IJIMM, Volume 3, Issue 1, 2025 ISSN: 2995-5319
http://medicaljournals.eu/index.php/IJIMM/issue/view/3

INTERNATIONAL JOURNAL OF INTEGRATIVE AND MODERN MEDICINE

Peculiarities of the Clinical Picture, Pathogenesis and Drug Treatment of Secondary Phacogenic Glaucoma

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Annotation: Pathologies of the lens often lead to increased intraocular pressure and the development of secondary glaucoma. The article presents a description of the clinical picture and pathogenetic aspects of the development of various forms of secondary phacogenic glaucoma: phacotopic, phacomorphic and phacolytic. A scheme of intensive drug therapy aimed at reducing intraocular pressure and neuroprotection of the retina is proposed. Based on the results of the study, an assessment of the effectiveness of hypotensive treatment was made, as well as the possibility of long-term drug control of ophthalmotonus against the background of lens pathologies. Ophthalmological examinations were performed in daylight, artificial light, and in the dark. Results and discussion. Studying the risk factors for secondary glaucoma, it was found that in 55 out of 142 patients (39% of cases), the development of secondary glaucoma was associated with lens pathologies (phacogenic glaucoma): luxation or subluxation of the lens, swelling cataract, lysis of hypermature cataract. Diagnostic criteria and clinical picture of glaucoma of phacogenic etiology were studied in 55 patients (68 eyes – 100%). Average IOP values during tonometry were 37.13 ± 4.45 mm Hg. The study revealed that in 42 out of 55 patients (42 eyes – 76.4% of cases) secondary glaucoma against the background of lens pathologies was noted unilaterally. In 13 patients (26 eyes – 23.6% of cases) phacogenic secondary glaucoma was noted bilaterally.

Keywords: intraocular pressure, glaucoma, cataract, lens, luxation.

Introduction. Glaucoma is one of the most pressing problems in ophthalmology. The diversity of clinical forms, various pathogenetic mechanisms of pathology development, untimely visit to a veterinary specialist - all this aggravates a disease that is already difficult to treat. Glaucoma is a large group of diseases of the organ of vision, characterized by a constant or periodic increase in intraocular pressure, due to a violation of the outflow of aqueous humor from the eye, and with the subsequent development of specific defects of the visual field and glaucomatous atrophy of the optic nerve [1]. In general, the clinical picture of an acute attack of glaucoma consists of five phases: the trigger phase, the compression phase, the reactive phase, the strangulation phase and the phase of regression [2]. The first phase or the trigger phase is characterized by a blockade of the angle of the anterior chamber by the root of the iris. The clinical picture of this phase is very poor: the bombage of the iris increases, the angle of the anterior chamber, where it was previously open, closes. The second phase is the compression phase and is characterized by increased bombage of the root part of the iris. The anterior chamber angle bay becomes inaccessible for penetration of chamber moisture into it. The moisture present in the anterior chamber angle flows out through the canal of the helmet. The iris sags even more and adjoins the corneal sclera. The trabecula shifts and thereby blocks the lumen of the sinus. All this leads to a sharp increase in intraocular pressure. The vision of the diseased eye deteriorates sharply, pain appears in the eye, around the eye and superciliary arch. Some patients note rainbow circles when looking at a light source. During examination, the ophthalmologist discovers stagnant injection of the eyeball, edema of the epithelium and stroma, decreased sensitivity of the cornea. The anterior chamber is shallow. The pupil is dilated. Bombage of the iris. Mydriasis. Gonioscopic picture: the angle of the anterior chamber is closed. Intraocular pressure continues to increase. The third phase is the reactive phase. The clinical picture in this phase increases even more, the permeability of the vessels increases. Edema of the iris occurs. The fluid of the anterior chamber is cloudy due to the large amount of protein. The intraocular pressure is very high. On the fundus: the optic disc is edematous,

the borders are blurred, the veins are dilated. Nausea and vomiting are possible. [3]. The fourth phase is the strangulation phase. As a result of the above-presented clinical pictures of an acute attack of angle-closure glaucoma (aseptic inflammation and necrosis in conditions of circulatory disorders, and mechanical effect of high intraocular pressure), segmental atrophy of the iris, dilation and dislocation of the pupil with the absence of pupillary and accommodative reactions, formation of posterior synechiae along the pupillary edge, goniosynechia, which cause organic blockade of the anterior chamber angle, occur. With a pronounced inflammatory reaction, pigment spraying and significant deposition of fibrin or fibrinous exudate in the anterior chamber and in the pupil area are often noted. The fifth phase is the phase of regression. It is characterized by significant inhibition of the production of aqueous humor in the eye chamber, which leads to a decrease in intraocular pressure in the posterior chamber, a decrease in iris bombage, its posterior displacement, and partial opening of the anterior chamber angle. However, with severe goniosynechiae, opening of the anterior chamber angle is almost impossible. And segmental atrophy of the iris, deformation and paralytic dilation of the pupil, goniosynechiae remain forever [4]. Cataract is a pathology of the lens, characterized by varying degrees of clouding of the lens substance or its capsule. According to the time of occurrence of cataracts, it is customary to divide them into two main groups: congenital and acquired. Acquired forms of lens clouding are divided into age-related, traumatic, complicated, radiation, etc. The thickness of the lens in adults is 3.6 mm [5]. It has been established that in the initial period of age-related cataracts, the water content increases, then the concentration of sodium, calcium, and chlorine ions increases, the content of amino acids increases, and at the same time the amount of water-soluble proteins, sulfhydryl groups, ATP, and glucose decreases. tathione, ascorbic acid, riboflavin. In the initial age-related cataract, the activity of enzymes involved in the glycolysis process decreases, oxygen consumption decreases sharply, and peroxidation is disrupted [6]. Age-related cataract is common among the elderly. Clouding of the lens can begin with the cortex or the nucleus. Depending on the location of the clouding in the lens, cataracts are divided into cortical and nuclear. According to the degree of maturity, age-related cataract has four forms: initial, immature, mature and overmature forms. In the initial cataract, clouding begins at the equator in the form of a spoke. It does not affect visual acuity. Patients complain of "flies" before the eyes, spots, diplopia, polyopia. The duration of this stage varies from person to person. Then comes the second stage - immature or swelling cataract. In this case, the clouding affects almost the entire cortical layer of the lens. Patients complain of a sharp decrease in visual acuity. Swelling of the lens often leads to a narrowing of the anterior chamber angle, which complicates the outflow of intraocular fluid and causes an increase in intraocular pressure. Subsequently, water is gradually lost, the opacity becomes more homogeneous. This state of the lens is called mature cataract or the third stage of the process. Object vision in this case drops to the level of sensation of light with the correct projection and color perception is preserved if there is no retinal pathology in the fundus. The last stage of age-related cataract is hypermature cataract. With hypermature cataract, there is also a risk of developing phacolytic glaucoma or iridocyclitis [7]. Modern treatment of cataracts is surgical [8]. The indication for surgical treatment is a decrease in visual acuity, leading to a limitation of working capacity or discomfort in everyday life. In this regard, hypermature cataracts are currently almost never found among the population. More often, patients come with immature, much less often - mature cataracts. The operation is performed at the microsurgical level using microsurgical techniques. Phacomorphic glaucoma. It develops due to swelling of the cortical layers of the lens in immature cataracts and is a type of secondary glaucoma. The clinical picture 15 Science of Life and Health No. 3, 2020 1963 occurs as an acute attack of primary angle-closure glaucoma. Unlike the latter, with phacomorphic glaucoma, the thickness of the lens increases. In addition, the conditions for the development of phacomorphic glaucoma are: nanophthalmos (small anterior - posterior size of the eyeball), hypermetropic refraction, small anterior chamber. According to the literature, in individuals with phacomorphic glaucoma, the anterior-posterior axis of the eyeball fluctuates within 19 mm - 23 mm, refraction is hypermetropia more than two diopters, the thickness of the lens is from 4.7 to 6 mm or more. Ultrasound biomicroscopy has established the anterior position of the ciliary body [9, 15]. According to Mustafina Zh.G., Imantaeva M.B., ethnic Kazakhs, compared with Caucasians, are statistically significantly more likely to have

hypermetropic refraction, a shallow anterior chamber, and a short anterior-posterior size of the eyeball [16, 17]. In this regard, phacomorphic glaucoma is more common among them and is one of the common types of glaucoma, which leads to visual impairment. Descriptions of cases of phacomorphic glaucoma in the literature are rare and are often unilateral [2, 3]. In this regard, we decided to present a case where the patient had bilateral phacomorphic glaucoma. Moreover, the increase in intraocular pressure in the second eye developed against the background of the initiated hypotensive and anti-edematous therapy. Materials and methods. Thus, a 63-year-old Kazakh woman, Zh., came to the emergency room of the Central City Clinical Hospital of Almaty at 4 p.m. complaining of pain in the right eye radiating to the same half of the head, nausea, and decreased visual acuity in this eye. According to the anamnesis, pain in the right eye appeared two weeks ago. She did not receive treatment at home and did not see a doctor. Due to the intensification of pain in the eye spreading to the right half of the head, a sharp drop in visual acuity in this eye, nausea, and a single vomiting, she went to the clinic. From the anamnesis of life: the patient wore glasses for work at +2.0 D since the age of 38. In recent years, she worked at close range without glasses. A year ago, she consulted an ophthalmologist, while the visual acuity of both eyes was 0.9, intraocular pressure was within 19 mm Hg. On admission (17:00): General condition of moderate severity. Blood pressure - 100/70 mm Hg (according to the patient, blood pressure was not higher than 100/70 mm Hg). Visual acuity of the right eye was equal to light perception with the correct projection of light, left eye - 0.4, not corr. Intraocular pressure of the right eye (Maklakov tonometer) - 44 mm Hg, left eye - 21 mm Hg. Right eye: pronounced congestive injection of the eyeball. Edema of the beef. Folds of Descemet's membrane. The anterior chamber is shallow. The pupil is oval, wide (5.0 mm in diameter), reacts sluggishly to light. The lens is thickened, the anterior capsule is tense, the anterior cortical layers are cloudy. Details of the fundus cannot be seen due to changes in the transparent media of the eye. Left eye: the eye is calm. The cornea is transparent. The anterior chamber is of medium depth, the humor is transparent. The pupil is round, 2.5 mm in diameter, the pupil reacts quickly to light. The anterior cortical layers of the lens are unevenly clouded, the nucleus is sclerotic. The reflex from the fundus is dim, details cannot be seen due to changes in the lens. The patient was hospitalized in the ophthalmology department of the clinic at 3 p.m. Prescribed: instillation of 0.5% thymmal solution into the right eye, 1% azopt solution twice a day, 0.25 mg diacarb twice a day orally, 200 ml 15% mannitol solution intravenously, lytic mixture intramuscularly (50% analgin - 2 ml, 1% diphenhydramine - 1 ml). At 5 p.m., that is, two hours after hospitalization, against the background of the treatment, the intraocular pressure of the right 16Science of Life and Health No. 3, 2020 eye decreased from 45 mm Hg to 36 mm Hg. The left eye is calm, intraocular pressure is 23 mm Hg. The general condition has improved, the headache has decreased, the treatment continues. Eleven hours after hospitalization, that is, at 3 a.m., the patient developed pain in the left eye, which spread to the same half of the head. However, the patient did not inform the medical staff on duty and "tolerated" the pain until the morning. At 8 a.m., during examination, the visual acuity of the right eye was equal to light perception with the correct projection of light, the left eye - 0.02, not corr. Intraocular pressure of the right eye - 36 mm Hg, left eye - 39 mm Hg. Against the background of treatment, the condition of the right eye remained without any significant changes. In the left eye, pronounced congestive injection and corneal edema appeared. The anterior chamber is shallow, the moisture is transparent. The pupil is wide, the diameter is 4.0 mm, the reaction of the pupil to light is sluggish. The cortical layers of the lens are unevenly clouded. It is impossible to examine the fundus in detail due to corneal edema and cataract. Since the patient was admitted at the end of the working day, an ultrasound examination of the eye was performed only the next day. Figure №1. Ultrasound examination of the right and left eyes On ultrasound: the anterior-posterior size of the right eye is 21.94 mm, the depth of the anterior chamber is 1.79 mm, the thickness of the lens is 5.34 mm. Left eye: anterior-posterior size is 21.93 mm, the depth of the anterior chamber is 1.95 mm, the thickness of the lens is 5.26 mm. Against the background of hypotensive, sedative, distraction therapy, the intraocular pressure of both eyes decreased insignificantly: right eye - from 45 to 36 mm Hg; left eye - from 39 to 34 mm Hg. According to the decision of the council, on the next day of hospitalization, phacoemulsification of cataract with implantation of an intraocular lens (IOL) of the right eye was performed. The operation was without

complications. The first day after the operation: visual acuity of the right eye was 0.01, the unoperated left eye - 0.02, correction also did not improve vision. Intraocular pressure of the right eye by palpation is normal, the left eye - 34 mm Hg. After the operation, on the first day, the complaint of pain disappeared and there was a slight increase in object vision of the right operated eye. Fundus: the optic disc is pale, the borders are clear; the arteries are sharply narrowed, the veins are full-blooded. The condition of the left eye remained at the same level. 17 Science of Life and Health No. 3, 2020 1963 Phaco-emulsification of cataract was planned for the second eye. Despite the conversation about the severity of the disease, the patient categorically refused surgical treatment of the left eye. Conclusion. The patient was diagnosed with bilateral swelling cataract, secondary phacomorphic glaucoma. The anterior-posterior size of the eyeball in both eyes was less than 22 mm. The patient had a history of mild hypermetropic refraction. Due to the increased refractive power of the lens, the patient had recently used. The lens clouding is the same in both eyes. In contrast to the literature, in our patient, phacomorphic glaucoma developed in both eyes with an interval of 14-15 days. Moreover, the increase in intraocular pressure in the second eye was observed against the background of general hypotensive treatment. The above-described case requires ophthalmologists, at an outpatient appointment, to pay close attention and conduct a detailed examination of the second eye for phacomorphic glaucoma in individuals with immature cataracts. Secondary glaucoma, which occurs as a result of concomitant ophthalmopathy, is considered the most severe type of glaucoma and in many cases leads to the loss of the eye as an organ [2, 4, 10]. It is known that diseases of the lens often lead to the development of secondary glaucoma (phacogenic) [9, 18]. In this regard, the identification of the characteristic clinical picture and symptoms of glaucoma is becoming increasingly important. against the background of lens pathologies, as well as the development of a modern approach to the treatment of this pathology.

The purpose and objectives of the study. To describe the clinical picture of phacogenic glaucoma, as well as to identify risk factors for the development of this ophthalmopathy. To propose an effective drug treatment regimen for secondary phacogenic glaucoma.

Research materials. Considering that the pathology of the organ of vision occurs in various age groups, belonging to both sexes, with different body weights, a group of 142 patients aged 40 to 65 years with a diagnosis of "secondary glaucoma" of unknown etiology was selected for the study. All patients had increased IOP (on average 38.63 ± 7.49 mm Hg). The studies were conducted from 2022 to 2024. at the Department of Ophthalmology of the Samarkand State Medical University and at an outpatient appointment at the polyclinic of the multidisciplinary clinic of the Samarkand State Medical University). Research methods. All patients with clinical signs of glaucoma were examined using the generally accepted method. Anamnesis vitae included information about the patient: age, gender, systemic diseases. Particular attention was paid to previous injuries to the organ of vision and operations performed in this area. Anamnesis morbi – information about the duration of the disease, clinical signs observed in the patient, the dynamics of the development of the pathology. When examining the area of the pathological process, the following ophthalmological examination methods were used: – external examination of the eye; – examination of the eye in transmitted directional light; – examination of the eye with lateral illumination; – direct and indirect ophthalmoscopy (Welch Allyn ophthalmoscope); – ophthalmobiomicroscopy at 6x magnification (Heine HRP 6x binocular loupes); – slit biomicroscopy at 10x magnification (Shin Nippon XL-1 slit lamp); – fundoscopy (Optibrand Clearview fundus camera); – gonioscopy (Goldmann and Barkan goniolenses); electroretinography (Acrivet RETIport ERG electroretinograph);– Maklakov applanation tonometry;– electronic tonometry (Tonovet tonometer);– ultrasound of the eyeball (Accu tomeB-scan Plus Vet 12–15 MHz ultrasound sensor);– fluorescein test (1% fluorescein solution). Ophthalmological examinations were performed in daylight, artificial light, and in the dark. Results and discussion. Studying the risk factors for secondary glaucoma, it was found that in 55 out of 142 patients (39% of cases), the development of secondary glaucoma was associated with lens pathologies (phacogenic glaucoma): luxation or subluxation of the lens, swelling cataract, lysis of hypermature cataract. Diagnostic criteria and clinical picture of glaucoma of phacogenic etiology were studied in 55 patients (68 eyes – 100%). Average IOP values during tonometry were 37.13 ± 4.45 mm Hg. The study revealed that in 42 out of 55 patients

(42 eyes – 76.4% of cases) secondary glaucoma against the background of lens pathologies was noted unilaterally. In 13 patients (26 eyes – 23.6% of cases) phacogenic secondary glaucoma was noted bilaterally. Characteristic clinical signs of secondary phacogenic glaucoma are: increased IOP (68 eyes – 100% of cases), decreased visual function (68 eyes – 100%), endothelial edema of the cornea (from slight to pronounced) (68 eyes – 100%), congestive injection of episcleral vessels (68 eyes – 100%), change in the depth of the PC of the eye (68 eyes – 100%), mydriasis (55 eyes – 80.9%), buphthalmos (55 eyes – 80.9%), clouding of the lens (52 eyes – 76.5%), dislocation of the lens (49 eyes – 72%). Of the non-specific signs of secondary phacogenic glaucoma, the following were observed: diagnostic clinical signs of secondary phacogenic glaucoma, as well as the number of affected eyes (in absolute values) and the number of affected eyes (in relative values), specific signs (characteristic of secondary glaucoma), increased IOP, decreased visual functions, endothelial edema of the cornea, congestive injection of episcleral vessels, changes in the depth of the PC of the eye, mydriasis, buphthalmos, lens opacity, lens dislocation, non-specific signs (characteristic of many ophthalmological diseases), pain on palpation of the eyeball, blepharospasm, mixed vascularization of the cornea, conjunctival hyperemia, ulcerative keratitis, luminescence of the intraocular fluid (Tyndall effect), iridodonesis, pain on palpation of the eyeball (55 eyes – 80.9% of cases), blepharospasm (20 eyes – 29.4%), mixed corneal vascularization (55 eyes – 80.9%), conjunctival hyperemia (20 eyes – 29.4%), ulcerative keratitis (20 eyes – 29.4%), Tyndall effect (33 eyes – 48.5%), iris tremor (iridodonesis) (49 eyes – 72%). The presence of these clinical signs is not pathognomonic for secondary phacogenic glaucoma, since they can be found in patients with uveitis, keratitis and conjunctivitis of various etiologies, as well as in case of eyeball trauma. In 49 eyes (72% of cases) we observed complete or partial displacement of the lens, as well as iridodonesis, which are characteristic signs of the development of the phacotopic form of secondary phacogenic glaucoma. In some cases, lens dislocation was accompanied by the presence of an intraocular inflammatory process - 14 eyes (20.6% of cases), as well as a decrease in lens transparency of varying intensity. In 19 eyes (28% of cases), the presence of exudate or luminescence of the intraocular fluid in the OC of the eye was detected, as well as lens opacity without signs of its displacement, which indicated the development of two other types of phacogenic secondary glaucoma - phacomorphic and phacolytic. Ultrasonographic picture of secondary phacogenic glaucoma was characterized by revealing signs of hyperechogenicity of the lens (52 eyes - 76.5% of cases) and its dislocation (49 eyes - 72% of cases). Pathogenetic aspects of secondary phacogenic glaucoma development. According to the literature, partial or complete displacement of the lens can provoke an increase in ophthalmotonus and lead to the development of secondary phacotopic glaucoma [3, 14]. Dislocation of the lens occurs as a result of rupture or weakening of the Zinn ligaments that hold it. A distinction is made between subluxation (subluxation) and luxation (dislocation) of the lens (Fig. 1). Subluxation is characterized by weakening or partial rupture of the Zinn ligaments. Luxation is characterized by a complete rupture of the ligaments and displacement of the lens either into the SC (partially or completely) or into the vitreous body. It is believed that the main cause of increased IOP in phacotopic glaucoma is a functional pupillary block due to the lens being pinched in the pupillary opening or displaced into the SC and the closure of the anterior chamber of the eye. Vitreous block may also develop – forward displacement of the vitreous body due to a violation of the position of the lens, which, in turn, leads to accumulation and stagnation of the vitreous fluid in the posterior segment of the eye and blockade of the pupillary opening by the vitreous body [3, 6, 14, 16]. Increased IOP in phacomorphic glaucoma develops as a result of an increase in the size of the lens itself and tight adherence of the posterior surface of the iris to the latter [3, 23]. Pupillary block develops – the vitreous fluid produced by the ciliary body does not enter the posterior chamber of the eye. This leads to increased IOP in the posterior chamber and protrusion. Partial or complete infringement of the anterior chamber of the eye by the root of the iris occurs, and the outflow of the vitreous fluid through the drainage system of the iridocorneal angle is impeded [4, 9, 12, 15]. Rupture of the lens capsule, as well as lysis of overripe cataracts, can lead to the development of lens-induced uveitis, blockage of the anterior chamber angle with inflammatory exudate and lens protein, and formation of posterior synechiae [22]. Such pathological changes in the hydrodynamic system of the eye can lead to an increase in IOP and the development of phacolytic glaucoma (Fig. 2).

This pathology is based on the closure of the iris-corneal angle by lens protein and macrophages containing lens substance, as well as inflammatory exudate. This occurs due to the development of overripe cataracts and leakage of proteins contained in the lens substance through the lens capsule into the intraocular fluid [7, 8, 19]. As a result, the trabecular gaps become clogged, which causes deterioration of the outflow of moisture through the drainage system of the eye and an increase in IOP [15] (Fig. 3). Also, an important factor aggravating the course of phacogenic glaucoma can be iridocyclitis, which we noted in some patients (48.5% of cases). This inflammatory process can be the result of microtrauma of the ciliary body, as well as irritation of the iris surface by an unstable lens. The release of fibrin, proteins and inflammatory cells into the IOG can provoke both blockage of the drainage system. Drug treatment of secondary phacogenic glaucoma. The treatment regimen for secondary phacogenic glaucoma included the use of both local and systemic therapy. At present, combination therapy of secondary glaucoma, based on a combination of several drugs from different pharmacological groups, is the most effective [2]. According to domestic and foreign sources, the main pathogenetic mechanism of visual impairment in glaucoma is associated with the impact of various damaging factors on the retina and optic nerve head: mechanical compression of optic nerve axons, impaired blood supply and microcirculation of the optic nerve head, development of ischemia and hypoxia of the nervous tissue, formation of excess free radicals, increased lipid peroxidation, release of glutamate and the flow of excess Ca2+ ions into the cell [1]. As a result, excitotoxic death of retinal ganglion cells occurs [5]. Therefore, therapy aimed at protecting the retina is a necessary link and is indicated in absolutely all treatment regimens for glaucoma in dogs. The main groups of drugs with neuroprotective action are antioxidants, nootropic agents, calcium channel blockers, NMDA receptor antagonists. Some antiglaucoma drugs (brimonidine, brinzolamide, betaxolol, etc.) have a direct neuroprotective effect, which was taken into account when prescribing hypotensive therapy [13, 17]. Thus, we proposed a hypotensive therapy regimen based on a combination of synthetic analogues of prostaglandins that improve the outflow of gastrointestinal tract fluid, as well as non-selective beta-blockers, alpha2-adrenomimetics, carbonic anhydrase inhibitors that inhibit the production of gastrointestinal tract fluid. The treatment was carried out in two stages. Drugs, frequency and duration Stage 1 of treatment Travoprost 0.004%, eye drops 1-2 drops 2 times a day, 5 days 25% mannitol solution 1-2 g/kg body weight, intravenously Brimonidine 0.2%, eye drops 1-2 drops 3-4 times a day, 5 days Diacarb, tablets 2.2 mg/kg body weight, per os every 12 hours Brinzolamide 1%, eye drops 1-2 drops 6-8 times a day, 5 days Timolol 0.5%, eye drops 1-2 drops 6-8 times a day, 5 days Stage 2 of treatment Travoprost 0.004%, eye drops (only for phacomorphic glaucoma) 1-2 drops 1 time per day, 60 days or more Mexidol vet», tablets 5-10 mg/kg body weight, per os, for 30 days Brimonidine 0.2%, eye drops 1-2 drops 2-3 times a day, 5 days Brinzolamide 1%, eye drops 1-2 drops 3-4 times a day, 60 days or more Timolol 0.5%, eye drops 1-2 drops 3-4 times a day, 60 days or more Indomethacin 0.1%, eye drops 1-2 drops 3-4 times a day, 30 days Emoxipin 1%, eye drops 1-2 drops, 3 times a day, 30 days As can be seen, drug therapy for secondary phacomorphic glaucoma at stage 1 included local use of hypotensive eye drops in combinations: travoprost 0.004% with a frequency of instillations 2 times a day, bri monidine 0.2% with a frequency of instillations of 3-4 times a day, brinzolamide 1% and timolol 0.5% with a frequency of instillations of 6-8 times a day, for a course of 5 days. Osmotic diuretics were used orally or parenterally: 25% mannitol solution: 1-2 g / kg of body weight intravenously, as well as carbonic anhydrase inhibitors: "Diacarb" (tablets) 2.2 mg / kg of body weight, per os every 12 hours. The patient was observed daily for 5 days, IOP was regularly measured to assess the hypotensive effect of the prescribed therapy. Upon achieving stable ophthalmotonus within the physiological norm, we proceeded to the 2nd stage of treatment, gradually reducing the frequency of eye drop instillations: 0.004% travoprost - once a day, 0.2% brimonidine - 2-3 times a day, 1% brinzolamide and 0.5% 3-4 times a day, for a course of 60 days or more. For neuroprotection and antioxidant therapy, we used "Emoxipin 1%" (eye drops) with a frequency of instillations of 3 times a day, "Mexidol-vet" (tablets) 5-10 mg / kg of body weight, per os, for a course of 30 days. To control the inflammatory process, non-steroidal anti-inflammatory drugs (0.1% indomethacin) were used in the form of eye drops with a frequency of 3-4 times a day (at least 30 days). Systemic administration of osmotic diuretics and carbonic anhydrase inhibitors was discontinued. It should be noted that in case

of lens subluxation in the posterior segment of the eye or lens incarceration in the pupillary orifice, synthetic prostaglandin analogues were not prescribed due to the development of persistent drug-induced miosis, which, in turn, can aggravate the course of glaucoma. At the same time, in case of lens subluxation in the posterior segment or luxation into the vitreous body, travoprost was the drug of choice due to the miosis it causes. This is explained by several reasons: 1) retention of the displaced lens in the posterior segment of the eyeball; 2) prevention of the risk of developing pupillary block due to lens displacement in the posterior segment; 3) prevention of vitreous hernia. We also took into account the presence of an inflammatory process (iridocyclitis), often accompanying secondary phacogenic glaucoma, which was detected in 48.5% of cases. Since synthetic prostaglandin analogues can aggravate intraocular inflammation and lead to dysfunction of the hematoophthalmic barrier, their use in such cases was limited or completely excluded [11]. In addition, we focused on the side effects of some drugs, in particular, on the effect of such drugs as timolol and brimonidine on the cardiovascular system. With systemic exposure, apathy, lethargy, bradycardia, vomiting and other symptoms can be observed [21]. The side effects of these drugs are most typical for small breeds of dogs weighing less than 10 kg. The effectiveness of the treatment was assessed after 7, 14, 30 and 60 days. At the repeated appointment, a general and comprehensive ophthalmological examination of the patient was performed. Thus, the level of ophthalmotonus after 7 days of treatment decreased by an average of 7.75 mm Hg, after 14 days - by 9.75 mm Hg, after 30 days - by 10.15 mm Hg, after 60 days - by 11.50 mm Hg. A decrease in IOP by an average of 31% from the initial level was noted. As can be seen, after 7 days of treatment of secondary phacogenic glaucoma, 24 patients (43.64% of cases) noted an improvement in the clinical picture and a decrease in IOP. When examining patients after 14 days, positive dynamics were noted in 31 patients (56.36% of cases). After 30 days of therapy, positive dynamics were noted in 39 patients (70.91% of cases). This result (39 patients) was maintained was obtained during a repeated examination of patients with secondary phacogenic glaucoma after 60 days of treatment. Based on the data obtained, we can conclude that the treatment regimen we proposed can be quite effective. In many ways, the success of this treatment is determined by the timing of contacting an ophthalmologist and strict adherence to the recommendation for the use of drugs. In some cases, we did not note significant positive dynamics in response to hypotensive therapy. We associated this result with the severity of pathological changes in the hydrodynamics of the eye, as well as some forms of secondary phacogenic glaucoma. Thus, glaucoma caused by swelling of the lens Dynamics of IOP reduction Local antihypertensive treatment regimen Travoprost 0.004%, Brimonidine 0.2%, Brinzol mid 1%, Timolol 0.5% Average IOP before treatment (mmHg) Average IOP, day 7 of treatment (mmHg) Average IOP, day 14 of treatment (mmHg) 37.13 ± 4.45 29.38 ± 4.33 (7.75–21%) 27.38 ± 4.07 (9.75–26%) Average IOP, day 30 of treatment (mmHg) 26.98 ± 3.80 (10.15–27%) Average IOP index, 60th day of treatment (mmHg) 25.63 ± 3.43 (11.50–31%) The eye condition after 30 days of therapy, IOP 19 mmHg (phacomorphic), in most cases was difficult to treat conservatively, while therapy for glaucoma caused by lens displacement (phacotopic) was more effective.

Conclusion. The phacolytic form of secondary phacogenic glaucoma should be noted separately. In cases where lens-induced uveitis was mild, hypotensive therapy in combination with anti-inflammatory drugs produced the desired result. In cases where the inflammatory process due to lysis of overripe cataract significantly impaired the functions of the hydrodynamic system of the eye, hypotensive therapy was ineffective. Conclusions. In conclusion, it should be said that lens pathologies (luxation, cataract) in patients are significant risk factors for secondary glaucoma. The clinical picture of secondary phacogenic glaucoma in patients is diverse and depends on many factors. We studied and identified the main symptoms characteristic of this disease: increased IOP, decreased visual function, endothelial edema of the cornea (from minor to pronounced), congestive injection of episcleral vessels, changes in the depth of the PC of the eye, mydriasis, buphthalmos, lens opacity, lens dislocation, iridodonesis, opacity of the intraocular pressure (Tyndall effect). Based on these clinical symptoms, we were able to determine that phacogenic glaucoma can be presented in several forms: - phacotopic, associated with luxation (subluxation) of the lens; - phacomorphic, associated with the development of swelling cataract; – phacolytic, associated with lysis of the lens substance and the development of lens-

induced uveitis. The scheme of intensive drug treatment of secondary phacogenic glaucoma proposed by us, based on combinations of several hypotensive drugs, turned out to be quite effective (70%). This allows us to consider this ophthalmopathy from the point of view of the possibilities of delayed surgery, when drug antiglaucoma and anti-inflammatory therapy is the basic preparation of the patient for surgical intervention - cataract phacoemulsification with simultaneous endoscopic cyclophotocoagulation.

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