Slovene
Glaucoma
Society

Sekcija
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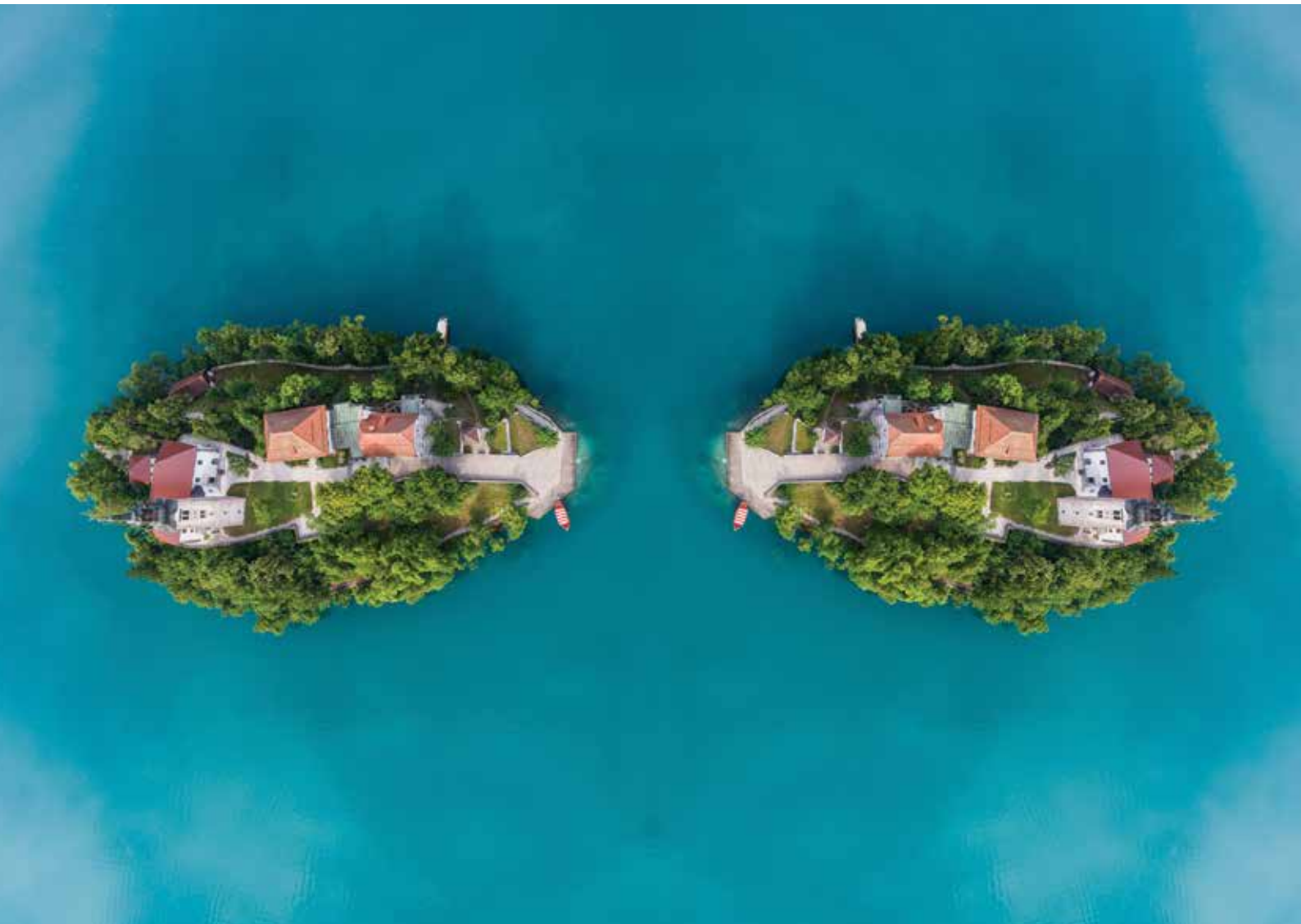
Slovenije

1. REGIONALNI GLAVKOMSKI SIMPOZIJ Z MEDNARODNO UDELEŽBO

1ST REGIONAL GLAUCOMA SYMPOSIUM WITH INTERNATIONAL PARTICIPATION

27 & 28 September 2024 Bled, Slovenija



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Rikli Balance Hotel, Bled, Slovenia

Slovene Glaucoma Society,
a group of the Slovenian Society of Ophthalmology

Scientific Secretariat

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Vesna Dimovska
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List of invited speakers

Gabor Hollo (Budapest)
Mia Zorić Geber (Zagreb)
Katia Novak-Lauš (Zagreb)
Vesna Dimovska (Skopje)
Nikola Babić (Novi Sad)
Sanja Sefić – Kasumović (Sarajevo)
Naser Salihu (Priština)
Qendrese Daka (Priština)
Jelena Radojičić (Podgorica)
Natalia Palarie (Chisinau, Moldova)

PRELIMINARY PROGRAMME

FRIDAY, 27 September 2024

08:30 – 09:30 REGISTRATION

INTRODUCTORY WORDS

09:30 – 10:45 GLAUCOMA EPIDEMIOLOGY, DIAGNOSTICS, TREATMENT
Moderators: Vesna Dimovska, Qendrese Daka, Tomaž Gračner

09:30 – 09:55 PREVALENCE OF GLAUCOMA IN THE CITY OF NOVI SAD, SERBIA AND CURRENT TRENDS IN THE REGION
Nikola Babić

09:55 – 10:20 GLAUCOMA ANGULARE AT CLINICAL CENTRE MONTENEGRO
Jelena Radojičić

10:20 - 10:45 GLAUCOMA DIAGNOSTICS
Barbara Cvenkel

10:45 – 11:15 COFFEE BREAK / EXHIBITION

11:15 – 12:05 GLAUCOMA - RISK FACTORS FOR PROGRESSION
Moderators: Katia Novak Lauš, Jelena Radojičić

11:15 – 11:40 MACULAR THICKNESS AS A BIOMARKER OF GLAUCOMA PROGRESSION
Vesna Dimovska

11:40 – 12:05 RISK FACTORS FOR OPEN ANGLE GLAUCOMA PROGRESSION: RESULTS OF A COCHRANE REVIEW
Mapa Plyasena, Qendrese Daka, Riaz Qureshi, Gloria Roberti, Manuele Michelessi, Tianjing Li, Yemisi Takwoingi, Gianni Virgili, Augusto Azuara-Bianco

12:05 – 12:50 *KEYNOTE LECTURE: Gabor Holló, Budapest, Hungary*
MEDICAL THERAPY OF GLAUCOMA IN 2024

13:00 – 14:30 LUNCH BREAK

14:30 – 16:00 DIAGNOSIS AND TREATMENT OF GLAUCOMA/LASER, SURGERY
Moderators: Mia Zorić Geber, Nikola Babić

14:30 – 14:55 CHALLENGES IN THE TREATMENT OF CHILDHOOD GLAUCOMA
Katia Novak Lauš

14:55 – 15:20 SELECTIVE LASER TRABECULOPLASTY
Makedonka Atanasovka Velkovska

15:20 – 15:45 NEOVASCULAR GLAUCOMA
Pia Klobučar

15:45 – 16:10	**COMBINATION OF ANTI-VEGF INJECTIONS AND MICROSECOND PULSE CYCLOPHOTOCOAGULATION IN THE MANAGEMENT OF NEOVASCULAR GLAUCOMA** ***Natalia Palarie, Natalia Palil***
16:10 – 16:17	**WHAT IS THE NEXT STEP IF TARGET INTRAOCULAR PRESSURE IS NOT ACHIEVED WITH MONOTHERAPY** ***Barbara Cvenkel (Invited by Inspharma)***
16:20	**COFFEE BREAK / EXHIBITION**
18:00	**DINNER BLED CASTLE**

SATURDAY, 28 September 2024

09:00 – 10:20	**GLAUCOMA SURGERY** ***Moderators: Sanja Sefić Kasumović, Naser Salihu, Barbara Cvenkel***
09:00 – 09:25	**UNDERSTANDING THE MANAGEMENT OF GLAUCOMA SURGERIES FORMING NEW DRAINAGE PATHWAYS** ***Mia Zorić Geber***
09:25 – 09:55	**THE EFFECT OF PHACOEMULSIFICATION ON THE INTRAOCULAR PRESSURE IN PATIENTS WITH PRIMARY OPEN-ANGLE GLAUCOMA** ***Tomaž Gračner***
09:55 – 10:20	**CATARACT SURGERY ON PATIENTS WITH END-STAGE GLAUCOMA** ***Sanja Sefić Kasumović***
10:20 – 10:50	**COFFEE BREAK / EXHIBITION**
10:50 – 12:05	**GLAUCOMA SURGERY** ***Moderators: Mia Zorić Geber, Naser Salihu, Vladimir Pfeiffer***
10:50 – 11:15	**AQUEOUS DRAINAGE DEVICE IMPLANTATION USING SCLERAL TUNNEL TECHNIQUE** ***Vladimir Pfeifer***
11:15 – 11:40	**OVERVIEW OF POST-OP AQUEOUS MISDIRECTION** ***Naser Salihu, Yilke Salihu***
11:40 – 12:05	**THE ROLE OF GLAUCOMA DRAINAGE DEVICES (GDD) IN TREATMENT OF SECONDARY GLAUCOMA (SECG) AFTER AN EARLY CONGENITAL CATARACT (CC) OPERATION** ***Manca Tekavčič Pompe, Špela Markelj, Barbara Ana Marinčič, Vladimir Pfeifer***
12:05 – 12:10	**CLOSING WORDS**
12:10	**LUNCH BREAK**

Prevalence of glaucoma in the city of Novi Sad, Serbia and current trends in the region

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Purpose: Our study aimed to estimate the prevalence of glaucoma and its subtypes in the population of Novi Sad, Vojvodina, Serbia, and provide the demographic and clinical analysis of glaucoma patients involved.

Methods: Our study was designed as an observational, retrospective, cross-sectional, monocentric, including all the patients with the address of residence within the city of Novi Sad, with clinically diagnosed glaucoma, at least in one eye, treated at the University Eye Clinic, Clinical Centre of Vojvodina, Novi Sad. We analyzed the five-year prevalence of different types of glaucoma, together with the characteristics of visual field and risk factors in the form of coexisting diabetes mellitus and arterial hypertension.

Results: Almost half of 3254 included patients (48.28%) were diagnosed with primary open-angle glaucoma (POAG), and its prevalence in the total population of Novi Sad was estimated to be 0.46%. The prevalence of other glaucoma types was as follows: primary angle-closure glaucoma (PACG) 0.17%, secondary glaucoma 0.09%, pseudoexfoliation glaucoma 0.09%, normal-tension glaucoma 0.13%, pigmentary glaucoma 0.01%, and juvenile glaucoma 0.01%. In the population above 40 years of age, the prevalence of all glaucoma cases was 1.9%, while the prevalence of POAG was 0.93%, and the prevalence of PACG was 0.35%.

Conclusion: Our study represents the first attempt to address the epidemiological problems of glaucoma in our region in a comprehensive, evidence-based way. The prevalence of various glaucoma types and observed age-specific prevalence trends were lower than those published by other authors involving comparable populations, and we offered several potential explanations for this in our paper.

Glaucoma angulare at Clinic Cente of Montenegro

Jelena Radojičić
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Purpose: Frequency of glaucoma angulare in 2023 in the Clinic Centre of Montenegro compared to other forms of glaucoma druring the some year.

Methods: 40 patients with glaucoma angulare were examined. Difference in frequency in age by gender, in relation to pressure heigh, the size of the excavation, change in field of vision.
Analysis in relation to frequency to the regions of Montenegro, frequency in relation to a positive family history, impact on visual acuity, method of treatment, with or without iridotomy.

Conclusion: Frequency of angulare glaucoma in Montenegro through all offices of Clinical Center is significantly lower than other forms of glaucoma.

Glaucoma diagnostics

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Early diagnosis and detection of the progression of the disease are crucial for the successful treatment of glaucoma. A clinical examination including gonioscopy, fundoscopy with assessment of the optic disc and retinal nerve fibre layer (RNFL) and visual field testing are strongly recommended at the initial and follow-up examination. Standard automated perimetry (SAP) is the reference standard for the assessment of visual function in glaucoma. It is dependent on patient co-operation and in some patients, there is a high variability in mean deviation over time, which reduces the ability to distinguish true change from noise. Electroretinography (ERG) is an objective method of assessing visual function, and both the pattern ERG and the photopic negative response of the ERG are sensitive markers of retinal ganglion cell dysfunction that may precede structural changes and visual field defects in SAP. Optical coherence tomography (OCT) of the optic disc, RNFL and macula allows objective quantification and contributes to early glaucoma diagnosis and monitoring. Non-invasive assessment of ocular microvasculature by OCT angiography (OCTA) complements OCT and can detect the reduction of superficial vessel density in the early stages of glaucoma. Deep learning (DL) can support the differentiation between healthy and pathological optic disc based on fundus photographs, while improving the quality and quantity of OCT data could be used for the diagnosis of glaucoma in the future.

Macular thickness as a biomarker of glaucoma progression

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Macular thickness measurements are considered to represent a surrogate indicator of retinal ganglion cell thickness and have proven to have clinical value for glaucoma diagnosing and detection of change. Therefore, establishing of baseline structural measurements, such as OCT-imaging and measurement of RNFL, ONH and macula should be interpreted complementary in order to increase sensitivity for detection of change over time/progression. The lack of a gold standard is causing difficulties in assessment and determination of best parameter to be used in clinical practice. Macular OCT- imaging is a crucial structural imaging modality for assessing the central retinal ganglion cells health (RGCs). Novel OCT technologies allow objective quantification in vivo of the key glaucomatous structural changes in retina and the optic nerve head. An ideal parameter for progression measurement should provide high reproducibility and utility at all disease stages. Most widely used parameters are circumpapillary RNFL (cRNFL) and macular measurements of GCIPL and GCC thickness. The GCC thickness measurement is most probably the optimal macular outcome measure for detection of glaucoma deterioration. New and more sophisticated OCT-devices have ability to obtain Bruch's membrane opening (BMO) as an anatomical point of reference for macular measurements in order to enhance measurement accuracy and relevance.

Conclusion: Macular thickness measurement has shown to be excellent adjuvant modality in glaucoma diagnosis, interpreted complementary to OCT, visual field and optic disc head examination. Macular parameters have the ability to detect or confirm change seen with other modality, or/and overcome some shortcomings of optic nerve assessment and RNFL measurements. Nowadays still remain some important questions that need to be raised, particularly regarding testing strategies to ensure the most effective use of OCT-imaging in clinical practice for glaucoma detection and monitoring progression.

Key words: OCT-imaging, glaucoma, macular thickness,progression

• *No conflict of interest do declare*

Risk factors for open angle glaucoma progression: results of a Cochrane review

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Purpose: Understanding prognostic factors for primary open-angle glaucoma (POAG)—the most common type of glaucoma—and pseudoexfoliative glaucoma (PXFG)—a common secondary form with high progression rates—is crucial for clinicians to estimate disease progression risk and identify those at early risk of sight loss. This review aimed to identify prognostic factors for the progression of functional visual outcomes and structural outcomes in adults with POAG and PXFG.

Methods: CENTRAL, Ovid MEDLINE, Ovid EMBASE, ClinicalTrials.Gov, and ICTRP were searched until September 2023. Randomized controlled trials, cohort studies, and case-control studies involving adults (≥18 years) of any gender with POAG or PXFG were included, excluding those with prior surgical glaucoma treatment. Studies with less than 2 years of follow-up or fewer than 200 participants were excluded. Data extraction was conducted using Covidence and SRDR+ platforms, with summary estimates transferred to MS Excel. The Quality in Prognosis Studies (QUIPS) tool assessed bias risk, with independent steps by two reviewers, and discrepancies resolved by a third. Meta-analyses were conducted for homogeneous outcomes using a random-effects model, and evidence certainty was evaluated with GRADE guidelines.

Results: Twenty-two studies out of 14,294 screened titles and abstracts were included. Sixteen used visual field (VF) deterioration alone to measure glaucoma progression, while five used both functional and structural outcomes. For intraocular pressure (IOP), the hazard ratio (HR) was significant, but the odds ratio (OR) was not. Disc haemorrhage was a significant prognostic factor in adjusted and unadjusted analyses. Combined analyses of two studies suggested females might have a 64% higher hazard of progression than males.

Conclusions: Evidence supports female sex, high baseline IOP, the presence of disc hemorrhages, and having glaucoma treatment as prognostic factors for VF progression in POAG. Evidence for other factors is less compelling or inconclusive. Further research on prognostic factors is needed.

Medical therapy of glaucoma in 2024

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Purpose: To review current classic, recently introduced and in the pipeline medical therapy options for glaucoma.

Methods: A narrative, wide literary review which includes current European guidelines, original clinical and research publications and relevant recent reviews.

Results: Current "classic" and recently introduced topical intraocular pressure (IOP) lowering medications are both monotherapies and fixed dose combinations, and need to be used according to the current European Guidelines. New active IOP lowering molecules recently introduced in clinical practice comprise omidenepag isopropyl (USA), Rho kinase inhibitor ripasudil (Japan), and netarsudil and its fixed dose combination with latanoprost (USA, Europe), and latanoprostane bunod (USA). Their mechanisms of action, IOP lowering efficacy and the related open questions will be discussed in detail. Further, new drug delivery methods, including drug eluting ocular rings, punctal plugs and intracameral implants will be discussed.

Conclusion: Both current classic and recently introduced IOP lowering topical medications offer powerful IOP reduction in glaucoma, but clinicians need to understand their mechanisms of action, side effect profile, and make educated selection on this basis. Despite of the recent dug delivery innovations a careful and comprehensive ophthalmological evaluation remains a most important part of successful long-term glaucoma care.

Challenges in the treatment of childhood glaucoma

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Prompt diagnosis of childhood glaucoma and appropriate treatment can minimize the degree of visual impairment. Examination should be done under anesthesia or sedation to make the correct diagnosis, perform surgery or plan further treatment. Intraocular pressure (IOP) measurement and optic disc appearance are key elements of examining children with glaucoma. Enlarging corneal diameter, increasing axial length and progressive myopia are signs of persistent elevated IOP that must be considered and regularly assessed. Some of the clinical signs of primary congenital glaucoma (PCG) are also found in other conditions and must be considered in the differential diagnosis. The most common signs are conditions with overlapping signs of corneal edema and opacity or conditions with overlapping signs of corneal enlargement. Treatment of PCG is exclusively surgical. Glaucoma surgery in children is more challenging than in adults for many reasons but mostly due to ocular enlargement and aggressive healing. The wide limbus with distorted limbal anatomy of buphthalmic eyes makes surgery different than in adults. Thin, elastic sclera with low rigidity increases the tendency of the anterior chamber to collapse and the posterior chamber to move forward which leads to hypotony. The thick Tenon capsule impedes filtration and contains a large reservoir of fibrocytes and fibroblasts involved in the inflammatory response and scarring. In secondary childhood glaucoma, it is essential to understand the mechanism of glaucoma development based on which the appropriate surgical technique will be chosen. Special attention should be given to children with uveitic glaucoma. The control of intraocular inflammation with adequate immunosuppression, and topical and systemic agents, is a crucial part of management. Lastly, lack of cooperation affects the degree of monitoring and post-op follow-up which can compromise surgical results. Multiple examinations under anesthesia may be required for IOP measurement and examination. Also, regional and socioeconomic differences play an important role in the postoperative period and parental compliance plays an important role in postoperative care. Concurrent with controlling IOP, ametropia correction and amblyopia management are essential to optimize long-term visual outcomes.

Selective laser trabeculoplasty

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Selective laser trabeculoplasty (SLT) is a laser-based medical procedure used to treat open-angle glaucoma, reducing intraocular pressure (IOP) by increasing outflow through the trabecular meshwork. SLT selectively targets pigmented trabecular meshwork cells using short, low-energy laser pulses, inducing a biological response that enhances outflow facility without causing significant thermal damage to surrounding tissues. This non-invasive, outpatient procedure has emerged as an effective alternative to traditional pharmacologic and surgical treatments for glaucoma. Clinical studies have demonstrated that SLT effectively reduces IOP (a mean IOP reduciton of 20-25%) in patients with primary open-angle glaucoma and ocular hypertension, with success rates comparable to those of argon laser trabeculoplasty (ALT) but with fewer complications and better repeatability. The mechanism of action involves the recruitment of macrophages and the remodeling of extracellular matrix components, leading to increased aqueous humor drainage. SLT's safety profile is favorable, with transient postoperative inflammation and IOP spikes being the most commonly reported adverse effects, which are typically self-limiting and manageable with topical medications. Given its efficacy, safety, and repeatability, SLT is recommended as a first-line or adjunctive treatment for open-angle glaucoma according to the latest EGS guidelines. Ongoing research aims to optimize treatment parameters, identify predictors of success, and elucidate the long-term outcomes of SLT. As the understanding of its underlying mechanisms advances, SLT holds promise for broader application and improved management of glaucoma, frequently delaying the need for more invasive surgical interventions.

Keywords: Selective laser trabeculoplasty, glaucom, therapy

Neovascular glaucoma

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Neovascular glaucoma (NVG) is a secondary angle-closure glaucoma that arises due to retinal ischemia, which can be caused by various conditions. The most common causes of NVG are central retinal vein occlusion and proliferative diabetic retinopathy. Initial ischemia in the anterior segment manifests as neovascularization (NV) at the pupillary margin and/or angle, making thorough clinical eye examination and gonioscopy crucial for patients with risk factors for its development. Treatment depends on the extent of involvement and neovascularization. The standard treatment is panretinal laser photocoagulation (PRP), which reduces ischemia and prevents the progression of NV and consequently the development of NVG. When intraocular pressure is already elevated due to angle closure with goniosynechiae, additional therapy—both medicinal and/or surgical—is required. Prognosis is generally poorer compared to other forms of secondary glaucoma. It depends on the extent of involvement at the initial examination, the level of intraocular pressure elevation, the degree of NV due to ischemia, the status of the angle, the presence of existing glaucomatous optic neuropathy and the patient adherence to treatment. A retrospective review found that 25% of eyes presenting with NVI/NVA and IOP < 21 mmHg progressed to NVG, with the majority progressing by 6 months. Anti-VEGF treatment is effective and induces temporary regression of NV, reduces vascular wall permeability and inflammation, and, in the case of open-angle NVG, provides time for PRP, but it does not replace careful monitoring and definitive NVG therapy. This presentation aims to highlight the importance of early detection of anterior segment ischemia to prevent the development of NVG with permanent vision loss and to present guidelines for managing patients with NVG.

Combination of anti-VEGF injections and microsecond pulse cyclophotocoagulation in the management of neovascular glaucoma

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Purpose: Neovascular glaucoma (NVG) is a particularly challenging and prognostically poor form of glaucoma. This research evaluated the effectiveness of a dual treatment approach combining intraocular VEGF inhibitor injection and microsecond pulse cyclophotocoagulation (µCPC) for managing secondary neovascular glaucoma.

Methods: The study included 58 patients (67 eyes) suffering from secondary neovascular glaucoma due to diabetes or thrombosis of the central retinal vein or its branches. The best corrected visual acuity (BCVA) ranged from hand motion to 0.4, with an average initial intraocular pressure (IOP) of 42 ± 12 mm Hg. Treatment consisted of an intraocular injection of Bevacizumab, a VEGF inhibitor, followed within 5-7 days by 810 nm infrared diode laser application in microsecond pulse mode at 2000 mW for a total duration of 220-240 seconds (145 – 160 J) and a duty cycle of 33.3%. Treatment success was determined by a decrease in anti-glaucoma drop (AGD) usage and maintaining an IOP between 11-21 mm Hg at the final follow-up. Follow-up assessments occurred at baseline, 1 week, and 1, 3, and 6 months post-treatment.

Results: On average, 1.3 treatments were administered per eye, with 20 eyes (30%) needing additional treatment with continuous-wave CPC within the first month. The mean IOP dropped to 28.5 ± 5.0 mm Hg after 1 week, 23.0 ± 5.3 mm Hg after 1 month, 19.5 ± 3.2 mm Hg after 3 months, and 18.5 ± 2.5 mm Hg after 6 months, showing a stable reduction in IOP starting at 3 months. The treatment was successful in 74% of cases. The use of AGD decreased from 2.0 ± 1.0 at baseline to 1.1 ± 1.2 at 1 month, then increased to 1.7 ± 1.0 at 3 months and 2.2 ± 1.2 by 6 months. No severe complications or hypotony were reported.

Conclusion: The combination of VEGF inhibitor injections and µCPC offers an effective, safe, and prompt treatment for NVG over a six-month period.

What is the next step if target intraocular pressure is not achieved with monotherapy?

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Background: The EGS guidelines recommend using the least amount of medication to achieve the desired reduction in intraocular pressure (IOP) and delay the progression of glaucoma. It is recommended to start with preservative-free prostaglandin analogues (PGA) as the treatment of first choice, as they are the most effective and best tolerated class of medication and only need to be administered once daily. However, despite their efficacy and good tolerability, PGA monotherapy does not achieve the target IOP in up to 80% of patients. In such cases, an additional drug from a different class should be considered. If available, a preservative-free fixed combination (FC) is preferable to two separate instillations of agents as it may decrease the patient's burden, especially if this FC is administered only once daily. Most commonly, PGA is used with a beta-blocker, which may improve local tolerability, but caution is advised in patients with relevant contraindications to beta-blockers (e.g. asthma, bradycardia). The preservative-free FC of latanoprost/timolol (Fixalpost, Laboratoires Théa, France) was as effective as the preserved FC latanoprost/timolol (Xalacom, Upjohn EESV, Netherlands), but was better tolerated after 3 months than the preserved comparator solution.

Conclusion: If target pressure is not achieved with preservative-free monotherapy, which is effective and well tolerated, the next step is the addition of an active substance, preferably in a preservative-free FC with PGA, which does not affect the patient's well-being and adherence with long-term treatment.

Understanding the management of glaucoma surgeries forming new drainage pathways

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Trabeculectomy and glaucoma drainage device (GDD) surgery are commonly performed on glaucoma patients who do not respond to medication. Compared to techniques that restore natural drainage pathways, whose effectiveness is limited by their capacity, these procedures create new drainage routes and are more effective. GDD implantation is performed in complicated cases of glaucoma, where conventional filtering surgery has failed or is likely to fail, such as in various secondary glaucoma. After GDD implantation, post-operative intraocular pressure (IOP) typically goes through three phases: an initial hypotensive phase that may occur within the first week after surgery, a hypertensive phase (HP) characterized by a significant rise in IOP, usually within 3 months post-surgery, followed by a steady phase where IOP gradually decreases and stabilizes. A unique clinical challenge in the postoperative care of GDD is the HP, a period of elevated IOP. Since most patients receiving GDDs have advanced glaucoma, this phase can further damage the optic nerve, necessitating additional treatments and potentially leading to implant failure. The incidence of HP after GDD implantation varies among studies and types of devices used, with a higher incidence reported in valved compared to non-valved GDDs. As GDD implantations become more prevalent, the HP is of increasing clinical relevance. This phase occurs secondary to bleb encapsulation, which is an integral part of the wound-healing process following device implantation. Understanding the basic science of wound healing is crucial for glaucoma clinicians. Numerous investigations have provided insights into preoperative, intraoperative, and postoperative management strategies that can minimize the occurrence of the HP. However, there is no clinical consensus or best-practice guideline for the prevention or management of this challenge.

The effect of phacoemulsification on the intraocular pressure in patients with primary open-angle glaucoma

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Purpose: To evaluate the effect of phacoemulsification and intraocular lens implantation (PHACO IOL) on intraocular pressure (IOP) in patients with primary open-angle glaucoma (POAG).

Methods: 65 eyes of 46 patients with POAG in which PHACO IOL was done were included in this retrospective analysis. The indication for PHACO IOL included a best corrected visual acuity (BCVA) of 0.7 (decimal equivalents of Snellen visual acuity) or worse with visual disturbance caused by the cataract. The IOP was measured and the number of antiglaucoma medications evaluated before and one week, 1, 3, 6, 12, 18, and 24 months after the PHACO IOL in all eyes. The BCVA before PHACO IOL and at the end of follow-up were evaluated.

Results: The mean IOP before surgery was 15.7 mmHg (SD 1.8). The mean IOP one week after PHACO IOL was 15.2 mmHg (SD 2.4), 1 month after PHACO IOL 15.3 mmHg (SD 2.3), 3 months after PHACO IOL 15.0 mmHg (SD 2.0), 6 months after PHACO IOL 15.3 mmHg (SD 2.2), 12 months after PHACO IOL 15.4 mmHg (SD 1.9), 18 months after PHACO IOL 15.6 mmHg (SD 1.9) and 24 monts after PHACO IOL 15.6 mmHg (SD 2.0). The mean IOP was significantly lower at 3 months ($p=0.003$) and 12 months ($p=0.049$) after PHACO IOL. The mean number of antiglaucoma medications before PHACO IOL was 1.89 (SD 0.9) and remained unchanged postoperatively at all follow-up visits in all eyes. The mean preoperative BCVA was 0.33 (SD 0.2), improving to a mean of 0.91 (SD 0.1) postoperatively at the end of follow-up ($p<0.0001$).

Conclusion: 24 months after the PHACO IOL in patients with POAG resulted in a almost unchanged IOP together with the unchanged number of antiglaucoma medications and a significantly improved BCVA.

Cataract surgery on patients with end-stage glaucoma

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Glaucoma and cataract are the top two leading causes of blindness worldwide significantly affect the visual impairment and quality of life. Cataract surgery is usually an operation without unexpected outcomes, except in advanced or end-stage glaucoma patients. Advanced glaucoma patients are more likely to have unexpected refractive surprise and even worse irreversible visual outcome after incisional operation (like phacoemulsification). It has been shown that a surgeon needs to make a number of adjustments to ensure a good outcome, especially in this group of patients. This presentation aims to assess all the special considerations that should be taken when approaching cataract surgery in patients with advanced glaucoma. Main questions that remain controversial are: how much is glaucoma contributing to the visual acuity reduction, which part is up to cataract, can the operation be postponed in those patients, and the risk of the “whipe-out” phenomenon. Critical clinical judgement, as well as realistic expectations are crucial in achieving a successful outcome in these patients.

Aqueous drainage device implantation using scleral tunnel technique

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Purpose: To describe and present surgical technique of glaucoma valve implantation, using scleral tunnel 4mm in length.

Methods: In children and adults special surgical technique utilizing 4 mm long scleral tunnel was used. The roof of scleral tunnel was cut in the middle horizontally to allow tube manipulation and insertion into the anterior chamber parallel to the iris. The valve itself was sutured in place 10 mm behind the limbus and put under the rectus muscles, dependent on type of valve. Different valve types were used utilizing the same technique.

Results: In up to 15 years follow up of different glaucoma valve design implantations utilizing the described surgical technique, also among children, no tube extrusion was observed. In all cases there was no tube prolapse or scleral melting.

Conclusion: The described surgical technique is safe, ensures 100% long term tube covering and prevents tube exposure, also in children up to 15 years after valve implantation. Also, in cases of advanced glaucoma, enlarged eyeball, and thin sclera where thickness of the tunnel covering sclera is less than 150 microns no protrusion was observed.

Overview of post-op aqueous misdirection

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Aqueous misdirection (AM) is a challenging form of secondary angle closure that presents with elevated intraocular pressure (IOP) and shallowing of the central and peripheral anterior chamber. It usually occurs during the early post-operative period after incisional surgery with an incidence of 0.6 to 4%. The pathogenesis of AM remains in¬completely understood and consensus on its treatment and nomenclature has not yet been achieved. Surgical risk factors are glaucoma surgery, most common after trabeculectomy or combined trabeculectomy and cataract surgery, but it has also been reported after bleb needling/ revision, cataract surgery, penetrating keratoplasty and laser iridotomy. Anatomic risk factors include short axial length, plateau iris configuration, anatomically narrow angle and primary angle-closure glaucoma. Other proposed risk factors include Pseudoexfoliation and female sex. Symptoms can include redness, eye pain, and decreasing vision. B-scan ultrasonography can help exclude choroidal detachments and suprachoroidal haemorrhage, which are two important differential diagnoses. Ultrasound biomicroscopy (UBM) can also be used to image the anterior chamber angle, iris configuration and lens configuration. Anterior segment optical coherence tomography (OCT) can be used to measure anterior chamber depth and configuration in cases where the cornea is sufficiently clear. Although good evidence on manage¬ment of AM is lacking, the mainstay of treatment involves IOP reduction through medical or surgical means. Medical management with cycloplegics-mydriatics, aqueous suppressants and hyperosmotics is effective in roughly half of cases of aqueous misdirection within five days, after which surgical intervention should be strongly considered. In many cases conservative treatment alone is insufficient and surgical and/or laser interventions are required. The goal of laser therapy is to establish a conduit for flow between the vitreous cavity and the anterior chamber. If the patient is refractory to both medical and laser management of aqueous misdirection, surgery is necessary to disrupt the anterior vitreous face or remove vitreous, thereby increasing aqueous flow to the anterior chamber. In conclusion, AM can represent a very serious clinical challenge. Though the aetiology is unclear it demonstrates how an imbalance in the dynamic between the lens, cilary body and vitreous can cause a potentially vision threatening condition. However, with prompt diagnosis and early initiation of treatment, aqueous misdirection can be resolved. Furthermore, steps can be taken to prevent occurrence in the fellow eye.

Key words: Aqueous misdirection, glaucoma.

The role of glaucoma drainage devices (GDD) in treatment of secondary glaucoma (secG) after an early congenital cataract (CC) operation

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Introduction: The aim of this study was to investigate the role of GDD in the treatment of secG after early CC surgery in our paediatric cohort.

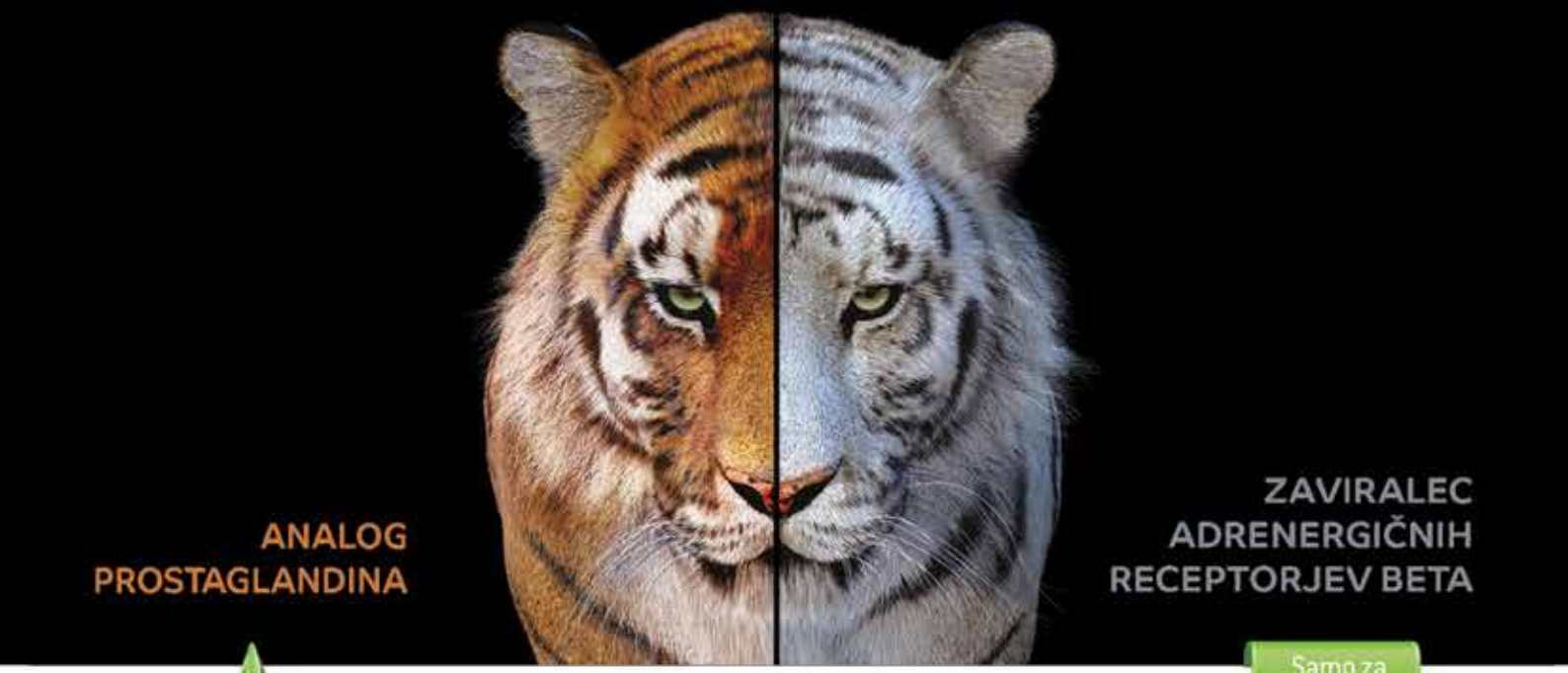
Methods: 23 eyes of 17 children who underwent surgery for CC within the first year of life were included in the study. 7 eyes received a primary IOL, 16 were left aphakic, the mean follow-up time was 5 years (range: 6-144 months). Implanted GDD: Bearveldt 250 in 19 eyes, Bearveldt 350 in 1 eye, Ahmed FP8 in 3 eyes. The surgical technique was comparable in all cases.

Results: The following secondary interventions were required: Correction of hypotony within the first postoperative week in 3 eyes (2-4 days postoperatively, 1 eye was lost), adding suture on the GDD due to hypotony in 2 eyes (3 and 4 months postoperatively), shortening of the tube due to corneal touch in 3 eyes (6-11 months postoperatively), implantation of an additional GDD in 2 eyes (17 and 30 months postoperatively) and removal of fibrotic tissue from the GDD in 4 eyes (3, 4, 8 and 10 years postoperatively).

Conclusions: The GDD is a useful option for the management of secG after CC surgery in children. The longer the follow-up time, the more eyes need GDD revision or further GDD implantation to control IOP. Reoperations due to tube-related complications are also relatively common.

ZNIŽANJE POVIŠANEGA OČESNEGA TLAKA PRI BOLNIKIH Z GLAVKOMOM Z ODPRTIM ZAKOTJEM IN OČESNO HIPERTENZIJO,
KI SE NISO ZADOSTNO ODZVALI NA LOKALNE ZAVIRALCE ADRENERGIČNIH RECEPTORJEV BETA ALI ANALOGE PROSTAGLANDINA

MOČNEJŠA SKUPAJ



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Indikacije: Znižanje povišanega očesnega tlaka pri odraslih bolnikih z glavkomom z odprtim zakotjem in očesno hipertenzijo pri bolnikih, ki se niso zadostno odzvali na lokalne zaviralce adrenergičnih recectorjev beta ali analoge prostaglandina. **Odmerjanje in način uporabe:** 1 kapljica v obolelo oko (obolela očesa) enkrat na dan. Zaradi zmanjšanja možnosti sistemske absorpcije je priporočljivo, da za dve minuti pritisnemo solzni mešiček ob notranji očesni kot takoj po vkapljanju kapljice. Leče je pred uporabo treba odstraniti. Vstavijo se lahko spet po 15 minutah. V primeru uporabe več lokalnih zdravil za oči mora med uporabo posameznih zdravil miniti vsaj 5 minut.
Kontraindikacije: Reaktivna bolezen dihal, sinusna bradikardija, sindrom bolnega sinusnega vozla, sinoatrijski srčni blok, atrioventrikularni blok druge ali tretje stopnje, ki ni nadzorovan s srčnim vzpodbujevalnikom, simptomatsko srčno popuščanje, kardiogeni šok; preobčutljivost na učinkovini ali pomožne snovi. **Opozorila, previdnostni ukrepi, interakcije:** Zaradi timolola lahko pride do pojava istih srčnožilnih, pljučnih ali drugih neželenih učinkov, kot jih opazimo pri sistemski uporabi zaviralcev beta. Pojavnost sistemskih neželenih učinkov po lokalni uporabi v očesu je nižja kot pri sistemski uporabi. Pri bolnikih s srčnožilnimi boleznimi, hipotenzijo in z blago do zmerno kronično obstruktivno pljučno boleznijo je potrebno skrbno pretehtati ali je zdravljenje z zaviralci beta primerno in jih nato skrbno spremljati. Pri bolnikih s hudimi perifernimi motnjami prekrvavitve je potrebna previdnost. Zaviralci beta lahko prekrijejo znake akutne hipoglikemije in hipertiroidizma. Zaviralci beta lahko okrepijo hipoglikemični učinek antidiabetičnih zdravil. Zaviralci beta za lokalno uporabo v očesu lahko povzročijo suhe oči, bolnike z boleznimi roženice je treba zdraviti previdno. Učinek na znotrajočesni tlak ali znani sistemski učinki zaradi zavore receptorjev beta se lahko okrepijo, če bolnik poleg timolola prejema tudi sistemski zaviralec beta. Bolniki z atopijo ali hudo anafilaktično reakcijo na različne alergene v anamnezi so lahko bolj dovzetni na ponavljajoče se izpostavljanje tem alergenom in se ne odzovejo na običajne odmerke adrenalina. Po uporabi raztopin za zniževanje tlaka po izvedeni trabekulektomiji so poročali o odstopu horoideje. Zaviralci beta za lokalno uporabo v očesu lahko zavrejo sistemske učinke agonistov receptorjev beta, npr. adrenalina; anesteziologa je potrebno opozoriti, da bolnik prejema timolol. Kadar se lokalni zaviralci beta za oči uporabljajo sočasno s peroralnimi zaviralci kalcijevih kanalčkov, zaviralci adrenergičnih receptorjev beta, antiaritmiki, glikozidi digitalis, parasimpatomimetiki ali gvaneticinom obstaja možnost, da se učinki seštevajo, kar vodi v hipotenzijo in/ali bradikardijo. Pri sočasni uporabi timolola z zaviralci CYP2D6 so poročali o povečanih učinkih zaradi sistemske zavore receptorjev beta (tj. upočasnjen srčni utrip, depresija). Sočasna uporaba večih prostaglandinov, analogov ali derivatov prostaglandinov ni priporočljiva. Latanoprost lahko postopoma spremeni barvo oči, ker poveča količino rjavega pigmenta v šarenici ter lahko postopoma spremeni trepalnice in dlačice na očesu in v njegovi okolici. Latanoprost je treba uporabljati previdno pri bolnikih z zgodovino herpetičnega keratitisa in ga ne uporabljati v primerih akutnega keratitisa, povzročenega z virusom herpesa simpleksa. Med uporabo latanoprosta so opisani primeri makularnega edema vključno s cistoidnim makularnim edemom, večinoma pri afakičnih bolnikih, pri pseudofakičnih bolnikih z rupturo zadnje lečne ovojnice ter pri bolnikih z znanimi dejavniki tveganja za cistoidni makularni edem. **Neželeni učinki.** *Povzetek varnostnega profila:* Pri latanoprostu je večina neželenih učinkov vezana na očesni sistem. V kliničnih raziskavah referenčnega zdravila, s konzervansom, se je pri 16 – 20 % bolnikov pojavila povečana pigmentacija šarenice, ki je lahko trajna. V odprti 5-letni raziskavi varnosti latanoprosta se je pri 33 % bolnikov pojavila pigmentacija šarenice. Drugi neželeni učinki na oči so običajno prehodni in se pojavijo neposredno ob samem vkapljanju zdravila. Pri timololu so najbolj resni neželeni učinki sistemskega značaja, vključno z bradikardijo, aritmijo, kongestivnim srčnim popuščanjem, bronhospazmom in alergijskimi reakcijami. Kot velja za druga zdravila, ki se jih uporabi lokalno v očeh, se timolol absorbira v sistemski krvni obtok. To lahko povzroči podobne neželene učinke, kot jim opazimo po sistemski uporabi zaviralcev beta, a je pojavnost le-teh po lokalni očesni uporabi manjša. **Imetnik dovoljenja za promet z zdravilom:** Laboratoires THEA, 12 rue Louis Blériot 63017 Clermont-Ferrand Cedex 2, Francija

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Literatura: 1. Centralna baza zdravil [Internet]. Seznam zdravil [citirano: 30.07.2024]. Dostopno na: http://www.cbz.si/cbz/bazazdr2.nsf/o/190SAOF3822EE471C12563860005AB6A?opendocument

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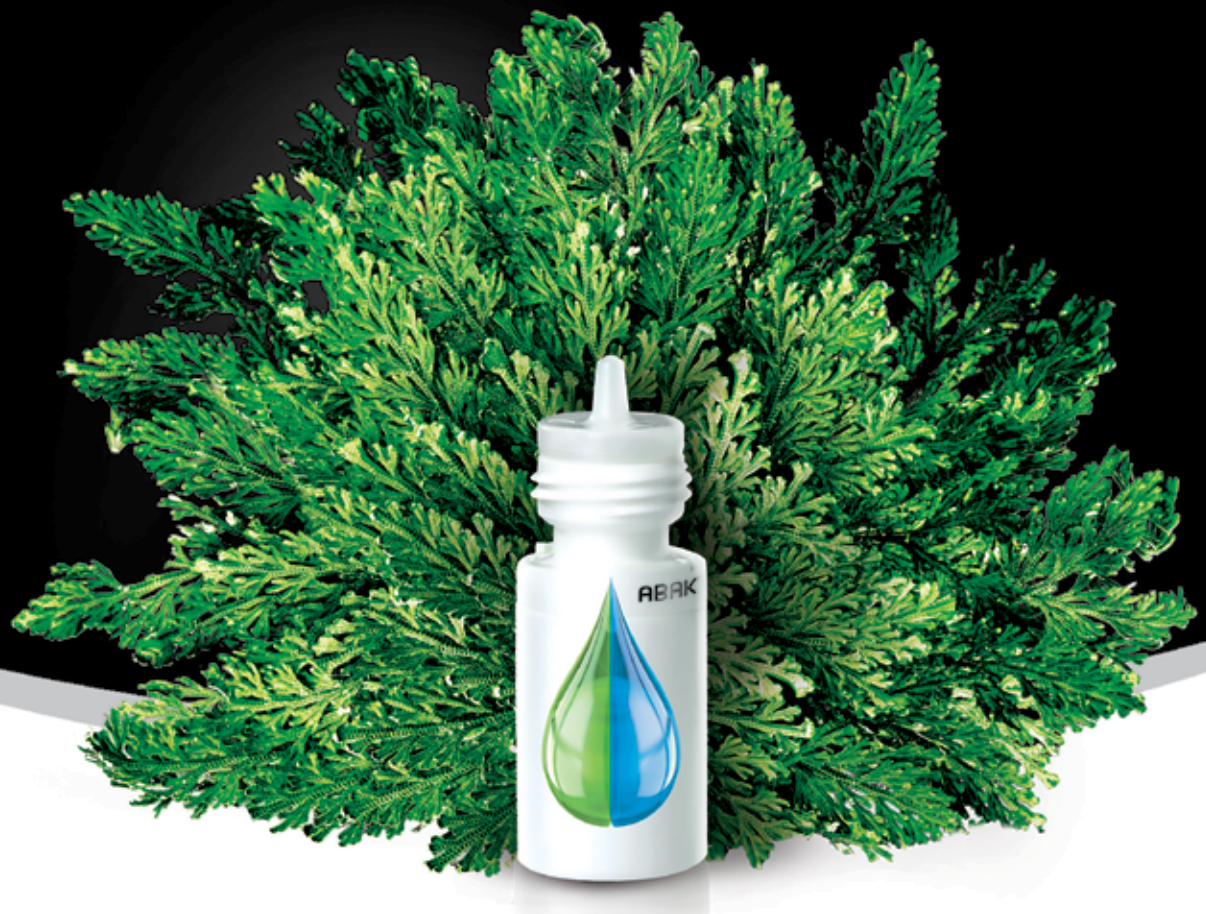

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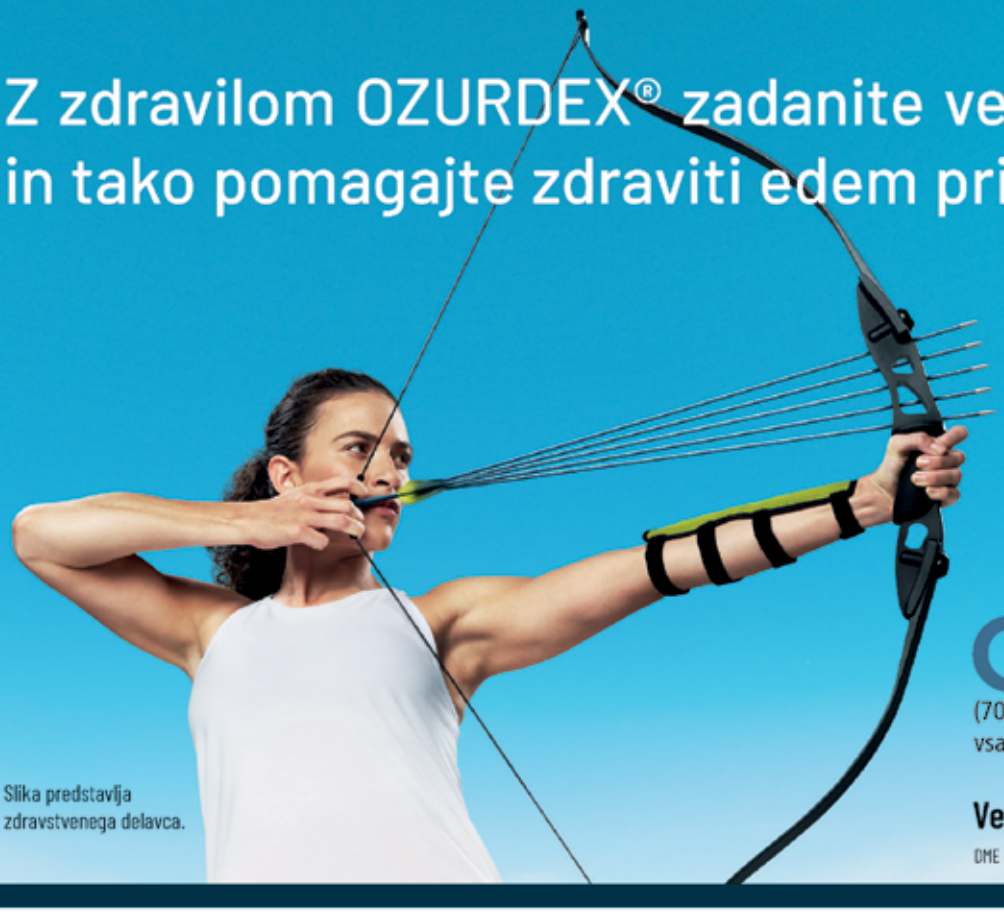

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DME – diabetični makularni edem, RVO – zapora retinalne vene

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OZURDEX 700 mikrogramov intravitrealni vsadek v aplikatorju

Kakovostna in količinska sestava: En vsadek vsebuje 700 mikrogramov deksametazona. **Terapevtske indikacije:** Zdravilo OZURDEX je indicirano za zdravljenje odraslih bolnikov z: okvaro vida zaradi diabetičnega makularnega edema (DME), ki imajo psevdofakijo ali se slabše odzivajo ali niso primerni za zdravljenje brez kortikosteroidov; makularnim edemom po okluziji veje retinalne vene (BRVO) ali okluziji centralne retinalne vene (CRVO); vnetjem posteriornega dela očesa, ki se kaže kot nenalezljivi uveitis. **Odmerjanje in način uporabe:** Zdravilo OZURDEX mora injicirati usposobljen oftalmolog z izkušnjami z intravitrealnim injiciranjem. Odmerjanje: Priporočeni odmerek je en vsadek zdravila OZURDEX v prizadeto oko. Sočasno injiciranje v obe očesi ni priporočljivo. *DME:* Pri bolnikih, zdravljenih z zdravilom OZURDEX, ki so se odzvali na prvo zdravljenje in za katere zdravnik meni, da bi jim ponovno zdravljenje koristilo, ne da bi bili pri tem izpostavljeni znatnemu tveganju, se lahko izvede ponovno zdravljenje. Ponovno zdravljenje se lahko izvede po približno šestih mesecih, če se bolniku poslabša vid in/ali odebeli mrežnica zaradi ponavljajočega se ali poslabšanega stanja diabetičnega makularnega edema. Na področju zdravljenja DME zaenkrat ni izkušenj o učinkovitosti ali varnosti večkratnega odmerjanja pri več kot 7 vsadkih. *RVO in uveitis:* O ponovnem odmerjanju je treba razmisliti, kadar se bolnik odzove na zdravljenje, nato pa se mu ostrina vida zmanjša, pri tem pa ponovno zdravljenje po mnenju zdravnika lahko koristi bolniku, ne da bi bil ta izpostavljen znatnemu tveganju. Bolniki, pri katerih pride do izboljšanega vida in se ta vzdržuje, se ne smejo ponovno zdraviti. Bolniki, pri katerih se pojavi poslabšanje vida, ki ga zdravilo OZURDEX ne ustavi, se ne smejo ponovno zdraviti. Podatki o ponovnem odmerjanju v presledku, krajšem od 6 mesecev, so zelo omejeni. Za informacije o trenutnih izkušnjah glede varnosti večkratne uporabe več kot dveh vsadkov pri nenalezljivem uveitisu posteriornega dela očesa in okluziji retinalne vene, glejte poglavje 4.8. SmPC. *Posebne populacije: Starejši bolniki (≥ 65 let):* Prilagajanje odmerka pri starejših bolnikih ni potrebno. *Okvara ledvic in jeter:* Zdravila OZURDEX niso preučevali pri bolnikih z okvaro ledvic in jeter, vendar pa pri tej populaciji ni potrebna posebna previdnost. *Pediatrična populacija:* Zdravilo OZURDEX ni namenjeno za uporabo pri pediatrični populaciji za indikaciji: diabetični makularni edem in makularni edem po okluziji veje retinalne vene (BRVO) ali okluziji centralne retinalne vene (CRVO). Varnost in učinkovitost zdravila OZURDEX pri pediatrični populaciji z uveitisom nista bili dokazani. Način uporabe: Zdravilo OZURDEX je intravitrealni vsadek v aplikatorju za enkratno uporabo, samo za intravitrealno uporabo. En aplikator se lahko uporabi samo za zdravljenje enega očesa. Postopek intravitrealnega injiciranja je treba opraviti v nadzorovanih aseptičnih pogojih, ki vključujejo uporabo sterilnih rokavic, sterilnega pregrinjala in sterilnega očesnega spekuluma (ali drugega ustreznega instrumenta). Bolnik si mora 3 dni pred vsakim injiciranjem in po njem v oko dajati širokospektralne antibiotične kapljice. Pred injiciranjem je treba razkužiti kožo okoli oči, veko in površino očesa (na primer z nanosom kapljic 5-odstotne raztopine povidonjodida na očesno veznico, kot je bilo to narejeno med kliničnimi preskušanji za odobritev zdravila OZURDEX) ter uporabiti zadostno lokalno anestezijo. Iz škatle vzemite mošnjiček iz folije in preglejte morebitne poškodbe na njem. Potem mošnjiček iz folije odprite na sterilnem polju in aplikator nežno položite na sterilni pladenj. Z aplikatorja previdno odstranite pokrovček. Aplikator je treba uporabiti takoj, ko odprete mošnjiček iz folije. Z eno roko primite aplikator in z njega naravnost povlecite varnostni zavihek. Zavihka ne obračajte ali upogibajte. Medtem ko poševni del igle držite stran od beločnice, jo potisnite približno 1 mm v beločnico, potem pa spremenite smer proti središču očesa v vitrealno votlino in potiskajte, dokler silikonski tulec ne pride v stik z očesno veznico. Počasi potiskajte sprožilni gumb, dokler ne zaslišite klika. Preden izvlečete aplikator iz očesa, se prepričajte, da je sprožilni gumb popolnoma pritisnjen in je poravnan v zaklenjenem položaju s površino aplikatorja. Igle odstranite v isti smeri, kot ste jo uporabili za uvajanje v steklovino. Takoj po injiciranju zdravila OZURDEX uporabite indirektno oftalmoskopijo v kvadrantu injiciranja, da potrdite uspešno vstavljanje vsadka. V veliki večini primerov je vsadek viden. Kadar ne vidite vsadka, uporabite sterilno vatno blazinico in nežno pritisnite na mesto injiciranja, da vsadek postane viden. Po intravitrealnem injiciranju je treba pri bolnikih nadaljevati zdravljenje s širokospektralnim antibiotikom. **Kontraindikacije:** Preobčutljivost na učinkovino ali katero koli pomožno snov; aktivna očesna ali obočesna okužba ali sum nanjo, vključno z večino virusnih bolezni roženice in očesne veznice, kot so aktivni epitelijski herpesni keratitis (dendritični keratitis), vakcinija, norice, mikobakterijske okužbe in glivične bolezni; napredovali glavkom, ki ga ni mogoče zadostno nadzorovati samo z zdravili; afakično oko z raztrgano posteriorno kapsulo leče; oko z umetno lečo v sprednjem prekatu (ACIOL), s pritrjeno umetno lečo na šarenico ali skozi beločnico in raztrgano posteriorno kapsulo leče. **Povzetek posebnih opozoril in previdnostnih ukrepov:** Intravitrealna injiciranja so lahko povezana z endoftalmitisom, intraokularnim vnetjem, zvišanim očesnim tlakom in odstopom mrežnice. Vedno je treba uporabljati ustrezne aseptične tehnike injiciranja. Po injiciranju je treba bolnike spremljati, da se lahko uvede zgodnje zdravljenje, če se pojavi okužba ali zvišan očesni tlak. Bolnikom je treba naročiti, naj takoj poročajo o kakršnih koli simptomih, ki kažejo na endoftalmitis, ali o katerih koli zgoraj omenjenih dogodkih. Pri bolnikih z raztrgano posteriorno kapsulo leče, na primer bolnikih s posteriorno lečo (na primer zaradi operacije katarakte), in/ali bolnikih z odprtino v šarenici proti steklovini (npr. zaradi iridektomije) z vitrektomijo v anamnezi ali brez nje, obstaja tveganje, da se vsadek premakne v sprednji prekat. Premik vsadka v sprednji prekat lahko povzroči edem roženice. Trdovratna huda oblika edema roženice se lahko stopnjuje, tako da je treba roženico presaditi. Pri kontraindiciranih bolnikih se zdravilo OZURDEX ne sme uporabljati, pri ostalih pa ga je treba uporabljati previdno in samo po temeljiti oceni tveganj in koristi. Take bolnike je treba skrbno spremljati, da se omogoči zgodnja diagnoza in obvladovanje morebitnega premika pripomočka. Uporaba kortikosteroidov lahko povzroči nastanek katarakt (vključno s posteriornimi subkapsularnimi kataraktami), zvišan očesni tlak, glavkom, ki ga povzročajo steroidi, in sekundarne očesne okužbe. Pri bolnikih z anamnezo virusne okužbe očesa (npr. s herpesom simpleksom) je treba kortikosteroide uporabljati previdno, pri bolnikih z aktivno okužbo očesa s herpesom simpleksom pa se sploh ne smejo uporabljati. Zdravila OZURDEX niso preučevali pri bolnikih z makularnim edemom, ki je posledica okluzije retinalne vene z obsežno retinalno ishemijo, zato se pri njih uporaba zdravila OZURDEX ne priporoča. Zdravilo OZURDEX je treba previdno uporabljati pri bolnikih, ki jemljejo antikoagulante ali antitrombotike. **Medsebojno delovanje z drugimi zdravili in druge oblike interakcij:** Sistemska absorpcija je minimalna, zato se ne pričakuje medsebojno delovanje z drugimi zdravili. **Plodnost, nosečnost in dojenje:** Nosečnost: Čeprav se pričakuje, da bo sistemska izpostavljenost deksametazonu po lokalnem, intraokularnem zdravljenju zelo majhna, se zdravilo Ozurdex med nosečnostjo ne priporoča, razen če morebitna korist upraviči morebitno tveganje za plod. Dojenje: Deksametazon se izloča v materino mleko. Zaradi poti uporabe in posledičnih sistemskih ravni se ne pričakujejo učinki na otroka, vendar pa se zdravilo OZURDEX med dojenjem ne priporoča, razen če je to nujno potrebno. Plodnost: Ni podatkov o učinkih na plodnost. **Vpliv na sposobnost vožnje in upravljanja strojev:** Zdravilo OZURDEX ima lahko zmeren vpliv na sposobnost vožnje in upravljanja strojev. Pri bolnikih se lahko po intravitrealnem injiciranju zdravila OZURDEX pojavi začasno poslabšanje vida. Dokler se to stanje ne popravi, bolniki ne smejo voziti in upravljati strojev. **Neželeni učinki:** Zelo pogosti: zvišan očesni tlak, katarakta, krvavitev očesne veznice. Pogosti: glavobol, očesna hipertenzija, subkapsularna katarakta, vitrealna krvavitev, zmanjšana ostrina vida, okvara/motnje vida, odstop steklovine, plavajoče motnjave v steklovini, motnjave v steklovini, blefaritis, bolečine v očesu, fotopsija, edem očesne veznice, hiperemija očesne veznice. Občasni: migrena, nekrotizirajoči retinitis, endoftalmitis, glavkom, odstop očesne mrežnice, raztrganina očesne mrežnice, hipotonija očesa, vnetje sprednjega prekata, celice/blešča v sprednjem prekatu, nenormalni občutek v očesu, srbenje vek, hiperemija beločnice, premik pripomočka (vsadka) z edemom roženice ali brez njega, zapleti pri vstavljanju pripomočka (napačna vstavitev vsadka). **Imetnik dovoljenja za promet z zdravilom:** AbbVie Deutschland GmbH & Co. KG, Knollstrasse, 67061 Ludwigshafen, Nemčija. **Način in režim izdajanja:** ZZ – Predpisovanje in izdaja zdravila je le na recept, zdravilo pa se uporablja samo v javnih zdravstvenih zavodih ter pri pravnih in fizičnih osebah, ki opravljajo zdravstveno dejavnost.

Pred predpisovanjem in uporabo, prosimo, preberite celoten Povzetek glavnih značilnosti zdravila.

Datum zadnje revizije besedila: 06/2024.

Reference: 1. OZURDEX® SPC. **2.** Garcia-Layana A et al. Ophthalmologica 2018; doi: 10.1159/000488800 (accessed September 2023). **3.** Wang K et al. Biol Pharm Bull 2008; 31(8): 1541-6. **4.** Rezar-Dreindl S et al. Acta Ophthalmol 2017; 95(2): e119-27. http://dx.doi.org/10.1155/2013/438412 (accessed September 2023). **5.** Edelman JL et al. Exp Eye Res 2005; 80: 249-58. **6.** Tamura H et al. Invest Ophthalmol Vis Sci 2005; 46(4): 1440-4. **7.** Nehme A and Edelman J. Invest Ophthalmol Vis Sci 2008; 49(5): 2030-8.

abbvie AbbVie d.o.o., Dolenjska cesta 242c , 1000 Ljubljana

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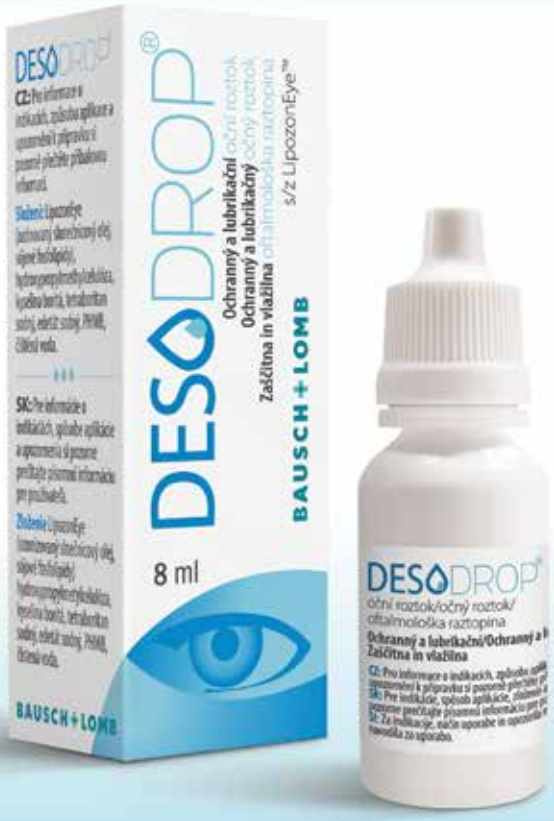

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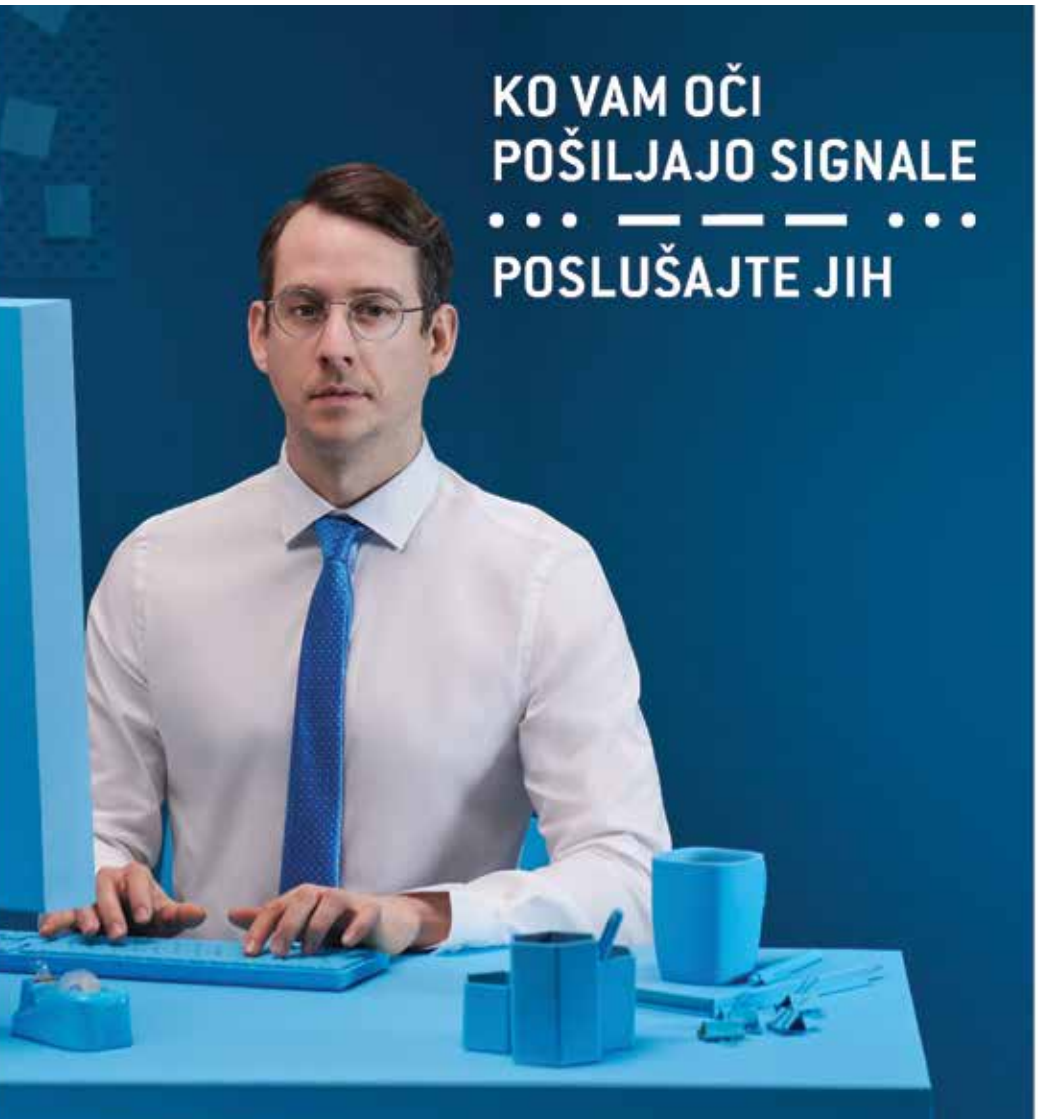

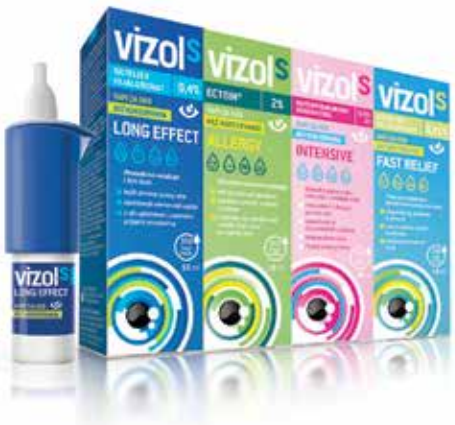

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