Effects of Trabeculectomy on Posterior Ciliary Artery Blood Flow and Ocular Pulse Amplitude

Trabekülektominin Posterior Siliyer Arter Kan Akımı ve Oküler Puls Amplitüdü Üzerine Etkileri

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ABSTRACT

Purpose: To investigate the effects of trabeculectomy surgery on intra-ocular pressure, ocular blood flow and ocular pulse amplitude.

Materials and methods: 25 eyes of 25 patients diagnosed with primary open angle glaucoma who had undergone trabeculectomy surgery were included in the prospective study. Before surgery, ocular pulse amplitude (OPA) and intraocular pressure were measured by Pascal dynamic contour tonometer (DCT), intraocular pressure was also measured by Goldmann applanation tonometer (GAT), central corneal thickness was measured by digital ultrasonic pachymeter and short posterior ciliary artery (SPCA) blood flow was measured by colored doppler imaging ultrasonography, respectively in all eyes. All measurements were repeated at first month after the surgery.

Results: Preoperative IOP and OPA values measured by DCT were 26.55±4.28 mmHg and 4.06±0.90, respectively. Nasal and temporal SPCA resistive index (RI) were 0.61±0.07 and 0.62±0.10, respectively. Postoperative 1st month IOP and OPA values measured by DCT were 14.36±2.49 mmHg and 2.52±0.56, respectively. Postoperative nasal and temporal SPCA RI were 0.49±0.09 and 0.52±0.09, respectively. There was a statistically significant decrease in IOP, OPA, nasal and temporal SPCA RI postoperatively.

Conclusion: IOP, OPA and RI were decreased, nasal and temporal SPCA maximum, minimum and mean blood flow velocities were increased postoperatively compared to the preoperative values. There was a strong positive correlation between OPA and nasal SPCA RI. OPA score which gives information about ocular blood flow and resistive index may be a good predictive indicator for glaucoma follow up.

Key word: Glaucoma, ocular pulse amplitude, resistive index, trabeculectomy, doppler ultrasound.

ÖZ

Amaç: Trabekülektomi cerrahisinin göz içi basıncı, oküler kan akımı ve oküler puls amplitüdü üzerine etkilerini araştırmak.

Gereç ve yöntem: Primer açık açılı glokom tanısı ile trabekülektomi cerrahisi geçiren 25 hastanın 25 gözü bu prospektif çalışmaya dahil edildi. Cerrahiden önce, oküler puls amplitüdü ve göz içi basıncı (GİB) Pascal dinamik kontür tonometre ile, ek olarak GİB Goldmann aplanasyon tonometrisi ile, merkezi kornea kalınlığı (MKK) dijital ultrasonik pakimetre ile, kısa posteriyor siliyer arter (KPSA) kan akımı renkli dopler ultrason ile ölçüldü. Tüm ölçümler cerrahi sonrası 1. ayda tekrarlandı.

Bulgular: Cerrahi öncesi Pascal dinamik kontür tonometre ile ölçülen GİB ve oküler puls amplitüdü sırası ile 26.55±4.28 mmHg ve 4.06±0.90 olarak kaydedildi. Nazal ve temporal KPSA resistif indeksi sırası ile 0.61±0.07 ve 0.62±0.10 olarak hesaplandı. Cerrahi sonrası 1. ayda Pascal dinamik kontür tonometre ile ölçülen GİB ve oküler puls amplitüdü sırası ile 14.36±2.49 mmHg ve 2.52±0.56 olarak ölçüldü. Cerrahi sonrası nazal ve temporal KPSA resistif indeksi sırası ile 0.49±0.09 ve 0.52±0.09 olarak hesaplandı. Göz içi basıncı, oküler puls amplitüdü, nazal ve temporal KPSA resistif indekslerinde cerrahi sonrası değerlerde istatistiksel olarak anlamlı bir azalma görüldü.

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Sonuç: Cerrahi öncesi değerler ile karşılaştırıldığında; cerrahi sonrasında GİB, oküler puls amplitüdü ve resistif indeks değerlerinde düşüş, nazal ve temporal KPSA maksimum, minimum ve ortalama kan akım hızlarında artış görüldü. Oküler puls amplitüdü ve nazal KPSA resistif indeksleri arasında kuvvetli bir ilişki bulundu. Oküler kan akımı ve resistif indeks hakkında bilgi veren oküler puls amplitüdü skoru, glokom takibinde iyi bir gösterge olabilir.

Anahtar kelimeler: Glokom, oküler puls amplitüdü, resistif indeks,trabekülektomi,dopler ultrason.

INTRODUCTION

Glaucoma is a progressive optic neuropathy characterised by progressive cupping of the optic disc and specific visual field defects.[1] Although the main reason of the optic nerve damage in glaucoma is the intraocular pressure (IOP), the weak relation between glaucoma progression and IOP level in normotensive glaucoma (NTG) shows that there are other factors involving in the pathogenesis of glaucoma related damage.[2] In clinical studies, it was reported that besides high IOP, vascular factors, age, genetic and demographic factors may play an important role in glaucoma etiopathology.[3-6] It is supposed that decrease of optic nerve blood flow due to vascular changes results with failure of neural tisssue nutrition and optic nerve damage.[7]

IOP is an important parameter in glaucoma diagnosis and management. It is not a constant value and shows pulsation related to the cardiac cycle.[8] The difference between systolic and diastolic IOP is called ocular pulse amplitude (OPA). OPA occurs as a result of volume variations of choroidal blood flow during cardiac cycle. The most important clinical features of OPA are to be indirect indicator of choroidal perfusion and to show ocular blood flow during heart beat.[9,10]

There are evidences that OPA may have an important role on glaucoma management.[11,12] Decrease in OPA which is an indirect indicator of choroidal blood flow, results with hipoxia and later on neuron loss. It was reported that this situation may cause glaucoma progression and optic nerve perfusion failure.

In this study, the effects of trabeculectomy on IOP, OPA, nasal and temporal SPCA blood flow were investigated.

MATERIAL AND METHODS

Thirty-two patients who have undergone to trabeculectomy surgery with the diagnose of primary open angle glaucoma (POAG) at ophthalmology department of XXXXXX University School of Medicine between September 2009 and December 2009 were included into the study. The study was approved by the institutional ethic committee. All procedures followed the Declaration of Helsinki rules and informed consent was obtained from the patients.

Seven patients were excluded because of postoperative complications and lack of adherence to the follow up. Twenty-five patients were taken into evaluation in the study. Inclusion criterias were: to be diagnosed with POAG and to have undergone trabeculectomy surgery. Exclusion criterias were to have previous glaucoma surgery, pigment dispersion or pseudoexfoliation syndromes.

All demographic characteristics of the patients were recorded. A standart ophthalmic examination containing IOP measurement, gonioscopy, slit-lamp biomicroscopy was performed.

Before surgery, ocular pulse amplitude (OPA) and intraocular pressure were measured by Pascal dynamic contour tonometer (DCT) (Ziemer ophthalmic systems AG, Switzerland), intraocular pressure was also measured by Goldmann applanation tonometer (GAT), central corneal thickness was measured by digital ultrasonic pachymeter (Tomey Bio-Pachymeter AL-3000, Tomey Corp., Japan) and short posterior ciliary artery (SPCA) blood flow was measured by color doppler imaging ultrasonography (LOGIQ 5 Pro,General Electric Medical Systems, Wisconsin,USA), respectively in all eyes.

Nasal and temporal SPCA blood flow velocity of all patients were evaluated. Peak systolic velocity (PSV) measured during systolic period was recorded as maximum velocity and end diastolic velocity (EDV) measured during diastolic period was recorded as minimum velocity. All the measurements were performed by the same radiologist (KA). Nasal and temporal SPCA blood flow maximum velocity, minimum velocity and mean velocity were recorded separately. Also, Pourcelet's RI value (PSV-EDV)/PSV was calculated for each arterial blood flow.

Trabeculectomy surgery under peribulber anesthesia was performed on all eyes by the same surgeon (NÖ). At first month of the surgery, ocular pulse amplitude (OPA) and intraocular pressure were measured by Pascal dynamic contour tonometer (DCT), intraocular pressure was also measured by Goldmann applanation tonometer (GAT), central corneal thickness was measured by digital ultrasonic pachymeter and short posterior ciliary artery (SPCA) blood flow was measured by colored doppler imaging ultrasonography, respectively in all eyes. Preoperative and postoperative measurements were compared statistically.

Statistical analysis was performed with Statistical Package for the Social Sciences (SPSS ver.18.0) and *P* values smaller than 0.05 were considered statistically significant. Quantitative variables are expressed as mean values±SD. One-sample Kolmogorov-Smirnov test was used to determine whether the data was distributed normally. For comparisons, a paired *t* test was used for normally distrubuted data and the Wilcoxon signed-rank test was used for the data not nor-

mally distributed. Pearson Correlation Analysis was used to investigate the relationship between quantitative variables.

RESULTS

Of 25 participants, 11 were male (44%) and 14 were female (56%) with a mean age of 40.4±12.62 years. Eight eyes were right and 17 were left with a total of 25 eyes. The mean corneal thickness was 538±32.29µm. Preoperative IOP and OPA values measured by DCT were 26.55±4.28 mmHg and 4.06±0.90, respectively. IOP measured by GAT was 26.92±4.75 mmHg. Nasal and temporal SPCA resistive index (RI) were 0.61±0.07 and 0.62±0.10, respectively. Postoperative 1st month IOP and OPA values measured by DCT were 14.36±2.49 mmHg and 2.52±0.56, respectively. IOP measured by GAT was 14.16±2.44 mmHg. Postoperative nasal and temporal SPCA RI were 0.49±0.09 and 0.52±0.09, respectively. There was a significant decrease in IOP after trabeculectomy surgery compared to the preoperative values measured by both DCT and GAT. There was a significant decrease in OPA values measured at 1st month postoperatively compared to the preoperative values (Table 1).

Postoperatively, nasal SPCA blood flow maximum velocity, minimum velocity and mean velocity were increased. RI was decreased. Increase in minimum velocity and mean velocity and decrease in RI were statistically significant (p<0,05). Increase in maximum velocity was not statistically significant (P=0,061) (Table 1).

Postoperatively, temporal SPCA blood flow maximum velocity, minimum velocity and mean velocity were increased. RI was decreased. Increase in maximum velocity, minimum velocity and mean velocity and decrease in RI were statistically significant (p<0,05) (Table 1).

As we investigated the correlation between preoperative and postoperative blood flow parameters and other parameters. There was a positive correlation between IOP and OPA. Also, there was a positive correlation between IOP and SPCA RI. There was a negative correlation between IOP and nasal SPCA EDV. There was a strong positive correlation between nasal SPCA RI and OPA, there was a negative correlation between nasal SPCA RI and EDV.

Table 1: *Comparison of preoperative and postoperative measurements*

	PREOPERATİVE Mean±SD Median (min-max)	**POSTOPERATİVE Mean±SD Median (min-max)**	**P-Value**
IOP(DCT)	26.55±4.28 26.40(20-40)	14.36±2.49 14.0(9-18.3)	<0.001
IOP(GAT)	26.92±4.75 26(20-42)	14.16±2.44 14(10-18)	<0.001
Nasal SPCA PSV	6.44±1.54 6.50(4.1-10.1)	6.90±1.55 6.70(4-10.31)	0.061
Nasal SPCA EDV	2.47±0.65 2.53(1.3-1.8)	3.49±0.93 3.50(5.5-1.9)	<0.001
Nasal SPCA MV	3.89±1.05 3.84(2-5.6)	4.45±1.32 4.28(2.45-7.22)	0.022
Nasal SPCA RI	0.61±0.07 0.63(0.47-0.745)	0.49±0.09 0.52(0.214-0.62)	<0.001
Temporal SPCA PSV	8.26±2.0 8.40(4.2-12.4)	9.38±1.81 9.77(6.4-12.8)	0.004
Temporal SPCA EDV	3.0±0.90 2.80(2-5.6)	4.52±1.19 4.30(2.4-7.2)	<0.001
Temporal SPCA MV	4.79±1.61 4.50(2.4-8.6)	5.92±1.75 5.89(3.01-9.2)	<0.001
Temporal SPCA RI	0,62±0,10 0,66(0,333-0,766)	0.52±0.09 0.55(0.316-0.656	0.001
OPA	4.06±0.90 3.80(2.5-6.3)	2.52±0.56 2.70(0.9-3.2)	<0.001

IOP: Intraocular pressure, DCT: Dynamic contour tonometer, GAT: Goldmann applanation tonometer, SPCA: Short posterior ciliary artery, EDV: End diastolic velocity, PSV: Peak systolic velocity, MV: Mean velocity , OPA: Ocular pulse amplitude, RI: Resistive index, Min: Minimum value, Max: Maximum value

DISCUSSION

The pathophysiology of the glaucoma is not well understood. The most important risk factor is high IOP.[6] It is suggested that high IOP directly damages axons and small blood vessels in the lamina cribrosa and causes glaucomatous changes.[13] It has been shown that especially fluctuation in IOP causes visual field defect progression but high IOP is not responsible alone.[14] In some studies, it has been reported that glaucomatous damage also occurs in some patients with controlled high IOP and with normal IOP.[15] Duo to these studies, there may be other factors playing role in glaucoma pathophysiology. Recently, the vascular theory is the most popular one and it is supported by many recent studies. Researchers investigating the validity of the vascular theory, have studied blood flow changes in healthy and glaucomatous subjects by various blood flow analyse methods. In a study reporting the relationship between glaucoma and ocular blood flow, Yamazaki et al. have compared normotensive glaucoma (NTG) patients with or without progression[16] and POAG patients with or without progression. Blood flow velocity of all patients was measured by color doppler ultrasonography and it was reported that, decreased PSV and EDV and increased RI were determined in central retinal artery and SPCA of NTG patients with progression compared to the NTG patients without progression. There was not a significant difference between POAG patients with and without progression in the same study.[16] Rankin et al. have compared ocular blood flow of central retinal artery and SPCA of POAG and NTG patients with healthy subjects by using color doppler ultrasonography. They have reported that in POAG and NTG patients, EDV was lower and RI was higher than healthy subjects.[17-18] Galassi et al. have reported decreased EDV and increased RI by color doppler ultrasonography in ophthalmic arteries of POAG patients with progression compared to the POAG patients without progression.[19] Martinez et al. have reported that POAG patients with high RI values in ophthalmic artery and SPCA have high progression rate than others.[20] Satılmıs et al. showed that decreased EDV in central retinal artery is associated with progression in POAG patients.[21] Zeitz et al. have compared the results of color doppler ultrasonography of SPCA in 114 NTG patients and 40 healthy subjects. NTG patients with progression had decreased PSV and EDV compared to the NTG patients without progression and healthy control group.[22]

There is limited data about changes on ocular hemodynamics after trabeculectomy surgery. Berisha et al. have reported an increase in optic nerve head blood flow and fundus pulsation amplitude after trabeculectomy surgery.[23] Trible et al. have reported a significant increase in central retinal artery, nasal and temporal short poserior ciliary artery EDV and PSV and a significant decrease in RI after trabeculectomy surgery.[24] Unlike previous study, Louis et al. have reported that there is not a significant change in central retinal artery, nasal and temporal short posterior ciliary artery EDV, PSV and RI after surgery.[25] Sakarya et al. have reported an increase in nasal posterior ciliary artery PSV, EDV and mean blood flow velocity and a decrease in RI.[26]

In our study, we have found that there are statistically significant changes in SPCA blood flow after trabeculectomy surgery in glaucoma patients. Especially, we have recorded a significant increase in temporal SPCA maximum, minimum and mean blood flow velocity and a decrease in RI. There was an increase in nasal SPCA minimum and mean blood flow velocity and there was a decrease in RI. These results are in accord with some of the studies in the literature.

It is difficult to show correlation between ocular perfusion failure and glaucoma progression. There is not a golden standard method to measure ocular blood flow. Recently, color doppler ultrasonography is used most commonly to measure blood flow velocity but the blood flow itself, its characteristics and vessel diameter may not be measured by this method. Also, it takes years to become a glaucoma patient, so long term follow up and more studies are needed to correlate glaucoma progression and characteristics of blood flow. Additionally, systemic and topical medications taken may affect ocular blood flow. Because of all these reasons, studies are limited. It is still not known whether ocular perfusion failure results with glaucoma or glaucoma itself contributes to ocular perfusion failure. There are some hypotheses in the literature suggesting that vascular pathology takes role in some glaucoma types.[19]

Some studies report that IOP changes central retinal artery and short posterior ciliary artery blood flow velocity but does not affect ophthalmic artery blood flow.[27] As IOP rises, RI increases, PSV and EDV decreases in arteries. Our study is in accord with these results for short posterior ciliary artery.

Ninety percent of the ocular blood flow is consisted of choroidal blood flow. OPA is the difference between systolic and diastolic IOP. It is accepted as the indicator of the ocular blood flow and it is also indirect indicator of the choroidal perfusion. Adequate ocular perfusion and perfusion pressure is needed to maintain blood circulation of ocular tissues particularly optic nerve head which is the most vulnerable to ischemia.[28] Ocular perfusion pressure is calculated as the difference between arterial blood pressure and IOP. Thus, blood flow difference at each heart beat must be higher than IOP to maintain adequate ocular blood flow. Ocular blood flow is 650-750 µl per minute and 2-5% of it is originated from retina and 85-90% of it is originated from the choroid. A part of this blood flow is pulsatile and changes at every heart beat. The other part of this blood flow is non-pulsatile.

OPA which is the indirect response of the pulsatile ocular blood flow is assessed as a criteria for evaluation of ischemic glaucomatous atrophy. Langham has found OPA as 1.5±0.11mmHg in healthy subjects using pneumotonome-

ter.[29] Alimgil et al. have found OPA as 2.6±0.7 mmHg in Turkish population using Langham pneumotonometer.[30] The first study to measure OPA with DCT is performed by Hoffmann et al. and they have found OPA as 3.08±0.92 mmHg in healthy subjects.[31] Kaufmann et al. have found OPA as 3.00 mmHg in healty subjects using same tonometer.[32] Özcetin et al. have found OPA as 2.8 mmHg in healthy subjects using DCT and pulsatile ocular blood flow analyser.[28] Erdurmus et al. have found OPA as 2.8 mmHg similarly.[33] Punjabi et al. have studied on 906 eyes of 501 subjects consisted of POAG, ocular hypertension(OHT), NTG, Pseudoexfoliative glaucoma (PEXG) and healthy subjects. They have reported that OPA values measured by DCT were highest in OHT group (3.61 mmHg) and lowest in healthy group (2.86 mmHg). It is reported that OPA has increased significantly with IOP rise in all groups.[28] Rompanian et al. have studied on OPA values in healthy subjects, OHT, POAG, NTG and operated trabeculectomy patients.[34] They have reported OPA as 3.1 mmHg, 3.6 mmHg, 3.1 mmHg, 2.9 mmHg and 2.4 mm Hg, respectively. Kaufmann et al. have reported that OPA value measured by DCT in 223 healthy eyes was 0.9-7.2 mmHg (mean 3 mmHg). They also have found that OPA is not affected by central corneal thickness, corneal curvature, anterior segment depth, age and sex. They have reported that OPA correlates with IOP and every 1 mmHg increase in IOP results with 0.12 mmHg increase in OPA.[32] In our study, we have found a statistically significant positive correlation between IOP and OPA in accord with the literature.

There are many studies in the literature reporting that OPA is higher in glaucoma patients than healthy subjects and OPA may take role in glaucoma etiopathology.[31,32,34] First studies about OPA changes in glaucoma were performed by using Langham pneumotonometer.[29] By using Langham pneumotonometer, Trew and Smith have reported that OPA is higher in OHT group than POAG and healthy group.[35] They have found that lowest OPA was in NTG group. There are various reports in the literature about OPA measurements in glaucomatous patients. Stalmans et al. have found that OPA was 2.6±1.3, 2.3±0.8 and 3.4±1.6 mmHg in POAG, NTG and healthy groups, respectively.[36]

In the literature, the effects of surgery on OPA have been investigated. Breusegem et al. performed trabeculectomy surgery on 48 eyes and evaluated the other eyes as control group. In this study, IOP and OPA measured by DCT before and 1st month after the surgery are 21.33 mmHg, 3.23 mmHg and 14.45 mmHg, 2.12 mmHg, respectively. OPA decreased statistically significantly after trabeculectomy surgery. In the study, it has been emphasized that there is a strong correlation between IOP changes and OPA.[37] Von Schulthess et al. investigated the effects of trabeculectomy on OPA in a similar study. They searched, whether OPA decrease at the early postoperative period may be a prognostic parameter for long term success of the surgery or not. The study included 14 patients with POAG and PEXG. They have reported that more than 2 mmHg decrease in OPA at early postoperative period may be a good prognostic parameter for long term IOP control.[38] In our study, mean OPA value before and after trabeculectomy surgery was determined as 4.06±0.90 and 2.52±0.56, respectively. Limited studies in the literature have similar results.

In the correlation analyses between blood flow velocity parameters and other factors, particularly relationship between IOP, OPA and vascular parameters were remarkable. In our study, there was a positive correlation between IOP (measured by GAT and DCT) and OPA before and after trabeculectomy surgery ($p=0.003$ $r=0.566$, $p=0.001$ $r=0.629$). There was a positive correlation between IOP (measured by GAT and DCT) and SPCA RI ($p=0.001$ $r=0.621$, $p=0.001$ $r=0.644$). There was a negative correlation between IOP (measured by GAT and DCT) and nasal SPCA EDV ($p=0.010$ $r=-0.503$, $p=0.005$ $r=-0.548$). There was a strong positive correlation between OPA and nasal SPCA RI ($p=0.022$ $r=0.456$). There was a negative correlation between nasal SPCA EDV and RI ($p<0.001$ $r=-0.688$).

There are various studies reporting that anti-glaucomatous medication change OPA. It has been reported in previous studies that topical carbonic anhydrase inhibitors[39], prostaglandin analogs[40] and beta blockers[41] decrease IOP and increase OPA. Also it has been shown that timolol decreases IOP but it does not have an effect on OPA[42]. In our study, patients were taking one or more anti-glaucomatous medications before surgery. These medications were stopped after surgery and therefore may affect OPA and RI. However, we did not investigate the effects of these medications in our study. This may be a limitation of our study. Effects of anti-glaucomatous medications on OPA and RI may be investigated in a different study.

Our study has some limitations. Calculation of ocular perfusion pressure would make it more valuable. Central retinal artery and ophthalmic artery blood flow was not analysed in this study. So, measurements of short posterior ciliary artery do not represent exactly the effects of trabeculectomy on ocular blood flow but give an idea.

As a result of our study, decrease in IOP after trabeculectomy surgery caused a decrease in nasal and temporal short posterior ciliary artery RI and OPA also an increase in maximum, mean and minimum blood flow velocity. OPA value which is easy to measure and has a high repeatability may give an idea about RI value which needs experience and difficult to measure. In our study, a strong positive correlation between OPA and nasal short posterior ciliary artery RI was determined. Because of strong correlation between OPA and nasal short posterior ciliary artery RI, OPA value may be a useful indicator for glaucoma follow up, ocular blood flow and RI. More comprehensive studies about OPA measurement may clearly explain this issue.

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