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Research Article



# The effects of non-prostaglandin hypotensive drops with preservative on central retinal thickness after phacoemulsification

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## ABSTRACT

**BACKGROUND:** Primary open angle glaucoma is often associated with cataract. The correlation between the use hypotensive drops, in particular non-prostaglandin hypotensive drops with preservative, and the change in central retinal thickness after phacoemulsification with intraocular lens implantation continues to be relevant.

**AIM:** To evaluate central retinal thickness and pseudophakic cystoid macular edema incidence after phacoemulsification with intraocular lens implantation in patients with primary open-angle glaucoma using non-prostaglandin hypotensive drops with preservatives.

**MATERIALS AND METHODS:** 94 patients (108 eyes) with cataract were enrolled in the study, divided into 3 groups: the first group — 21 patients (27 eyes), and the second group — 21 patients (23 eyes) with primary open-angle glaucoma using non-prostaglandin hypotensive drops with preservative; the third (control) group included 52 patients (58 eyes) without ocular comorbidities. All patients underwent uncomplicated phacoemulsification with intraocular lens implantation. In the post-op period, patients of the first group received topical antibiotics and steroids, patients of the second and the third groups received the same treatment and non-steroidal anti-inflammatory drops as well. Central retinal thickness was measured using optical coherence tomography before surgery, 2 weeks, 2 and 6 months after surgery.

**RESULTS:** The central retinal thickness increase in comparison with baseline values was more significant in the first group than in the second and the third groups, and the time of recovery to baseline values during 6 months after surgery was longer. Pseudophakic cystoid macular edema was not identified in any group.

**CONCLUSIONS:** The use non-prostaglandin drops with preservative in patients with primary open-angle glaucoma does not affect pseudophakic cystoid macular edema development after uncomplicated phacoemulsification. Instillations of non-steroidal anti-inflammatory drops in the post-op period reduce the time of central retinal thickness recovery to baseline value.

**Keywords:** central retinal thickness; phacoemulsification; primary open-angle glaucoma; pseudophakic cystoid macular edema; non-steroidal anti-inflammatory drops; preservative; optical coherence tomography.

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Научная статья

# Влияние непростагландиновых гипотензивных препаратов с консервантом на толщину центральной зоны сетчатки после факоэмульсификации

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## АННОТАЦИЯ

**Актуальность.** Первичная открытоугольная глаукома — частое сопутствующее заболевание у пациентов с катарактой. Остаётся актуальной взаимосвязь применения гипотензивных капель, в частности непростагландиновых гипотензивных препаратов с консервантом, и изменение толщины центральной зоны сетчатки после факоэмульсификации с имплантацией интраокулярной линзы.

**Цель** — оценка толщины центральной зоны сетчатки и частоты развития псевдофакичного кистозного макулярного отёка после факоэмульсификации с имплантацией интраокулярной линзы у больных первичной открытоугольной глаукомой, получающих непростагландиновые гипотензивные препараты с консервантом.

**Материалы и методы.** В исследование включены 94 пациента (108 глаз) с катарактой, которые были распределены на 3 группы: I группа — 21 пациент (27 глаз), II группа — 21 пациент (23 глаза) с первичной открытоугольной глаукомой, получающие непростагландиновые препараты с консервантом, контрольная (III) группа — 52 пациента (58 глаз) без сопутствующей офтальмопатологии. Всем пациентам выполнена неосложнённая факоэмульсификация с имплантацией интраокулярной линзы. В послеоперационном периоде пациенты I группы получали антибактериальные препараты и кортикостероиды, пациенты II и III групп те же препараты и нестероидные противовоспалительные препараты. Проведена оценка толщины центральной зоны сетчатки с помощью оптической когерентной томографии до операции, через 2 нед. и через 2 и 6 мес. после оперативного вмешательства.

**Результаты.** Увеличение толщины центральной зоны сетчатки по сравнению с исходными значениями было более значимым в I группе, чем в II и III группах, а восстановление её до исходных значений в течение 6 мес. после факоэмульсификации было дольше. Псевдофакичный кистозный макулярный отёк ни в одной из групп не выявлен.

**Заключение.** Применение непростагландиновых препаратов с консервантом у пациентов с первичной открытоугольной глаукомой не влияет на развитие псевдофакичного кистозного макулярного отёка после проведения неосложнённой факоэмульсификации. Инстилляции нестероидных противовоспалительных препаратов в послеоперационном периоде сокращают сроки восстановления толщины центральной зоны сетчатки до исходных значений.

**Ключевые слова:** толщина центральной зоны сетчатки; факоэмульсификация; первичная открытоугольная глаукома; псевдофакичный кистозный макулярный отёк; нестероидные противовоспалительные препараты; консервант; оптическая когерентная томография.

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## BACKGROUND

Various systemic and local medications may lead to macular edema (ME) development. The safety issue when using hypotensive eyedrops in primary open-angle glaucoma patients after phacoemulsification (PE) remains relevant. Among ophthalmologic medications, ME could be caused by prostaglandin analogues [1–3], epinephrine [4, 5], dipivefrine [6], betaxolol [7], brimonidin [8], timolol, and preservatives used in composition of eyedrops [9]. Previously, the influence of prostaglandin analogues on the central retinal thickness and the prevalence of the pseudophakic cystoid macular edema were studied in primary open-angle glaucoma patients after uncomplicated PE [1–3, 10]. In our study, we evaluated in primary open-angle glaucoma patients the influence on the same parameters of non-prostaglandin hypotensive medications with preservative.

As known, the pathogenesis of the pseudophakic cystoid macular edema is still not understood [11, 12], but based on previous studies, the following risk factors of its development are specified:

- presence of ocular diseases such as uveitis [13], diabetic retinopathy [14–16], central retinal vein occlusion [14, 15], “wet” form of age-related macular degeneration [17], pigment retinitis [18], vitreoretinal traction syndrome [19], epiretinal membrane [14, 16, 20, 21], history of retinal detachment surgery [14, 21];
- intraoperative complications of PE [14, 15];
- perioperative use of prostaglandin analogues [20] and of beta-blockers [22];
- use for treatment of concomitant diseases of systemic drugs having among side-effects the risk of ME development — antidiabetic (thiazolidinediones [23]), antitumor medications (paclitaxel or docetaxel [24], tamoxifen, taxanes), interferons and niacin [25–29], as well as medications for multiple sclerosis therapy (fingolimod [30]).

Although glaucoma is not a risk factor for macular edema development after PE, it was shown that compensated intraocular pressure in primary open-angle glaucoma, which causes the lesion of the retinal nerve fiber layer and visual field defects, enhances its probability [31].

Preservatives as a component of eyedrops provide the solution's stability and prevent the bacterial contamination in non-disposable vials. Among basic preservatives, used in ophthalmological practice, range quaternary ammonium salts (benzalkonium chloride), alcohols, phenols (chlorobutanol, chlorocresol), esters of parahydroxybenzoic acid, metalorganic compounds of mercury (thimerosal). Most frequently in the composition of ophthalmic solutions, benzalkonium chloride is used.

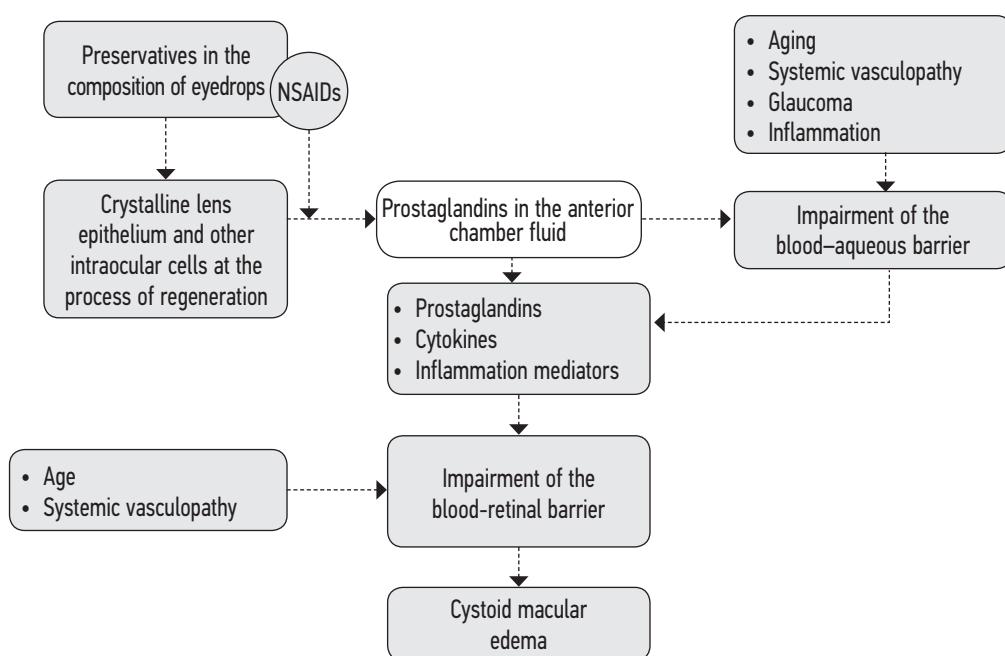
It is known that the preservative contained in the preparation, may exert a higher toxic effect than the active ingredient [32, 33]. In 2001, K. Miyake et al. published a study, in which for the first time it was proven that both timolol and preservative benzalkonium chloride cause the impairment of the blood-retinal barrier, and lead to the higher prevalence of the pseudophakic cystoid macular edema at the early postoperative period [9]. In one year, K. Miyake et al. proposed a hypothesis of the development of the so-called pseudophakic preservative maculopathy, according to which the main factor predisposing to the pseudophakic cystoid macular edema occurrence, is the added preservative benzalkonium chloride, and not the active substance of the preparation [34]. The preservative gets in contact with intraocular cells, in particular with the crystalline lens epithelium; the synthesis of prostaglandins, cytokines and other inflammatory mediators increases resulting in aggravation of the blood-retinal barrier impairment and increasing risk of the pseudophakic cystoid macular edema development (Fig. 1). The use of non-steroidal anti-inflammatory drugs in the postoperative period reduces the intensity of the processes described above.

Main visualization methods used for the ME diagnosis are the optical coherence tomography (OCT) and the fluorescein angiography (FA) of the retina. Of them, OCT is acknowledged as the most sensitive method to determine the localization and the amount of ME.

The aim is to estimate the central retinal thickness based on the OCT data in primary open-angle glaucoma patients using non-prostaglandin medications with preservative before and after PE with IOL implantation, as well as the prevalence of the pseudophakic cystoid macular edema development.

## MATERIALS AND METHODS

In the scope of the study, 94 patients (108 eyes) were examined, who were admitted to the Ophthalmology department No. 5 of the Saint Petersburg city budget health care institution “City multifunctional hospital No. 2” for cataract surgery. Patients were divided into 3 groups: group I (21 patient, 27 eyes, mean age  $74.5 \pm 6.9$  years), and group II (21 patient, 23 eyes, mean age  $75.2 \pm 6.1$  years) — primary open-angle glaucoma patients using non-prostaglandin medications with preservative; group III — a control one (52 patients, 58 eyes, mean age  $70.9 \pm 8.1$  years) without concomitant primary open-angle glaucoma. During the postoperative period, group I patients received antibacterial medications and steroids, group II and group III patients received the same therapy and non-steroidal anti-inflammatory drugs.



**Fig. 1.** The pathogenesis of “pseudophakic preservative maculopathy” by K. Miyake [35]

**Рис. 1.** Патогенез развития «псевдофакичной консервантной макулопатии» по К. Miyake [35]. НПВП — нестероидные противовоспалительные препараты

#### Inclusion criteria:

- cataract of various density;
- compensated primary open-angle glaucoma stages I–III on instillations of non-prostaglandin medications with preservative;
- absence of intraoperative complications (posterior capsule of the lens rupture, herniation of the vitreous, remnants of lens material, etc.).

#### Exclusion criteria:

- prostaglandin analogs instillations;
- performed posterior capsulorhexis;
- presence of uveitis, “wet” form of the age-related macular degeneration, macular hole, secondary glaucoma, vascular diseases of the retina, vitreomacular traction syndrome, pigment retinitis, epiretinal membrane, refractive amblyopia;
- history of any ophthalmic surgery or ocular injury;
- concomitant diabetes mellitus (DM);
- use of systemic medications which could cause ME development.

The diagnosis was made based on complaints, disease history, analysis of objective examination and imaging results. All patients underwent a standard ophthalmological examination (autorefractometry, visual acuity testing, tonometry, visual field testing, biomicroscopy, gonioscopy, and ophthalmoscopy), estimation of central retinal thickness based on the OCT data, before surgery, in 2 weeks, 2 and 6 months after PE with IOL implantation.

The IOP measurement was performed using the iCare TA01i device (ICare, Finland), the visual field testing (static threshold automated perimetry) was performed

using the perigraph “Pericom” (Optimed, Russia). The measurement of the central retinal thickness and data analysis were performed using the OCT machine (Optovue RTVue100, Optovue, USA) according to the Retina thickness map protocol (retinal thickness in 1 mm area) by the same investigator being unaware of clinical data.

In all patients, a standard PE using the Infiniti device (Alcon, USA) was performed, with implantation of various IOL models. Surgeries were performed by the same surgeon; any intraoperative complications were absent. At the end of the procedure, ultrasound, timing, and hydrodynamic parameters of PE were captured.

In the postoperative period, instillations of drops were carried out according to standard protocols: group I and group II patients on hypotensive monotherapy with timolol/betaxolol instilled the medication 2 times a day, dorzolamide — 3 times a day, combinations of dorzolamide/brinzolamide with timolol — 2 times a day. To all patients, levofloxacin 4 times a day during 2 weeks and dexamethasone 0.1% 4 times a day tapering for 4 weeks. To group I and group II patients, nepafenac 0.1% 3 times a day was prescribed for 4 weeks.

The statistical processing of the material was performed using the IBM SPSS Statistics 26 program. Mean value and standard deviation were calculated, differences between groups in OCT data before and after surgery were determined using the one-way analysis of variance (ANOVA). Differences between the results before surgery and post-operative ones at various times in each group were established using the dispersion analysis for repeated measurements (RMANOVA).

## RESULTS

In the majority of cases, timolol was the active component in the used non-prostaglandin hypotensive medications with preservative: in the group I — 84% (Fig. 2), in the group II — 73% (Fig. 3).

In 24 hours after surgery, in all patients, at biomicroscopy a moderate combined redness was noted, the cornea was transparent or a mild keratopathy was present, a mild (+++) opalescence was found in the anterior chamber fluid, pupillary reaction was preserved, IOL was in a correct position. The posterior capsule was preserved and intact.

The obtained results of PE parameters in groups are shown in the table 1. After surgery, in all patients, high visual functions (Table 2) were registered. The IOP in all patients was normalized during all the follow-up period, data are presented in the Table 3. The central retinal thickness values according to the OCT data before and after surgery are shown in the Table 4, and the dynamics of their changes at different timepoints — on Fig. 4 and 5.

In our study, the Shapiro-Wilk's test and the analysis of variance showed that numeric data correspond to normal distribution and to homogeneous variance. For all obtained results, multiple comparisons were made (LSD test with Bonferroni correction).

**Table 1.** The phacoemulsification parameters in studied groups,  $M \pm SD$ ,  $n = 108$

**Таблица 1.** Показатели параметров факоэмульсификации в исследуемых группах,  $M \pm SD$ ,  $n = 108$

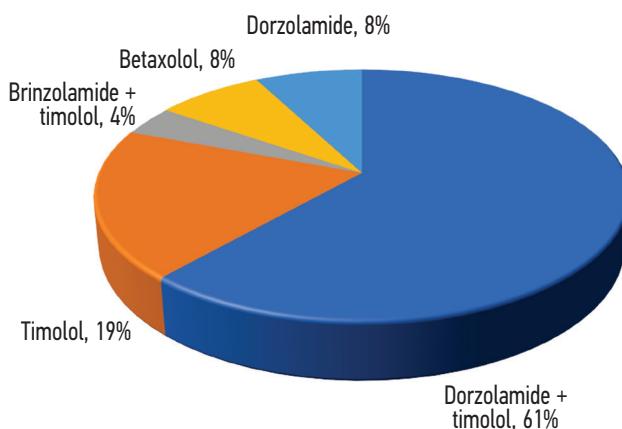
Group	CDE, kJ	BSS, ml	Time of the procedure, min
I, $n = 27$	$11.1 \pm 10.1$	$59.1 \pm 14.3$	$8.5 \pm 2.4$
II, $n = 23$	$10.2 \pm 6.1$	$55.4 \pm 13.9$	$6.8 \pm 1.1$
III (control), $n = 58$	$10.3 \pm 7.7$	$52.3 \pm 12.7$	$7.1 \pm 2.3$

Note. CDE — Cumulative dissipated energy; BSS — balanced salt solution (irrigation solution).

**Table 2.** Visual acuity testing data in studied groups,  $M \pm SD$ ,  $n = 108$

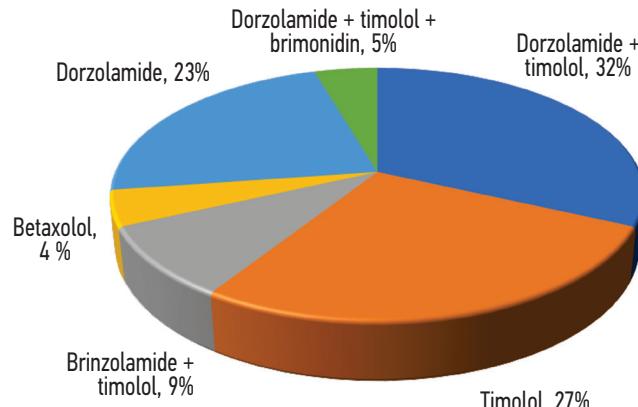
**Таблица 2.** Данные визометрии в исследуемых группах,  $M \pm SD$ ,  $n = 108$

Group	Before surgery	After surgery		
		in 2 weeks	in 2 months	in 6 months
I, $n = 27$	$0.2 \pm 0.2$	$0.8 \pm 0.2$	$0.9 \pm 0.2$	$0.9 \pm 0.1$
II, $n = 23$	$0.2 \pm 0.2$	$0.8 \pm 0.2$	$0.9 \pm 0.2$	$0.8 \pm 0.2$
III (control), $n = 58$	$0.3 \pm 0.2$	$0.9 \pm 0.1$	$0.9 \pm 0.1$	$0.9 \pm 0.1$



**Fig. 2.** Analysis of medications used in the first group (non-prostaglandin hypotensive drops with preservative)

**Рис. 2.** Анализ применяемых препаратов в I группе (непростагландиновые препараты с консервантом)



**Fig. 3.** Analysis of medications used in the second group (non-prostaglandin hypotensive drops with preservative + nonsteroidal anti-inflammatory drops)

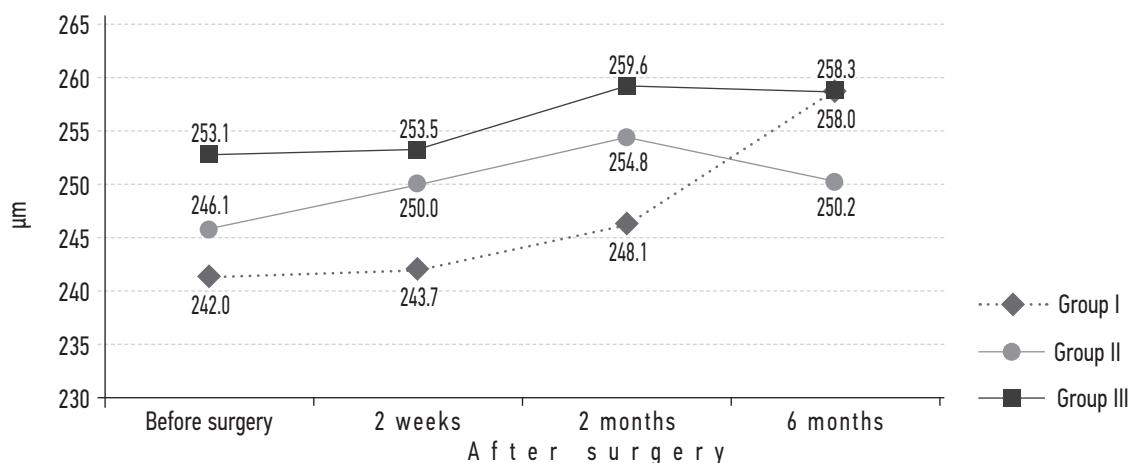
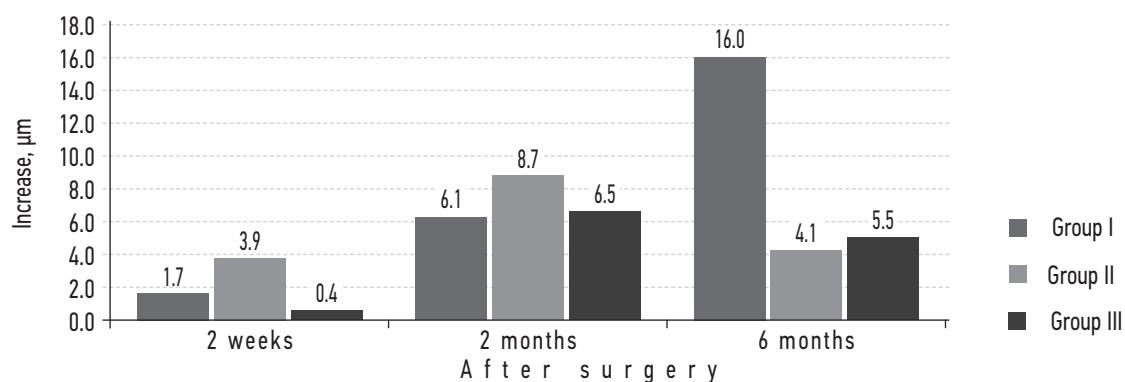
**Рис. 3.** Анализ применяемых препаратов во II группе (непростагландиновые препараты с консервантом + нестероидные противовоспалительные препараты)

**Table 3.** Intraocular pressure (mm Hg) data in studied groups measured with ICare tonometer,  $M \pm SD$ ,  $n = 108$ **Таблица 3.** Результаты внутриглазного давления по ICare в исследуемых группах,  $M \pm SD$ ,  $n = 108$ 

Group	Before surgery, mm Hg	After surgery, mm Hg		
		in 2 weeks	in 2 months	in 6 months
I, $n = 27$	$14.2 \pm 2.4$	$14.1 \pm 2.9$	$12.6 \pm 2.5$	$12.8 \pm 3.1$
II, $n = 23$	$15.9 \pm 4.7$	$13.3 \pm 2.5$	$13.3 \pm 3.5$	$13.9 \pm 3.0$
III (control), $n = 58$	$15.2 \pm 3.8$	$14.5 \pm 3.2$	$13.1 \pm 3.2$	$13.1 \pm 3.4$

**Table 4.** Central retinal thickness ( $\mu\text{m}$ ) in studied groups,  $M \pm SD$ ,  $n = 108$ **Таблица 4.** Толщина центральной зоны сетчатки в исследуемых группах,  $M \pm SD$ ,  $n = 108$ 

Group	Before surgery, $\mu\text{m}$	After surgery, $\mu\text{m}$		
		in 2 weeks	in 2 months	in 6 months
I, $n = 27$	$242.0 \pm 18.4$	$243.7 \pm 16.3$	$248.1 \pm 19.3$	$258.0 \pm 16.1$
II, $n = 23$	$246.1 \pm 22.4$	$250.0 \pm 20.9$	$254.8 \pm 18.9$	$250.2 \pm 21.7$
III (control), $n = 58$	$253.1 \pm 18.1$	$253.5 \pm 19.7$	$259.6 \pm 18.2$	$258.3 \pm 16.6$

**Fig. 4.** Dynamics of the central retinal thickness change in studied groups**Рис. 4.** Динамика изменения толщины центральной зоны сетчатки в исследуемых группах**Fig. 5.** Dynamics of the central retinal thickness change in studied groups compared to preoperative values at different observation periods**Рис. 5.** Динамика изменения толщины центральной зоны сетчатки в исследуемых группах по сравнению с дооперационными значениями в разные сроки наблюдения

A statistically significant difference was revealed at preoperative estimation of the central retinal thickness between the main group and the control one ( $p < 0.05$ ). At the postoperative period, at all follow-up times, there was no statistically significant difference ( $p > 0.05$ ).

In OCT data, between pre- and postoperative results, the following statistically significant differences were found: in the group I — in 2 and 6 months ( $p = 0.033$ ,  $p = 0.002$ , respectively), in the group II — in 2 weeks, 2 and 6 months ( $p = 0.015$ ,  $p = 0.008$ ,  $p = 0.029$ ,

respectively), in the group III (control group) — in 2 months after PE ( $p = 0.003$ ).

Within the framework of the study, in none of the patients, a pseudophakic cystoid macular edema was found.

Comparing the PE parameters between the main groups and the control one, a statistically significant difference was established only concerning the time, spent for the surgical procedure ( $p < 0.05$ ).

## DISCUSSION

In our study, all patients did not have any systemic and ophthalmic diseases enhancing the likelihood of the pseudophakic cystoid macular edema development. It is recognized that in diabetic patients with good glycemic control, the risk of complications after cataract surgery, including the pseudophakic cystoid macular edema, does not increase [31]. However, in some systemic diseases, including diabetes mellitus, the permeability of the vascular wall increases [35], and as a consequence the risk of ME development increases as well. For this reason, we excluded diabetic patients from the investigation.

We revealed that the central retinal thickness in patients from all groups gradually increased during 6 months after surgery. In group I patients (without instillations of non-steroidal anti-inflammatory drugs), there was no tendency to its recovery to baseline values, in contrast to patients of groups II and III treated by non-steroidal anti-inflammatory drugs (Table 4, Fig. 4, 5). At the same time, in none of the groups, baseline preoperative values of the retinal thickness in the fovea were reached during 6 months. The received data confirm once more the conclusions of the study by S.Y. Astakhov et al. [10], that in patients treated by hypotensive medications, after PE, the central retinal thickness returns to baseline values only during about one year.

In several studies, the thickening of the central retinal area within normal limits was found after PE during 6 months when using antibacterial and steroid therapy [36, 37]. However, some authors received opposite effects in terms of decreasing retinal thickness

in the fovea after PE in 2, 4 and 8 weeks. This could be related to the measurement's error due to the lens opacification and the transparency restoration of optic media [40–42]. We also revealed that the use of non-steroidal anti-inflammatory medications enhances the restoration of retinal thickness during the post-operative period.

## CONCLUSIONS

The use of non-prostaglandin hypotensive eyedrops with preservative in patients with primary open-angle glaucoma does not influence the pseudophakic cystoid macular edema development after PE, in the absence of intra- and post-operative complications. Instillations of non-steroidal anti-inflammatory drugs during the post-operative period reduce the time of central retinal area thickness restoration to baseline values.

## ADDITIONAL INFORMATION

**Authors' contribution.** Thereby, all authors made a substantial contribution to the conception of the study, acquisition, analysis, interpretation of data for the work, drafting and revising the article, final approval of the version to be published and agree to be accountable for all aspects of the study. Contribution of each author: X. Wang — concept and design of the study, collection and processing of materials, analysis of the data obtained, writing of the text, literature review; S.Yu. Astakhov — concept and design of the study, analysis of the data obtained; A.S. Cherkashina — writing of the text, literature review; L.K. Anikina — diagnostic studies, writing of the text; T.R. Parasunko — collection and processing of materials, literature review; V.V. Potemkin — surgical treatment, analysis of the data obtained; A.R. Potemkina — diagnostic studies.

**Competing interests.** The authors declare that they have no competing interests.

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**Consent for publication.** Written consent was obtained from the patients for publication of relevant medical information within the manuscript.

## REFERENCES

- Miyake K, Ibaraki N. Prostaglandins and cystoid macular edema. *Surv Ophthalmol*. 2002;47(S1):S203–S218. DOI: 10.1016/s0039-6257(02)00294-1
- Warwar RE, Bullock JD, Ballal D. Cystoid macular edema and anterior uveitis associated with latanoprost use: experience and incidence in a retrospective review of 94 patients. *Ophthalmology*. 1998;105(2):263–268. DOI: 10.1016/s0161-6420(98)92977-3
- Lima MC, Paranhos A Jr, Salim S, et al. Visually significant cystoid macular edema in pseudophakic and aphakic patients with glaucoma receiving latanoprost. *J Glaucoma*. 2000;9(4):317–321. DOI: 10.1097/00061198-200008000-00006
- Michels RG, Maumenee AE. Cystoid macular edema associated with topically applied epinephrine in aphakic eyes. *Am J Ophthalmol*. 1975;80(3):379–388. DOI: 10.1016/0002-9394(75)90522-x
- Kolker AE, Becker B. Epinephrine maculopathy. *Arch Ophthalmol*. 1968;79(5):552–562. DOI: 10.1001/archopt.1968.03850040554010
- Mehelas TJ, Kollarits CR, Martin WG. Cystoid macular edema presumably induced by dipivefrin hydrochloride (Propine). *Am J Ophthalmol*. 1982;94(5):682. DOI: 10.1016/0002-9394(82)90019-8
- Hesse RJ, Swan JL. Aphakic cystoid macular edema secondary to betaxolol therapy. *Ophthalmic Surg Lasers Imaging Retina*. 1988;19(8):562–564. DOI: 10.3928/1542-8877-19880801-10

- 8.** Kim P, Lertsumitkul S. Cystoid macular oedema associated with brimonidine therapy. *Clin Exp Ophthalmol.* 2003;31(2):165–166. DOI: 10.1046/j.1442-9071.2003.00628.x
- 9.** Miyake K, Octa I, Ibaraki N, et al. Enhanced disruption of the blood-aqueous barrier and the incidence of angiographic cystoid macular edema by topical timolol and its preservative in early post-operative pseudophakia. *Arch Ophthalmol.* 2001;119(3):387–394. DOI: 10.1001/archoph.119.3.387
- 10.** Astakhov SY, Astakhov YS, Gobedzhishvili MV. The influence of prostaglandin analogs on the retinal thickness after phacoemulsification with intraocular lens implantation in primary open-angle glaucoma patients. *Ophthalmology Reports.* 2014;7(3):73–76. (In Russ.) DOI: 10.17816/OV2014373-76
- 11.** Irvine SR. A newly defined vitreous syndrome following cataract surgery: Interpreted according to recent concepts of the structure of the vitreous. The seventh Francis I. Proctor Lecture. *Am J Ophthalmol.* 1953;36(5):599–619. DOI: 10.1016/0002-9394(53)90302-x
- 12.** Flach AJ. The incidence, pathogenesis and treatment of cystoid macular edema following cataract surgery. *Trans Am Ophthalmol Soc.* 1998;96:557–634.
- 13.** Lardenoye CWTA, van Kooij B, Rothova A. Impact of macular edema on visual acuity in uveitis. *Ophthalmology.* 2006;113(8):1446–1449. DOI: 10.1016/j.ophtha.2006.03.027
- 14.** Chu CJ, Johnston RL, Buscombe C, et al. Risk factors and incidence of macular edema after cataract surgery: a database study of 81984 eyes. *Ophthalmology.* 2016;123(2):316–323. DOI: 10.1016/j.ophtha.2015.10.001
- 15.** Rotsos TG, Moschos MM. Cystoid macular edema. *Clin Ophthalmol.* 2008;2(4):919–930. DOI: 10.2147/ophth.s4033
- 16.** Kokorev VL. The analysis of risk factors of development of macular edema after phacoemulsification. *Ophthalmology in Russia.* 2019;16(2):185–191. (In Russ.) DOI: 10.18008/1816-5095-2019-2-185-191
- 17.** Shah N, Maguire MG, Martin DF, et al. Angiographic cystoid macular edema and outcomes in the comparison of age-related macular degeneration treatments trials. *Ophthalmology.* 2016;123(4):858–864. DOI: 10.1016/j.ophtha.2015.11.030
- 18.** Hajali M, Fishman GA, Anderson RJ. The prevalence of cystoid macular oedema in retinitis pigmentosa patients determined by optical coherence tomography. *Br J Ophthalmol.* 2008;92(8):1065–1068. DOI: 10.1136/bjo.2008.138560
- 19.** Johnson MW. Perifoveal vitreous detachment and its macular complications. *Trans Am Ophthalmol Soc.* 2005;103:537–567.
- 20.** Henderson BA, Kim JY, Ament CS, et al. Clinical pseudophakic cystoid macular edema: risk factors for development and duration after treatment. *J Cataract Refract Surg.* 2007;33(9):1550–1558. DOI: 10.1016/j.jcrs.2007.05.013
- 21.** Schaub F, Adler W, Enders P, et al. Preexisting epiretinal membrane is associated with pseudophakic cystoid macular edema. *Graefe's Arch Clin Exp Ophthalmol.* 2018;256:909–917. DOI: 10.1007/s00417-018-3954-4
- 22.** Wendel C, Zakrzewski H, Carleton B, et al. Association of post-operative topical prostaglandin analog or beta-blocker use and incidence of pseudophakic cystoid macular edema. *J Glaucoma.* 2018;27(5):402–406. DOI: 10.1097/IJG.0000000000000929
- 23.** Idris I, Warren G, Donnelly R. Association between thiazolidinedione treatment and risk of macular edema among patients with type 2 diabetes. *Arch Int Med.* 2012;172(13):1005–1011. DOI: 10.1001/archinternmed.2012.1938
- 24.** Yokoe T, Fukada I, Kobayashi K, et al. Cystoid macular edema during treatment with paclitaxel and bevacizumab in a patient with metastatic breast cancer: a case report and literature review. *Case Rep Oncol.* 2017;10(2):605–612. DOI: 10.1159/000477897
- 25.** Gass JDM. Nicotinic acid maculopathy. *Am J Ophthalmol.* 1973;76(4):500–510. DOI: 10.1016/0002-9394(73)90738-1
- 26.** Millay RH, Klein ML, Illingworth DR. Niacin maculopathy. *Ophthalmology.* 1988;95(7):930–936. DOI: 10.1016/s0161-6420(88)33073-3
- 27.** Fraunfelder FW, Fraunfelder FT, Illingworth DR. Adverse ocular effects associated with niacin therapy. *Br J Ophthalmol.* 1995;79(1):54–56. DOI: 10.1136/bjo.79.1.54
- 28.** Callanan D, Blodi BA, Martin DF. Macular edema associated with nicotinic acid (niacin). *JAMA.* 1998;279(21):1702–1702. DOI: 10.1001/jama.279.21.1702-b
- 29.** Domanico D, Verboschi F, Altimari S, et al. Ocular effects of niacin: a review of the literature. *Med Hypothesis Discov Innov Ophthalmol.* 2015;4(2):64–71.
- 30.** Jain N, Bhatti MT. Fingolimod-associated macular edema: incidence, detection, and management. *Neurology.* 2012;78(9):672–680. DOI: 10.1212/WNL.0b013e318248dea
- 31.** Lee KM, Lee EJ, Kim T-W, Kim H. Pseudophakic macular edema in primary open-angle glaucoma: a prospective study using spectral-domain optical coherence tomography. *Am J Ophthalmol.* 2017;179:97–109. DOI: 10.1016/j.ajo.2017.05.001
- 32.** Williams DE, Nguyen KD, Shapourifar-Tehrani S, et al. Effects of timolol, betaxolol, and levobunolol on human tenon's fibroblasts in tissue culture. *Investig Ophthalmol Vis Sci.* 1992;33(7):2233–2241.
- 33.** Baudouin C, Pisella P-J, Fillacier K, et al. Ocular surface inflammatory changes induced by topical antiglaucoma drugs: human and animal studies. *Ophthalmology.* 1999;106(3):556–563. DOI: 10.1016/S0161-6420(99)90116-1
- 34.** Miyake K, Ibaraki N, Goto Y, et al. ESCRS Binkhorst lecture 2002: pseudophakic preservative maculopathy. *J Cataract Refract Surg.* 2003;29(9):1800–1810. DOI: 10.1016/s0886-3350(03)00560-1
- 35.** Sánchez-Tocino H, Alvarez-Vidal A, Maldonado MJ, et al. Retinal thickness study with optical coherence tomography in patients with diabetes. *Invest Ophthalmol Vis Sci.* 2002;43(5):1588–1594.
- 36.** von Jagow B, Ohrloff C, Kohnen T. Macular thickness after uneventful cataract surgery determined by optical coherence tomography. *Graefes Arch Clin Exp Ophthalmol.* 2007;245:1765–1771. DOI: 10.1007/s00417-007-0605-6
- 37.** Perente I, Utine CA, Ozturker C, et al. Evaluation of macular changes after uncomplicated phacoemulsification surgery by optical coherence tomography. *Curr Eye Res.* 2007;32(3):241–247. DOI: 10.1080/02713680601160610
- 38.** Ching H-Y, Wong AC, Wong C-C, et al. Cystoid macular oedema and changes in retinal thickness after phacoemulsification with optical coherence tomography. *Eye.* 2006;20(3):297–303. DOI: 10.1038/sj.eye.6701864
- 39.** Wang X, Astakhov SY, Potemkin VV, et al. Evaluation of retinal thickness and of pseudophakic cystoid macular edema incidence in patients with primary open-angle glaucoma treated with prostaglandin analogues. *Ophthalmology Reports.* 2021;14(2):17–26. (In Russ.) DOI: 10.17816/OV64116
- 40.** Kurt A, Kılıç R. The effects of uncomplicated cataract surgery on retinal layer thickness. *J Ophthalmol.* 2018;2018:7218639. DOI: 10.1155/2018/7218639

**41.** Cagini C, Fiore T, Laccheri B, et al. Macular thickness measured by optical coherence tomography in a healthy population before and after uncomplicated cataract phacoemulsification surgery. *Curr Eye Res.* 2009;34(12):1036–1041. DOI: 10.3109/02713680903288937

**42.** Falcão MS, Gonçalves N, Freitas-Costa P, et al. Choroidal and macular thickness changes induced by cataract surgery. *Clin Ophthalmol.* 2013;8:55–60. DOI: 10.2147/OPTH.S53989

## СПИСОК ЛИТЕРАТУРЫ

1. Miyake K., Ibaraki N. Prostaglandins and cystoid macular edema // *Surv Ophthalmol.* 2002. Vol. 47, No. S1. P. S203–S218. DOI: 10.1016/s0039-6257(02)00294-1
2. Warwar R.E., Bullock J.D., Ballal D. Cystoid macular edema and anterior uveitis associated with latanoprost use: experience and incidence in a retrospective review of 94 patients // *Ophthalmology.* 1998. Vol. 105, No. 2. P. 263–268. DOI: 10.1016/s0161-6420(98)92977-3
3. Lima M.C., Paranhos A. Jr., Salim S., et al. Visually significant cystoid macular edema in pseudophakic and aphakic patients with glaucoma receiving latanoprost // *J Glaucoma.* 2000. Vol. 9, No. 4. P. 317–321. DOI: 10.1097/00061198-200008000-00006
4. Michels R.G., Maumenee A.E. Cystoid macular edema associated with topically applied epinephrine in aphakic eyes // *Am J Ophthalmol.* 1975. Vol. 80, No. 3. P. 379–388. DOI: 10.1016/0002-9394(75)90522-x
5. Kolker A.E., Becker B. Epinephrine maculopathy // *Arch Ophthalmol.* 1968. Vol. 79, No. 5. P. 552–562. DOI: 10.1001/archophth.1968.03850040554010
6. Mehelas T.J., Kollarits C.R., Martin W.G. Cystoid macular edema presumably induced by dipivefrin hydrochloride (Propine) // *Am J Ophthalmol.* 1982. Vol. 94, No. 5. P. 682. DOI: 10.1016/0002-9394(82)90019-8
7. Hesse R.J., Swan J.L. Aphakic cystoid macular edema secondary to betaxolol therapy // *Ophthalmic Surg Lasers Imaging Retina.* 1988. Vol. 19, No. 8. P. 562–564. DOI: 10.3928/1542-8877-19880801-10
8. Kim P., Lertsumitkul S. Cystoid macular oedema associated with brimonidine therapy // *Clin Exp Ophthalmol.* 2003. Vol. 31, No. 2. P. 165–166. DOI: 10.1046/j.1442-9071.2003.00628.x
9. Miyake K., Octa I., Ibaraki N., et al. Enhanced disruption of the blood-aqueous barrier and the incidence of angiographic cystoid macular edema by topical timolol and its preservative in early post-operative pseudophakia // *Arch Ophthalmol.* 2001. Vol. 119, No. 3. P. 387–394. DOI: 10.1001/archophth.119.3.387
10. Астахов С.Ю., Астахов Ю.С., Гобеджишвили М.В. Влияние лечения аналогами простагландинов на толщину сетчатки после факоэмультсионной имплантации интраокулярной линзы у больных первичной открытогоугольной глаукомой // Офтальмологические ведомости. 2014. Т. 7, № 3. С. 73–76. DOI: 10.17816/OV2014373-76
11. Irvine S.R. A newly defined vitreous syndrome following cataract surgery: Interpreted according to recent concepts of the structure of the vitreous. The seventh Francis I. Proctor Lecture // *Am J Ophthalmol.* 1953. Vol. 36, No. 5. P. 599–619. DOI: 10.1016/0002-9394(53)90302-x
12. Flach A.J. The incidence, pathogenesis and treatment of cystoid macular edema following cataract surgery // *Trans Am Ophthalmol Soc.* 1998. Vol. 96. P. 557–634.
13. Lardenoye C.W.T.A., van Kooij B., Rothova A. Impact of macular edema on visual acuity in uveitis // *Ophthalmology.* 2006. Vol. 113, No. 8. P. 1446–1449. DOI: 10.1016/j.ophtha.2006.03.027
14. Chu C.J., Johnston R.L., Buscombe C., et al. Risk factors and incidence of macular edema after cataract surgery: a database study of 81984 eyes // *Ophthalmology.* 2016. Vol. 123, No. 2. P. 316–323. DOI: 10.1016/j.ophtha.2015.10.001
15. Rotsos T.G., Moschos M.M. Cystoid macular edema // *Clin Ophthalmol.* 2008. Vol. 2, No. 4. P. 919–930. DOI: 10.2147/ophth.s4033
16. Кокорев В.Л. Анализ факторов риска развития макулярного отёка после факоэмультсионной катаракты // Офтальмология. 2019. Т. 16, № 2. С. 185–191. DOI: 10.18008/1816-5095-2019-2-185-191
17. Shah N., Maguire M.G., Martin D.F., et al. Angiographic cystoid macular edema and outcomes in the comparison of age-related macular degeneration treatments trials // *Ophthalmology.* 2016. Vol. 123, No. 4. P. 858–864. DOI: 10.1016/j.ophtha.2015.11.030
18. Hajali M., Fishman G.A., Anderson R.J. The prevalence of cystoid macular oedema in retinitis pigmentosa patients determined by optical coherence tomography // *Br J Ophthalmol.* 2008. Vol. 92, No. 8. P. 1065–1068. DOI: 10.1136/bjo.2008.138560
19. Johnson M.W. Perifoveal vitreous detachment and its macular complications // *Trans Am Ophthalmol Soc.* 2005. Vol. 103. P. 537–567.
20. Henderson B.A., Kim J.Y., Ament C.S., et al. Clinical pseudophakic cystoid macular edema: risk factors for development and duration after treatment // *J Cataract Refract Surg.* 2007. Vol. 33, No. 9. P. 1550–1558. DOI: 10.1016/j.jcrs.2007.05.013
21. Schaub F., Adler W., Enders P., et al. Preexisting epiretinal membrane is associated with pseudophakic cystoid macular edema // *Graefe's Arch Clin Exp Ophthalmol.* 2018. Vol. 256. P. 909–917. DOI: 10.1007/s00417-018-3954-4
22. Wendel C., Zakrzewski H., Carleton B., et al. Association of post-operative topical prostaglandin analog or beta-blocker use and incidence of pseudophakic cystoid macular edema // *J Glaucoma.* 2018. Vol. 27, No. 5. P. 402–406. DOI: 10.1097/IJG.0000000000000929
23. Idris I., Warren G., Donnelly R. Association between thiazolidinedione treatment and risk of macular edema among patients with type 2 diabetes // *Arch Int Med.* 2012. Vol. 172, No. 13. P. 1005–1011. DOI: 10.1001/archinternmed.2012.1938
24. Yokoe T., Fukada I., Kobayashi K., et al. Cystoid macular edema during treatment with paclitaxel and bevacizumab in a patient with metastatic breast cancer: a case report and literature review // *Case Rep Oncol.* 2017. Vol. 10, No. 2. P. 605–612. DOI: 10.1159/0004778972.
25. Gass J.D.M. Nicotinic acid maculopathy // *Am J Ophthalmol.* 1973. Vol. 76, No. 4. P. 500–510. DOI: 10.1016/0002-9394(73)90738-1
26. Millay R.H., Klein M.L., Illingworth D.R. Niacin maculopathy // *Ophthalmology.* 1988. Vol. 95, No. 7. P. 930–936. DOI: 10.1016/s0161-6420(88)33073-3
27. Fraunfelder F.W., Fraunfelder F.T., Illingworth D.R. Adverse ocular effects associated with niacin therapy // *Br J Ophthalmol.* 1995. Vol. 79, No. 1. P. 54–56. DOI: 10.1136/bjo.79.1.54
28. Callanan D., Blodi B.A., Martin D.F. Macular edema associated with nicotinic acid (niacin) // *JAMA.* 1998. Vol. 279, No. 21. P. 1702–1702. DOI: 10.1001/jama.279.21.1702-b

- 29.** Domanico D., Verboschi F., Altimari S., et al. Ocular effects of niacin: a review of the literature // Med Hypothesis Discov Innov Ophthalmol. 2015. Vol. 4, No. 2. P. 64–71.
- 30.** Jain N., Bhatti M.T. Fingolimod-associated macular edema: incidence, detection, and management // Neurology. 2012. Vol. 78, No. 9. P. 672–680. DOI: 10.1212/WNL.0b013e318248deea
- 31.** Lee K.M., Lee E.J., Kim T.-W., Kim H. Pseudophakic macular edema in primary open-angle glaucoma: a prospective study using spectral-domain optical coherence tomography // Am J Ophthalmol. 2017. Vol. 179. P. 97–109. DOI: 10.1016/j.ajo.2017.05.001
- 32.** Williams D.E., Nguyen K.D., Shapourifar-Tehrani S., et al. Effects of timolol, betaxolol, and levobunolol on human tenon's fibroblasts in tissue culture // Investig Ophthalmol Vis Sci. 1992. Vol. 33, No. 7. P. 2233–2241.
- 33.** Baudouin C., Pisella P.-J., Fillacier K., et al. Ocular surface inflammatory changes induced by topical antiglaucoma drugs: human and animal studies // Ophthalmology. 1999. Vol. 106, No. 3. P. 556–563. DOI: 10.1016/S0161-6420(99)90116-1
- 34.** Miyake K., Ibaraki N., Goto Y., et al. ESCRS Binkhorst lecture 2002: pseudophakic preservative maculopathy // J Cataract Refract Surg. 2003. Vol. 29, No. 9. P. 1800–1810. DOI: 10.1016/s0886-3350(03)00560-1
- 35.** Sánchez-Tocino H., Alvarez-Vidal A., Maldonado M.J., et al. Retinal thickness study with optical coherence tomography in patients with diabetes // Invest Ophthalmol Vis Sci. 2002. Vol. 43, No. 5. P. 1588–1594.
- 36.** von Jagow B., Ohrloff C., Kohnen T. Macular thickness after uneventful cataract surgery determined by optical coherence tomography // Graefe's Arch Clin Exp Ophthalmol. 2007. Vol. 245. P. 1765–1771. DOI: 10.1007/s00417-007-0605-6
- 37.** Perente I., Utine C.A., Ozturker C., et al. Evaluation of macular changes after uncomplicated phacoemulsification surgery by optical coherence tomography // Curr Eye Res. 2007. Vol. 32, No. 3. P. 241–247. DOI: 10.1080/02713680601160610
- 38.** Ching H.-Y., Wong A.C., Wong C.-C., et al. Cystoid macular oedema and changes in retinal thickness after phacoemulsification with optical coherence tomography // Eye. 2006. Vol. 20, No. 3. P. 297–303. DOI: 10.1038/sj.eye.6701864
- 39.** Ван С., Астахов С.Ю., Потемкин В.В., и др. Оценка толщины сетчатки и частоты развития псевдофакичного кистозного макулярного отёка у больных ПОУГ, получающих аналоги простагландинов // Офтальмологические ведомости. 2021. Т. 14, № 2. С. 17–26. DOI: 10.17816/OV64116
- 40.** Kurt A., Kılıç R. The effects of uncomplicated cataract surgery on retinal layer thickness // J Ophthalmol. 2018. Vol. 2018. ID7218639. DOI: 10.1155/2018/7218639
- 41.** Cagini C., Fiore T., Laccheri B., et al. Macular thickness measured by optical coherence tomography in a healthy population before and after uncomplicated cataract phacoemulsification surgery // Curr Eye Res. 2009. Vol. 34, No. 12. P. 1036–1041. DOI: 10.3109/02713680903288937
- 42.** Falcão M.S., Gonçalves N., Freitas-Costa P., et al. Choroidal and macular thickness changes induced by cataract surgery // Clin Ophthalmol. 2013. Vol. 8. P. 55–60. DOI: 10.2147/OPTH.S53989

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