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Therapeutic options in the treatment of inflammatory glaucoma

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HIGHLIGHTS

The essence of treatment of inflammatory glaucoma is to control inflammation and maintain normal intraocular pressure. When choosing treatment, the etiology of the inflammation of the ocular vascular membrane should be taken into account.

ABSTRACT

Therapeutic options in inflammatory glaucoma, both pharmacological and surgical methods, have been presented in this paper. Main goals of pharmacotherapy are: controlling inflammation and reducing intraocular pressure. Anti-inflammatory drugs primarily include topical glucocorticoids but also non-steroidal anti-inflammatory drugs and immunomodulatory therapy. Elevated intraocular pressure is treated with β-blockers, carbonic anhydrase inhibitors, prostaglandin analogues and α_2-adrenergic receptor agonists. If pharmacological treatment does not produce the desired results, surgical treatment must be considered. Due to the risk of reactivation of inflammation, it is recommended that it be performed 3 months after the inflammation has healed. Currently, trabeculectomy remains the gold standard. Among the invasive treatment techniques, we discuss trabeculectomy with the use of mitomycin C, micro-hook trabeculectomy, laser therapy, glaucoma drainage implants, deep sclerectomy and viscocanalostomy.

Key words: glaucoma, uveitic glaucoma, inflammation of the ocular vascular membrane, intraocular pressure, ocular hypertension, trabeculectomy, topical glucocorticoids

INTRODUCTION

Glaucoma is a group of eye diseases characterized by damage to the optic nerve, which ultimately leads to visual field deficits and even total loss of vision. One type of glaucoma is inflammatory glaucoma, which is caused by inflammation of the ocular vascular membrane, most commonly its anterior segment. Inflammatory glaucoma develops in 10–20% of patients diagnosed with inflammation of the ocular vascular membrane, with a significantly higher prevalence in chronic rather than acute cases of ocular vascular membrane inflammation [1, 2]. Inflammation of the ocular vascular membrane has a broad etiology that needs to be identified for appropriate treatment selection. Complications leading to glaucoma commonly arise in heterochromic Fuchs' syndrome, Posner–Schlossman syndrome, juvenile idiopathic arthritis, Behçet's disease, Vogt–Koyanagi–Harada syndrome, sympathetic ophthalmia, and sarcoidosis, while infectious etiologies include inflammation of the ocular vascular membrane due to *herpes simplex virus* (HSV), *Varicella zoster virus* (VZV), syphilis, toxoplasmosis, and Lyme disease [1, 3, 4]. Due to the ongoing inflammatory state, treating this condition is very challenging. It is important not to overlook the treatment of the underlying disease, therefore collaboration between specialists, individual patient approach, and holistic patient care is essential during therapy. It is worth noting, that unlike other types of glaucoma, the inflammatory form also affects young individuals who have a prospect of a long life ahead. Therefore, it is crucial to carefully select appropriate therapeutic strategies so that these patients can maintain the best possible quality of vision for as long as possible [5].

PHARMACOLOGICAL TREATMENT

The primary therapeutic goal in the treatment of inflammatory glaucoma is to simultaneously control inflammation of the ocular vascular membrane and maintain appropriate intraocular pressure (IOP). It is important to prevent the formation of peripheral anterior or posterior synechiae. An increase in IOP can be caused by both the ongoing inflammatory state and the use of glucocorticoids, which are the gold standard in the treatment of inflammation of the ocular vascular membrane. In cases where inflammation is the main cause of ocular hypertension, simply controlling the inflammatory state may prevent sudden increases in IOP [1]. Proper IOPs achieved through appropriate treatment of inflammation allow for the discontinuation of hypotensive medications, so understanding the etiology is crucial in treatment planning.

First-line pharmacological treatment for inflammatory glaucoma includes 1% prednisolone acetate [2]. This potent, local glucocorticoid carries a significant risk of IOP elevation, especially in individuals who are steroid-responsive. The stronger the glucocorticoid used, the higher the likelihood of increased IOP [6]. Additionally, it has been shown that the method of administration affects IOP values. Intravitreal or periocular glucocorticoid injection leads to greater elevation [2]. Strong glucocorticoids include prednisolone, betamethasone, and dexamethasone; however, due to their greater penetration into the aqueous humor compared to prednisolone, betamethasone and dexamethasone exhibit 5–7 times stronger systemic anti-inflammatory effects [7]. Glucocorticoids increase aqueous humor secretion and hinder its outflow, which can lead to the development of ocular hypertension [8]. Prolonged use of strong glucocorticoids carries a range of adverse effects, including systemic ones, so the initiation of treatment should be carefully considered in each case. Risk factors for IOP elevation after glucocorticoid use, such as a positive family history, primary open-angle glaucoma, high myopia, diabetes, and connective tissue diseases, should be individually assessed for each patient [9]. It is recommended to start glucocorticoid therapy with a high dose and gradually decrease it in proportion to the reduction in inflammatory activity [10]. Many researchers compare available glucocorticoids based on their impact on IOP elevation. Shokoohi-Rad et al. report that fluorometholone shows the lowest increase in IOP compared to betamethasone and loteprednol etabonate. Researchers categorized the obtained results of IOP elevation after glucocorticoid use into 3 categories: below 6 mmHg, 6–10 mmHg, and above 10 mmHg. The use of fluorometholone did not cause an increase above 10 mmHg, while loteprednol etabonate fared worst in this group, with such an increase observed in 15% of patients, and with betamethasone in 13%. An increase in IOP of 6–10 mmHg during fluorometholone use was 4%, betamethasone 6%, loteprednol etabonate 5%, and an increase below 6 mmHg was observed in 96%, 81%, and 80% of the study participants, respectively [11]. Similar conclusions were reported by Yoo et al. [12], although there are studies where no induction of ocular hypertension was observed after the use of fluorometholone or loteprednol [13]. Clobetasol propionate also shows a smaller impact on IOP elevation compared to dexamethasone and hydrocortisone [14]. Unfortunately, as the tendency to induce ocular hypertension decreases, so does the anti-inflammatory potency of these substances. The anti-inflammatory effect of 0.1% fluorometholone and 0.1% clobetasol propionate is weak [15]. Rimexolone induces ocular hypertension, but compared to dexamethasone [16] and prednisolone [17], the induction is lower. The benefit of using rimexolone is its high anti-inflammatory activity comparable to the equivalent concentration of prednisolone acetate [18].

In case of ineffectiveness or intolerance to glucocorticoid treatment, *The Ocular Immunology and Uveitis Foundation* recommends the use of non-steroidal anti-inflammatory

TABLE 1

Comparison of anti-inflammatory potency and equivalent glucocorticoid dose [19].

Generic name	Trade name	Relative anti-inflammatory activity	Relative sodium-retaining activity	Equivalent dose (mg)
Hydrocortisone (cortisol)	Coref, Hydrocortone	1.0	1.0	20.00
Cortisone acetate	Cortisone, Cortone	0.8	0.8	25.00
Prednisone	Prednicen-M, Orasone, Deltasone, Meticorten	4.0	0.8	5.00
Prednisolone	Prednicen-M, Delta-Cortef, Sterane	4.0	0.8	5.00
Triamcinolone	Aristocort, Kenacort	5.0	0.0	4.00
Methylprednisolone	Medrol	5.0	0.0	4.00
Paramethasone acetate	Haldrone	10.0	0.0	2.00
Fludrocortisone acetate	Florinef	20.0	125.0	0.10
Dexamethasone	Decadron, Hexadrol	25.0	0.0	0.75
Betamethasone	Celestone	25.0	0.0	0.75

drugs and immunomodulatory therapy. Foster et al. suggest that after initial anti-inflammatory therapy, efforts should be made to maintain remission without glucocorticoid use, as most inflammation of the ocular vascular membrane is curable. They propose initiating immunomodulatory drugs for at least 2 years, followed by gradual dose reduction. In immunomodulatory therapy, we use groups of substances such as: antimetabolites, calcineurin inhibitors, alkylating agents, and biological drugs [10]. There are reports of non-steroidal anti-inflammatory drug interactions with latanoprost and brimonidine, weakening their hypotensive effect [20], making them not the primary recommended treatment in inflammatory glaucoma.

Methotrexate is a folate antagonist. As a result of the drug's action, DNA, RNA, and protein synthesis are inhibited, as it specifically targets proliferating cells, particularly in the S phase of the cell cycle. Alongside methotrexate, other antimetabolites include azathioprine and mycophenolate mofetil, which are prodrugs that inhibit purine synthesis necessary for the proper maturation of T lymphocytes [10]. Heo et al. report that these drugs effectively control inflammation, and only a small percentage of patients discontinued methotrexate due to adverse effects, with gastrointestinal discomfort, bone marrow suppression, and elevated liver enzyme activity being the most commonly mentioned adverse effects. The highest incidence of side effects occurred with azathioprine (24% of patients), while the lowest (12%) was observed with mycophenolate mofetil [21].

In the treatment of inflammation of the ocular vascular membrane, calcineurin inhibitors such as cyclosporine A and tacrolimus are not recommended as monotherapy due to their low effectiveness.

Chlorambucil and cyclophosphamide are strongly immunomodulating alkylating drugs used only in very severe inflammation of the ocular vascular membrane when other treatments have not yielded the desired results. Their use carries the risk of serious adverse effects such as bone marrow suppression, carcinogenic effects, or infertility, hence regular blood morphology tests are necessary [10].

Biological drugs include: monoclonal antibodies against tumor necrosis factor α, such as adalimumab and infliximab, as well as anti-CD20 antibodies (rituximab), anti-IL-6 (tocilizumab), anti-IL-17 (secukinumab), and anti-IL-12 and anti-IL-23 (ustekinumab). The only drug from this group registered for the treatment of inflammation of the ocular vascular membrane is adalimumab [22]. Biological drugs, due to an increasing number of publications indicating their effectiveness and relatively few adverse effects, may soon be widely used in the anti-inflammatory treatment of the ocular vascular membrane.

To prevent the formation of posterior synechiae, which can worsen visual acuity, local mydriatics and cycloplegics can be used. These drugs alleviate symptoms such as pain and photophobia. The reduction of inflammation occurs as a result of the relaxation of the structures of the anterior segment of the ocular vascular membrane [10]. These include atropine, homatropine, tropicamide, and cyclopentolate.

Another significant aspect of pharmacological treatment is lowering IOP. Unfortunately, at this stage, we also encounter obstacles because most hypotensive drugs used during active inflammation demonstrate weakened effectiveness or show none at all [8].

The primary hypotensive drugs include β-blockers and carbonic anhydrase inhibitors, which reduce aqueous humor production. The efficacy of lowering IOP is comparable for β-blockers, carbonic anhydrase inhibitors, and α_2-adrenergic antagonists in combination with prostaglandin analogues, with the latter performing the worst [23]. Topically applied β-blockers such as timolol, betaxolol, levobunolol, metipranolol, and carteolol are well tolerated and highly effective in lowering IOP [24]. They have the lowest risk of adverse effects such as conjunctival hyperemia [25]. However, they cannot be used in all patients due to contraindications such as obstructive airway disease or cardiac rhythm

disturbances. Among β-blockers, timolol is the most commonly used drug, recommended for use up to twice daily. Possible adverse effects include burning, stinging, itching, and tearing of the eyes [26].

Topical carbonic anhydrase inhibitors such as dorzolamide and brinzolamide are used up to 2–3 times a day [1]. Among them, dorzolamide is the leader and is often prescribed in cases of contraindications to β-blockers or as adjunctive therapy [26]. In terms of effectiveness, it is comparable to betaxolol and only slightly less effective than timolol [27]. Unfortunately, allergic reactions have been reported quite often by patients using dorzolamide [28].

In cases where lowering IOP is ineffective, acetazolamide can be used systemically at a dose of 250–1000 mg per day [1]. It is used to treat acute episodes of ocular hypertension and before filtering surgeries [5]. However, its administration carries the risk of systemic adverse effects such as diuresis disturbances, gastrointestinal disorders, limb paresthesia, metabolic changes, and malaise [2].

Another option for treating ocular hypertension is the α_2-adrenergic receptor agonist brimonidine. Its hypotensive effect is achieved by simultaneously reducing aqueous humor production and increasing uveoscleral outflow [22]. Currently, prostaglandin analogues are considered the gold standard despite reports of their potential proinflammatory effects. A meta-analysis conducted by Hu et al. showed that the incidence of cystoid macular edema or recurrence of ocular vascular membrane inflammation in individuals treated with prostaglandin analogues (without prior ocular surgery) was 0.22% and 0.09%, respectively [29]. Prostaglandin analogues lower IOP by improving uveoscleral outflow (unconventional pathway) and trabecular meshwork outflow. In monotherapy, prostaglandin analogues are the most effective in lowering IOP [25]. According to Siddique et al. bimatoprost is a well-tolerated drug that effectively lowers IOP [1]. The safety and effectiveness of using prostaglandin analogues such as latanoprost, bimatoprost, and travoprost were demonstrated in a study by Sallam et al. [30]. A significant evidence of no significant increase in the risk of occurrence or exacerbation of inflammation with latanoprost therapy is a large randomized double-blind trial developed by Watson and Stjernschantz. Interestingly, an increase in iris pigmentation was observed during latanoprost use in the study [31]. Another atypical side effect is the increase, elongation, thickening, and increased pigmentation of eyelashes [32], which may be a desirable effect for some patients. However, caution should be exercised during prostaglandin analog therapy, and the possibility of recurrence of inflammation should be considered. The use of these drugs is relatively contraindicated in the case of cystoid macular edema episodes and herpetic ocular vascular membrane inflammation [33].

SURGICAL TREATMENT

Surgical treatment is indicated in cases of ineffectiveness or intolerance to pharmacological treatment. Studies indicate that approximately 30% of uveitic glaucoma patients will require surgical intervention [4]. The main problem causing complications during uveitic glaucoma surgery is inflammation. It is important to avoid qualifying for invasive treatment methods during active inflammation.

It is recommended to perform a planned surgical procedure deferred for 3 months after reducing inflammation, through preoperative administration of glucocorticoids [33]. Glaucoma surgeries, due to the high risk of complications, including vision loss, are carried out urgently, which significantly reduces the effectiveness of treatment due to limited possibilities. Preoperative control of inflammation plays a crucial role as it is responsible for surgical failure. Postoperative fibrosis and proinflammatory cytokines are the main complicating factors. During treatment, one should not forget about the side effects of glucocorticoid use. The duration of glaucoma and ocular vascular membrane inflammation is also important; the longer it lasts, the higher the likelihood of reoperation [34].

There are many different surgical methods used to treat uveitic glaucoma, each of which may lead to the reactivation of inflammation. Trabeculectomy is currently considered the gold standard among surgical procedures for patients with uveitic glaucoma. However, due to its invasiveness, new methods are continually sought to improve treatment efficacy [33].

Preferred trabeculectomy with the use of mitomycin C involves filtering fluid from the anterior chamber through a bleb to control IOP [35]. Antimetabolites such as mitomycin C or 5-fluorouracil, which reduce bleb scarring and prolong the functionality of the filtration fistula, despite their benefits, may expose patients to intraoperative wound leakage, increased risk of infection, hypotony, maculopathy, and choroidal detachment [33].

Microhook trabeculectomy as a new minimally invasive surgical technique for glaucoma treatment has yielded satisfactory results. It achieved IOP control with simultaneous absence of exacerbation of inflammation after 6 weeks post-operation. Postoperative complications occurred in 58%, but they were mild and transient [36]. The advantages of this method include no interference with the conjunctiva and sclera, reduced damage to Descemet's membrane and the iris, and a wider incision in the trabecular meshwork. Microhook trabeculectomy allows for an incision of up to approximately 240°, which results in faster IOP reduction. However, it may also lead to side effects such as anterior chamber hemorrhage and transient IOP elevation [37]. In a study by Mori et al. comparing trabeculectomy with microhook trabeculectomy 1 year after surgery, the surgical success rate was 71.2% for the conventional method and

74.2% for patients undergoing microhook trabeculectomy [38].

Another option is laser therapy, which is mainly used in the treatment of narrow-angle inflammatory glaucoma. Peripheral iridotomy is indicated for acute angle-closure glaucoma caused by pupillary blockage. The severity of the inflammatory process and issues with maintaining the patency of the iridotomy are the main factors contributing to postoperative failure [33]. Transscleral diode laser cyclophotocoagulation (TDLC) involves using laser radiation to ablate the epithelium of the ciliary body. Destroying the epithelium of the ciliary body aims to minimize aqueous humor secretion. The TDLC technique is used in the treatment of severe cases of uveitic glaucoma that are resistant to other therapy methods. In TDLC, the energy level generated by the laser is crucial. Cakir et al. demonstrated that the risk of exacerbating inflammatory reactions correlates with the amount of energy delivered [39]. Excessive energy power delivered to the eye and a large area of treatment increase the risk of TDLC procedure complications. Persistent hypotonia and even phthisis bulbi can be among the consequences of the TDLC procedure [40].

In cases of failure of the first-line method, glaucoma drainage devices are used [37]. The aqueous shunt surgery involves creating a new pathway for aqueous humor flow from the anterior chamber to a fibrous bleb around the implant. Valve-equipped shunts such as Ahmed and valveless shunts such as Baerveldt and Molteno are distinguished. These methods significantly lower IOP [41].

According to Mercieca et al. non-penetrating procedures such as deep sclerectomy and viscocanalostomy may have a beneficial effect on patients with uveitic glaucoma or after previous surgery. The study showed a lower incidence of postoperative inflammatory complications than the first-line method. Deep sclerectomy has been recognized as a safe and effective procedure for lowering IOP in glaucoma. Like with other techniques, some patients will require reoperation after a longer period [42].

In the study by El-Saied et al. a comparative analysis did not show a significant difference between trabeculectomy, TDLC, and Ahmed glaucoma valve. In the longer term, the impact on lowering IOP and the overall success rate were similar. Postoperatively, due to limited inflammation in the anterior chamber with the use of Ahmed glaucoma valve, the authors point to the superiority of this method [43].

CONCLUSION

Treatment of inflammatory glaucoma is undeniably a challenging clinical task, often involving doctors from various specialties. Rapid control of the inflammatory process seems to be crucial, which is why initiating anti-inflammatory treatment with high doses of topical glucocorticoids is recommended, followed by maintaining remission through immunomodulatory therapy. Strong glucocorticoids can be safely used for short periods, but if the clinical situation requires longer use, other options for anti-inflammatory drugs should be considered. To normalize IOP, individual selection of hypotensive medication for each patient is recommended, but currently, prostaglandin analogues appear to be the most appropriate choice if there are no contraindications. Despite the wide range of invasive procedures available, trabeculectomy remains the gold standard. The choice of surgical method can be tailored individually to the patient's needs and the operator's competence. It is important to consider the risk of reactivation of inflammation due to surgical intervention, which is why achieving remission through appropriate pharmacotherapy is preferred. Of course, if these actions do not yield results, delaying the decision to implement invasive treatment due to the irreversible effects of ongoing inflammation and ocular hypertension is not advisable.

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References

1. Siddique SS, Suelves AM, Baheti U et al. Glaucoma and uveitis. Surv Ophthalmol. 2013; 58(1): 1-10. http://doi.org/10.1016/j.survophthal.2012.04.006.
2. Sayed MS, Lee RK. Current management approaches for uveitic glaucoma. Int Ophthalmol Clin. 2015; 55(3): 141-60. http://doi.org/10.1097/IIO.0000000000000071.
3. Łazicka-Gałecka M, Guszkowska M, Gałecki T et al Epidemiology, pathophysiology and diagnosis of uveitic glaucoma and ocular hypertension secondary to uveitis. Klinika Oczna/Acta Ophthalmologica Polonica. 2023; 125(1): 7-12. http://doi.org/10.5114/ko.2023.126355.
4. Muñoz-Negrete FJ, Moreno-Montañés J, Hernández-Martínez P et al. Current Approach in the Diagnosis and Management of Uveitic Glaucoma. Biomed Res Int. 2015; 2015: 742792. http://doi.org/10.1155/2015/742792.
5. Sherman ER, Cafiero-Chin M. Overcoming diagnostic and treatment challenges in uveitic glaucoma. Clin Exp Optom. 2019; 102(2): 109-15. http://doi.org/10.1111/cxo.12811.
6. Cantrill HL, Palmberg PF, Zink HA et al. Comparison of in vitro potency of corticosteroids with ability to raise intraocular pressure. Am J Ophthalmol. 1975; 79(6): 1012-7. http://doi.org/10.1016/0002-9394(75)90687-x.
7. McGhee CN, Dean S, Danesh-Meyer H. Locally administered ocular corticosteroids: benefits and risks. Drug Saf. 2002; 25(1): 33-55. http://doi.org/10.2165/00002018-200225010-00004.
8. Sung VC, Barton K. Management of inflammatory glaucomas. Curr Opin Ophthalmol. 2004; 15(2): 136-40. http://doi.org/10.1097/00055735-200404000-00014.
9. Phulke S, Kaushik S, Kaur S et al. Steroid-induced Glaucoma: An Avoidable Irreversible Blindness. J Curr Glaucoma Pract. 2017; 11(2): 67-72. http://doi.org/10.5005/jp-journals-l0028-1226.
10. Foster CS, Kothari S, Anesi SD et al. The Ocular Immunology and Uveitis Foundation preferred practice patterns of uveitis management. Surv Ophthalmol. 2016; 61(1): 1-17. http://doi.org/10.1016/j.survophthal.2015.07.001.
11. Shokoohi-Rad S, Daneshvar R, Jafarian-Shahri M et al. Comparison between Betamethasone, Fluorometholone and Loteprednol Etabonate on intraocular pressure in patients after keratorefractive surgery. J Curr Ophthalmol. 2017; 30(2): 130-5. http://doi.org/10.1016/j.joco.2017.11.008.
12. Yoo YJ, Yang HK, Hwang JM. Efficacy and Safety of Loteprednol 0.5% and Fluorometholone 0.1% After Strabismus Surgery in Children. J Ocul Pharmacol Ther. 2018; 34(6): 468-76. http://doi.org/10.1089/jop.2017.0145.
13. Karimian F, Faramarzi A, Fekri S et al. Comparison of Loteprednol with Fluorometholone after Myopic Photorefractive Keratectomy. J Ophthalmic Vis Res. 2017; 12(1): 11-6. http://doi.org/10.4103/2008-322X.200161.
14. Eilon LA, Walker SR. Clinical evaluation of clobetasone butyrate eye drops in the treatment of anterior uveitis and its effects on intraocular pressure. Br J Ophthalmol. 1981; 65(9): 644-7. http://doi.org/10.1136/bjo.65.9.644.
15. Leibowitz HM, Ryan WJ Jr, Kupferman A. Comparative anti-inflammatory efficacy of topical corticosteroids with low glaucoma-inducing potential. Arch Ophthalmol. 1992; 110(1): 118-20. http://doi.org/10.1001/archopht.1992.01080130120038.
16. Al Hanaineh AT, Hassanein DH, Abdelbaky SH et al. Steroid-induced ocular hypertension in the pediatric age group. Eur J Ophthalmol. 2018; 28(4): 372-7. http://doi.org/10.1177/1120672118757434.
17. Leibowitz HM, Bartlett JD, Rich R et al. Intraocular pressure-raising potential of 1.0% rimexolone in patients responding to corticosteroids. Arch Ophthalmol. 1996; 114(8): 933-7. http://doi.org/10.1001/archopht.1996.01100140141005.
18. Arellanes-García L, Padilla-Aguilar G, Navarro-López P et al. Comparación de la eficacia de la prednisolona y la rimexolona en el tratamiento de iridociclitis aguda en pacientes HLA-B27 positivos [Efficacy of prednisolone and rimexolone in HLA-B27 positive patients with acute anterior uveitis]. Gac Med Mex. 2005; 141(5): 363-6.
19. Sendrowski DP, Jaanus SD, Semes LP et al. Anti-Inflammatory Drugs. In: Clinical ocular pharmacology. Fifth edition. Bartlett JD, Jaanus SD (eds.). Butterworth-Heinemann, St. Louis 2008: 223.
20. Sponsel WE, Paris G, Trigo Y et al. Latanoprost and brimonidine: therapeutic and physiologic assessment before and after oral nonsteroidal anti-inflammatory therapy. Am J Ophthalmol. 2002; 133(1): 11-8. http://doi.org/10.1016/s0002-9394(01)01286-7.
21. Heo J, Sepah YJ, Yohannan J et al. The role of biologic agents in the management of non-infectious uveitis. Expert Opin Biol Ther. 2012; 12(8): 995-1008. http://doi.org/10.1517/14712598.2012.688021.
22. Škrlová E, Svozílková P, Heissigerová J et al. Pathogenesis and current methods of treatment of secondary uveitic glaucoma. A review. Cesk Slov Oftalmol. 2023; 79(3): 111-115. http://doi.org/10.31348/2023/7.
23. Tanna AP, Rademaker AW, Stewart WC et al. Meta-analysis of the efficacy and safety of alpha2-adrenergic agonists, beta-adrenergic antagonists, and topical carbonic anhydrase inhibitors with prostaglandin analogs. Arch Ophthalmol. 2010; 128(7): 825-33. http://doi.org/10.1001/archophthalmol.2010.131.
24. Zimmerman TJ. Topical ophthalmic beta blockers: a comparative review. J Ocul Pharmacol. 1993; 9(4): 373-84. http://doi.org/10.1089/jop.1993.9.373.

25. Li F, Huang W, Zhang X. Efficacy and safety of different regimens for primary open-angle glaucoma or ocular hypertension: a systematic review and network meta-analysis. Acta Ophthalmol. 2018; 96(3): e277-e284. http://doi.org/10.1111/aos.13568.
26. Gupta SK, Niranjan DG, Agrawal SS et al. Recent advances in pharmacotherapy of glaucoma. Indian J Pharmacol. 2008; 40(5): 197-208. http://doi.org/10.4103/0253-7613.44151.
27. Strahlman E, Tipping R, Vogel R. A double-masked, randomized 1-year study comparing dorzolamide (Trusopt), timolol, and betaxolol. International Dorzolamide Study Group. Arch Ophthalmol. 1995; 113(8): 1009-16. http://doi.org/10.1001/archopht.1995.01100080061030.
28. Talluto DM, Wyse TB, Krupin T. Topical carbonic anhydrase inhibitors. Curr Opin Ophthalmol. 1997; 8(2): 2-6. http://doi.org/10.1097/00055735-199704000-00002.
29. Hu J, Vu JT, Hong B et al. Uveitis and cystoid macular oedema secondary to topical prostaglandin analogue use in ocular hypertension and open angle glaucoma. Br J Ophthalmol. 2020; 104: 1040-4.
30. Sallam A, Sheth HG, Habot-Wilner Z et al. Outcome of raised intraocular pressure in uveitic eyes with and without a corticosteroid-induced hypertensive response. Am J Ophthalmol. 2009; 148(2): 207-13.e1. http://doi.org/10.1016/j.ajo.2009.02.032.
31. Watson P, Stjernschantz J. A six-month, randomized, double-masked study comparing latanoprost with timolol in open-angle glaucoma and ocular hypertension. The Latanoprost Study Group. Ophthalmology. 1996; 103(1): 126-37. http://doi.org/10.1016/s0161-6420(96)30750-1.
32. Johnstone MA. Hypertrichosis and increased pigmentation of eyelashes and adjacent hair in the region of the ipsilateral eyelids of patients treated with unilateral topical latanoprost. Am J Ophthalmol. 1997; 124(4): 544-7. http://doi.org/10.1016/s0002-9394(14)70870-0.
33. Łazicka-Gałecka M, Guszkowska M, Gałecki T et al. Review of pharmacological and surgical treatment options of uveitic glaucoma. Klinika Oczna/Acta Ophthalmologica Polonica. 2023; 125(2): 65-74. http://doi.org/10.5114/ko.2022.117207.
34. Magliyah MS, Badawi AH, Alshamrani AA et al. The Effect of Perioperative Uveitis Control on the Success of Glaucoma Surgery in Uveitic Glaucoma. Clin Ophthalmol. 2021; 15: 1465-75. http://doi.org/10.2147/OPTH.S301648.
35. Kanaya R, Kijima R, Shinmei Y et al. Surgical Outcomes of Trabeculectomy in Uveitic Glaucoma: A Long-Term, Single-Center, Retrospective Case-Control Study. J Ophthalmol. 2021; 2021: 5550776. http://doi.org/10.1155/2021/5550776.
36. Sotani N, Kusuhara S, Matsumiya W et al. Outcomes of Microhook ab Interno Trabeculotomy in Consecutive 36 Eyes with Uveitic Glaucoma. J Clin Med. 2022; 11(13): 3768. http://doi.org/10.3390/jcm11133768.
37. Yokoyama H, Takata M, Gomi F. One-year outcomes of microhook trabeculotomy versus suture trabeculotomy ab interno. Graefes Arch Clin Exp Ophthalmol. 2022; 260(1): 215-24. http://doi.org/10.1007/s00417-021-05333-7.
38. Mori S, Tanito M, Shoji N et al. Noninferiority of Microhook to Trabectome: Trabectome versus Ab Interno Microhook Trabeculotomy Comparative Study (Tram Trac Study). Ophthalmology Glaucoma. 2022; 5(4): 452-61. http://doi.org/10.1016/j.ogla.2021.11.005.
39. Cakir I, Altan C, Yalcinkaya G et al. Anterior chamber laser flare photometry after diode laser cyclophotocoagulation. Photodiagnosis Photodyn Ther. 2022; 37: 102580. http://doi.org/10.1016/j.pdpdt.2021.102580.
40. Aujla JS, Lee GA, Vincent SJ et al. Incidence of hypotony and sympathetic ophthalmia following trans-scleral cyclophotocoagulation for glaucoma and a report of risk factors. Clin Exp Ophthalmol. 2013; 41(8): 761-72. http://doi.org/10.1111/ceo.12088.
41. Chow A, Burkemper B, Varma R et al. Comparison of surgical outcomes of trabeculectomy, Ahmed shunt, and Baerveldt shunt in uveitic glaucoma. J Ophthalmic Inflamm Infect. 2018; 8(1): 9. http://doi.org/10.1186/s12348-018-0150-y.
42. Mercieca K, Steeples L, Anand N; Medscape. Deep sclerectomy for uveitic glaucoma: long-term outcomes. Eye (Lond). 2017; 31(7): 1008-19. http://doi.org/10.1038/eye.2017.80.
43. El-Saied HMA, Abdelhakim MASE. Different surgical modalities for management of uveitic glaucoma: 2 year comparative study. Acta Ophthalmol. 2022; 100(1): e246-52. http://doi.org/10.1111/aos.14889.

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