.42± 3.35	0.05
	RI	0.71± 0.09	0.62± 0.07	0.0002
NPSCA	PSV	21.15±11.63	17.04± 8.20	0.30
	EDV	5.52± 3.30	6.79± 3.60	0.09
	RI	0.74± 0.06	0.61± 0.07	$3.87x10^{-7}$
TPSCA	PSV	24.58±16.54	25.21±17.54	0.88
	EDV	6.75± 4.34	9.90± 7.63	0.06
	RI	0.73± 0.07	0.62± 0.06	$1.03x10^{-7}$

For each vessel systolic and diastolic velocities (PSV and EDV) and resistivity index (RI) were calculated. The statistical significance of the difference between injured and healthy eyes is shown in the last column (significance limit = p<0.05)
CDI = Color Doppler Imaging; OA = Ophthalmic Artery; PSV = Peak Systolic Velocity; EDV = End Diastolic Velocity; CRA = Central Retinal Artery; NSPCA = Nasal Short Posterior Ciliary Arteries; TSPCA = Temporal Short Posterior Ciliary Arteries

difference resulted highly significant (p=0.002). In a subgroup analysis comparing blunt and penetrating trauma (Tab. IV) or operated and nonoperated eyes (Tab. V) penetrated and operated eyes had worse results than blunt trauma and nonoperated eyes, but the difference was not statistically significant (p always greater than 0.05).

CDI

CDI results are described in Table VI.

- OA: PSV was 44.42±28.02 cm/sec in trauma eyes and 37.64±22.42 cm/sec in fellow eyes (p<0.05); EDV was 11.48±7.63 cm/sec in trauma eyes and 12.97±8.08 cm/sec in fellow eyes (p>0.05). OA RI was 0.74±0.07 in trauma eyes and 0.66±0.06 in fellow eyes ($p=3.32x10^{-6}$ – p<0.0001).
- Central retinal artery (CRA): PSV was 17.10±6.45 cm/sec in trauma eyes and 16.68±8.13 cm/sec in fellow eyes (p>0.05); EDV was 4.96±2.71 cm/sec in trauma eyes and 6.41±3.35 cm/sec in fellow eyes (p=0.0507); RI was 0.71±0.09 in trauma eyes and 0.62±0.07 in fellow eyes (p<0.0005).
- Nasal SPCA (NSPCA): PSV was 21.15±11.63 cm/sec in trauma eyes and 17.04±8.20 cm/sec in fellow eyes (p>0.05); EDV was 5.52±3.30 cm/sec in trauma eyes and 6.79±3.60 cm/sec in fellow eyes (p>0.05); RI was 0.74±0.06 in trauma eyes and 0.61±0.07 in fellow eyes ($p=3.87x10^{-7}$ – p< 0.0001).
- Temporal SPCA (TSPCA): PSV was 24.58±16.54 cm/sec in trauma eyes and 25.21±17.54 cm/sec in fellow eyes (p>0.05); EDV was 6.75±4.34 cm/sec in trauma eyes and 9.90±7.63 cm/sec in fellow eyes (p>0.05); RI was 0.73±0.07 in trauma eyes and 0.62±0.06 in fellow eyes ($p=1.03x10^{-7}$ – p<0.0001).

The subgroup analysis dividing and comparing penetrating from blunt injuries gave for all studied variables results not statistically different between the two groups (Tab. IV).

Further analysis comparing the eyes that were operated with those not operated confirmed the significant difference in CDI RI between traumatized and fellow eyes in both subgroups (operated and nonoperated), while the difference between operated and nonoperated eyes was not significant (Tab. V) even if penetrated or operated eyes had higher RI than blunt trauma or nonoperated eyes.

Using the Pearson's test we studied the correlations between IOP and ocular blood flow parameters and between OBF and CDI measures. The results are summarized in Tables VII and VIII.

Considering the relationship between the IOP and the flow measures (Tab. VII) we note that the pOBF value shows a slight (R=–0.15) inverse correlation with IOP in the injured eyes that is not present in healthy

eyes (R=0.08); the CDI RI of ophthalmic, nasal, and temporal short posterior ciliary arteries in injured eyes have no correlation with IOP, while in healthy eyes there is a slight to fair positive correlation between IOP and RI in the same arteries.There are no clear correlations between pOBF and CDI values (Tab. VIII).

TABLE VII - CORRELATION (R value–Pearson's test) BETWEEN INTRAOCULAR PRESSURE AND DIFFERENT OCULAR BLOOD FLOW PARAMETERS IN INJURED AND HEALTHY EYES

Ocular blood flow parameters	Intraocular pressure	
	Injured eye	Fellow eye
pOBF	–0.15	0.08
OA-PSV	0.16	–0.15
OA-EDV	–0.06	–0.18
OA-RI	0.01	0.37
CRA-PSV	0.16	–0.15
CRA-EDV	0.30	–0.10
CRA-RI	–0.26	–0.09
NPSCA-PSV	0.40	0.04
NPSCA-EDV	0.33	–0.06
NPSCA-RI	–0.10	0.24
TPSCA-PSV	–0.04	–0.01
TPSCA-EDV	–0.01	–0.03
TPSCA-RI	–0.04	0.19

pOBF= Pulsatile ocular blood flow; OA = Ophthalmic artery; PSV = Peak systolic velocity; EDV = End diastolic velocity; RI = Resistivity index; CRA = Central retinal artery; NSPCA = Nasal short posterior ciliary arteries; TSPCA = Temporal short posterior ciliary arteries

TABLE VIII - CORRELATION (R value–Pearson's test) BETWEEN PULSATILE OCULAR BLOOD FLOW (pOBF) VALUE AND DIFFERENT CDI PARAMETERS

CDI parameters	POBF	
	Injured eye	Fellow eye
OA-PSV	–0.43	–0.34
OA-EDV	–0.38	–0.22
OA-RI	–0.02	–0.08
CRA-PSV	–0.16	–0.26
CRA-EDV	–0.03	–0.21
CRA-RI	–0.06	–0.11
NPSCA-PSV	–0.33	0.41
NPSCA-EDV	–0.46	0.36
NPSCA-RI	0.39	0.03
TPSCA-PSV	–0.42	–0.32
TPSCA-EDV	–0.42	–0.30
TPSCA-RI	0.04	–0.15

OA = Ophthalmic artery; PSV = Peak systolic velocity; EDV = End diastolic velocity; RI = Resistivity index; CRA = Central retinal artery; NSPCA = Nasal short posterior ciliary arteries; TSPCA = Temporal short posterior ciliary arteries

DISCUSSION

In an attempt to draw some conclusion from the collected data we underline four points.

The relevant result of our study is the very significant difference in ocular blood flow measures between injured and fellow healthy eyes: more than 2 years after the trauma, the pOBF is significantly reduced in injured eyes while the CDI RI of all retrobulbar vessels is strongly increased, pointing out an increase in overall resistance to blood flow in injured eyes. The systolic velocity is significantly higher in injured than in normal eyes, while all other parameters are not significantly different between the two groups. The increase of ophthalmic artery systolic velocity in injured eyes may be explained with a relative stenosis due to scarring or increase of orbital tissues rigidity.

The IOP of injured eyes is higher than in normal eyes, even if most injured eyes have IOP within the normal range without therapy (only 8% of eyes received IOP lowering drugs). The strong increase in resistance to blood flow in injured eyes does not seem to correlate with the increased IOP, but is an independent phenomenon. Harris et al (28-30) found that RI of CRA and SPCA is correlated with IOP, while OA RI is not. Our results are only partially in agreement as in normal eyes we found that SPCA and OA RI seem to be correlated with IOP in healthy eyes and this correlation is lost in injured eyes.

- There are no significant differences between blunt and penetrating injuries (Tab. IV): CDI values were almost identical. OA RI is higher in penetrating than in blunt injuries; similarly, pOBF values are lower in penetrating injuries but the differences are not significant, perhaps due to the small numbers, and it would therefore be interesting to extend the observation to more numerous groups. Similarly, comparing operated and nonoperated eyes we found that perfusion parameters (pOBF and CDI RI of all retrobulbar arteries) were worse in operated eyes but again the difference did not attain statistical significance (Tab. V).
- The correlations between the data obtained by two different technologies to assess the ocular perfusion dynamics give results that are not easily interpreted (Tab. VIII). The pOBF is lower and the RI of retrobulbar vessels is higher in injured eyes but the two variables do not correlate each other, and

they correlate differently between the two eyes and the different arteries: both methods show an impairment of ocular perfusion but the eyes that have more reduced OBF are not those with higher RI (as one could expect). We believe that the two methods are hardly comparable each other, as they measure different variables at different anatomic levels, with very different technologies (31, 32).

We conclude from collected data that ocular trauma causes a significant and longstanding impairment of ocular circulation that is apparently independent from IOP elevation and may act as an independent risk factor in the development of post-traumatic glaucoma. We do not know if this impairment is functional (increased vasoconstriction/vasospasm) or structural (increase in ocular wall rigidity, scarring of perioocular tissues). We hope to have more information on this point repeating the pOBF and CDI measures after administration of drugs with vasodilator effect.

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