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Secondary glaucoma associated with aphakia following complicated surgery for senile cataract – a review

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ABSTRACT

Aphakia is an independent risk factor for the development of secondary glaucoma. The literature on this type of glaucoma focuses mainly on pediatric patients, less often adults. The aim of the work is to present the current knowledge on the epidemiology, causes of development and possible treatment of secondary glaucoma in adult aphakic patients after cataract surgery. According to the literature, in the years 1970-1980, glaucoma in aphakic eyes accounted for 37.7% of cases of secondary glaucoma in the population of adults and children, and at the beginning of the 21st century this percentage decreased to 11%. Nevertheless, secondary glaucoma in aphakic eyes is diagnosed in 17.7% of cases. Secondary glaucoma in aphakia is characterized by unclear etiopathogenesis, and often diagnostic and therapeutic difficulties. The most common pathomechanisms of this type of glaucoma include: peripheral anterior synechiae, pupillary block, the presence of a loose vitreous in an anterior chamber and an angle region. Less common ones include: epithelial downgrowth into the anterior chamber, ciliovitreal block (malignant glaucoma), the presence of degenerated red blood cells in the angle region (ghost cell glaucoma) and the so far little known mechanisms related to chronic inflammation. Treatment of secondary glaucoma in aphakic eyes includes a wide range of methods which should be used considering the possible pathomechanism in advance. The success of trabeculectomy is not satisfactory. It seems that glaucoma drainage devices can be used with therapeutic effect. There is still a need for more research to expand our knowledge on this topic.

KEY WORDS: aphakia, secondary glaucoma, aphakic glaucoma, complicated cataract surgery.

INTRODUCTION

Aphakia is defined as an absence of a lens. It can be congenital or acquired. Congenital aphakia is subdivided into: primary (no lens formation) and secondary (resorption of the lens after its partial formation and before birth). The acquired one is subdivided into: postoperative and traumatic [1, 2].

Aphakia is an independent risk factor for the development of secondary glaucoma [3]. The literature focuses mainly on pediatric patients [4].

The aim of the work is to present the current state of knowledge on secondary glaucoma in aphakic patients following complicated surgery for senile cataract. Currently, this issue is not widely discussed due to the small number of aphakic patients. The development of surgical techniques and the introduction of phacoemulsification to cataract surgery significantly reduced the percentage of aphakic patients [4-8]. For this reason, the issue of secondary glaucoma in aphakia is not of much interest today. It should be remembered that aphakic patients are still referred to an ophthalmologist; therefore the presentation of current data on the relationship between aphakia and the occurrence of secondary glaucoma in adults seems to be most justified.

APHAKIA – EPIDEMIOLOGY

The prevalence of pseudophakia/aphakia in the adult population of the United States of America in 2000 was estimated at 5.1% (6.1 million people) and, according to follow-up estimates, was expected to increase to 9.5 million of the US population in 2020 [9]. It was to result from the expected increase in the prevalence of cataracts, and thus the number of cataract surgery [9].

Contemporaneously, the most common form of aphakia in the adult population is postoperative aphakia following complicated surgery for senile cataract (43.36% [3], 56.41% [2]). The most common complications that result in leaving

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aphakia are: posterior lens capsule rupture without or with displacement of the lens nucleus fragments, when there is insufficient capsular support for implantation of an artificial intraocular lens.

According to a large multicenter study conducted in 1997-2001, in the Swedish population [7], postoperative aphakia occurred in 0.49% of eyes after cataract surgery (1 in 200 cataract operations out of 287,951 analyzed procedures, of which approximately 95% were performed by phacoemulsification). However, a much higher percentage of postoperative aphakia was observed in the same years in developing countries [5, 6]. In 2000, in a study [5] on the Indian population aged 50 years and older, postoperative aphakia affected 43.2% of eyes (n = 386) undergoing cataract surgery (n = 893). In this study, a high percentage of the postoperative aphakia was significantly related to the surgical technique: 61.4% (n = 237) of aphakic eyes had undergone an intracapsular cataract extraction (ICCE), 33.7% (n = 130) an extracapsular cataract extraction (ECCE), and in 4.9% (n = 19) the surgical technique could not be determined. The financial aspect was probably not without significance – the implantation of an artificial intraocular lens was not completely free.

The aim of modern cataract surgery is usually, apart from cataract removal, also to implant an artificial intraocular lens into the lens capsule. Postoperative aphakia is usually the result of intraoperative complications [4]. The incidence of one of the most serious of them, i.e. dropped nucleus into the vitreous, showed a significant tendency to decrease from year to year (data from a very large retrospective cross-sectional study conducted in 2008-2018 on a population of 1,715,348 patients from 18 European countries) [8]. This trend can be explained, among other factors, by an increasingly improved surgical technique.

Therefore, the reduction in the frequency of postoperative aphakia was achieved primarily due to the progress of cataract surgery [4] characterized by the introduction of modern surgical techniques (ECCE, phacoemulsification) and their further improvement.

The following conditions can increase the risk of postoperative aphakia: corneal opacities, dense cataract, glaucoma, high myopia, previous vitrectomy, traumatic cataract or pseudoexfoliation syndrome [3, 4, 7]. Less frequently, postoperative aphakia is an intended effect in cases such as: congenital cataract, traumatic cataract, high myopia and aphakia in the fellow eye [4]. In a Swedish study, in 87.1% of cases postoperative aphakia was not planned preoperatively [10].

Taking the above into consideration, it should be mentioned that in groups at high risk of postoperative aphakia its frequency increases significantly. According to a Russian study, postoperative aphakia affected as many as 90.8% of cases (177/195 eyes) among patients with traumatic cataracts following a penetrating eyeball trauma and a history of removal of an intraocular foreign body [11].

As reported by Arvind *et al.* [3], aphakia is an independent risk factor for the development of secondary glaucoma.

SECONDARY GLAUCOMA IN APHAKIA – EPIDEMIOLOGY

The prevalence of secondary glaucoma in aphakic patients decreased at the turn of the 20th/21st centuries. In the years 1970-1980, secondary glaucoma in aphakic patients accounted for 37.7% of cases of secondary glaucoma in the combined adult and pediatric population (Indian population study), being by far the most common type. At the beginning of the 21st century, according to a large study (n = 2650; Indian population study 2005), the percentage had decreased to 11% [12]. The reasons for the decreasing trend of the prevalence of secondary glaucoma in aphakic patients are seen, as in the case of the prevalence of aphakia, in the progression of cataract surgery [12].

Currently, secondary glaucoma in aphakic or pseudophakic patients affects only about 2.2% of the adult population with glaucoma or ocular hypertension according to a large study of 5,530 Korean patients from 2001 to 2016 [13]. On the other hand, as many as 17.7% of adult aphakic patients have secondary glaucoma (Indian population study) [3].

Taking into account the above epidemiological data, despite the low percentage of secondary glaucoma in aphakic patients among other types of glaucoma, due to the expected increase in the prevalence of aphakia, and therefore most likely also secondary glaucoma in aphakia, we would like to draw attention to the importance of this issue. Our position is also argued by the lessons of previous experiences, which show that this type of glaucoma has already been a significant problem resulting from unclear etiopathogenesis, diagnostic difficulties and, as a result, therapeutic difficulties [4, 14]. This may be argued by the mean vertical cup/disc ratio among patients with secondary glaucoma in aphakia, which, according to a Turkish study from 1990 to 2011, was as high as 0.78 ±0.24 at the end of the study [4].

SECONDARY GLAUCOMA IN APHAKIA – ETIOPATHOGENESIS

The term "aphakic glaucoma" suggests that aphakia is *per se* the cause of glaucoma [15]. However, the etiopathogenesis of glaucoma associated with aphakia is not entirely clear. The aphakia causes complicated mechanical and biochemical changes in the structures of the anterior segment of the eyeball and the vitreous [4]. However, the term "aphakic glaucoma" is a term commonly used by many clinicians [15].

In pediatric ophthalmology, it specifically refers to glaucoma that occurs after congenital cataract surgery [15]. On the website of the American Academy of Ophthalmology, in the expansion of the definition of "aphakic glaucoma" is a description of this disease only in this context [16, as of 05/12/2020].

The term "aphakic glaucoma", referring to adult patients, does not appear at all in the guidelines of the European Glaucoma Society [17]. Nevertheless, there are publications using this term in adult patients [14, 18-20]. Importantly, the term seems to have purposely been used in these publications to clearly emphasize the fact that the group of aphakic patients with primary glaucoma (including previously undiagnosed

glaucoma) and patients with glaucoma secondary to diseases unrelated to aphakia (e.g. recurrent inflammatory glaucoma) were excluded from the considerations [14, 19]. Considering the above, we would like to point out that in our study, concerning adult patients, we use the terminology of "secondary glaucoma in aphakia" with the reservation that the current study involves glaucoma associated with pathomechanisms developing as a result of aphakia following complicated surgery for senile cataract.

Several mechanisms may be involved in the development of secondary glaucoma in aphakia (the analysis excludes patients with a history of traumatic cataracts and/or *pars plana* vitrectomy):

1. Pupillary block [14, 18, 21]:
   a) associated with complete adhesion of the iris to the intact hyaloid membrane, lens capsule or residual cortical masses [14, 21],
   b) associated with increasing vitreous herniation into the pupil, without rupture of the hyaloid membrane [14, 18, 21].

2. Disruption of the hyaloid membrane with the presence of a loose vitreous in the deep anterior chamber and angle region, blocking the outflow of aqueous humor [14, 21, 22]. This mechanism occurs without pupillary block [21]. It is a type of open-angle glaucoma, with a possible acute course [22].

3. Peripheral anterior synechiae leading to closure of the angle [4, 14]. The causes of peripheral anterior synechiae formation include: shallowing of the anterior chamber due to leakage from the postoperative wound, as well as postoperative iritis and iridocyclitis [14].

4. Corneal epithelium downgrowth by the postoperative wound leading to closure of the angle [4, 14, 19, 23, 24].

5. Ciliovitreal block (malignant glaucoma) [4, 14, 25-28], in which the pressure of the vitreous on the ciliary body, as well as blockage of the space between the ciliary body and the iris by the vitreous, leads to an incorrect direction of the outflow of aqueous humor towards the rearward [28] and as a result to closure of the angle [26].

6. Ghost cell glaucoma mechanism [4, 29, 30]. This situation occurs in patients with/after long-term vitreous hemorrhage and rupture of the anterior face of the vitreous (posttraumatic or postoperative, including cataract surgery) [30]. Within 1-3 weeks after vitreous hemorrhage, partially degenerated erythrocytes are formed, which remain in the vitreous for months after the bleeding has stopped [29]. These degenerated erythrocytes can get into the anterior chamber by disrupting the anterior face of the vitreous and cause direct obstruction of the trabecular meshwork with a secondary intraocular pressure (IOP) increase [29]. It is a mechanism of the development of ghost cell glaucoma – a type of secondary open-angle glaucoma [29].

7. Protracted inflammation [4, 31-33].

There are discrepancies between studies with regard to the most frequent pathomechanism of secondary glaucoma in aphakic patients. According to the study by Ekşioğlu *et al.* [4] carried out on 29 aphakic eyes (patients after cataract surgery in adulthood) with secondary glaucoma, the most common pathomechanism was the presence of anterior synechiae (44.8%, 13/29), followed by the vitreous in the anterior chamber (17.3%, 5/29). Similarly, according to the study by Agarwal *et al.* [14], the most common were peripheral anterior synechiae, 50.5% (57/113), followed by pupillary block, 17.7% (20/113), and the presence of a loose vitreous in the anterior chamber, 15.9% (18/113). In turn, according to the study by Kessing *et al.* [18] carried out on 16 eyes (patients after senile cataract surgery) with secondary glaucoma in aphakia the most – as many as 7 – eyes (44%) presented pupillary block (which in 5 eyes was associated with increasing vitreous herniation into the pupil, without rupture of the hyaloid membrane).

Sometimes, despite a thorough ophthalmological examination aimed at possible pathomechanisms, it is not possible to establish the cause of secondary glaucoma in the aphakic eye. In the study by Ekşioğlu *et al.* as many as 37.9% (11/29) of cases remained idiopathic. This leads to the search for other possible pathomechanisms, including inflammation.

On the basis of their *ex vivo* studies, Michael *et al.* [33] described changes in the trabecular meshwork cells caused by interleukin-4 and vascular endothelial growth factor (VEGF) – both secreted by the lens epithelial cells – and suggested their involvement in the pathogenesis of this type of glaucoma. However, a study by Stech *et al.* [34] revealed no differences in the prevalence of aphakic glaucoma over the 11-year follow-up period between children who underwent lensectomy with and without removal of the peripheral lens capsule. However, this study was conducted in a pediatric population that cannot be compared to adult patients. In children, there are many factors that may affect the research result, which are absent in adult patients, such as the immaturity of the angle. Therefore, it is not known what results would be provided by analogous analysis in adult patients.

There are also hypotheses suggesting the role of anterior chamber exposure to the chemical microenvironment of the posterior chamber in the pathogenesis of secondary glaucoma in aphakic patients [4, 32]. An adverse effect of vitreous excitatory amino acids on the angle is speculated, among other things [32]. However, these hypotheses are not proven [4].

Finally, the preclinical study by Kugelberg *et al.* [31] indicated a significant role of processes at the cellular level in the development of secondary glaucoma after lensectomy in animals. Researchers have demonstrated that inhibition of proliferation (with 5-fluorouracil) significantly reduces the incidence of postoperative glaucoma after lensectomy in infant rabbits. Perhaps further research in the field of cell biology and immunology will allow us to better understand the etiopathogenesis of secondary glaucoma in aphakia, especially in those patients who do not present the already known pathomechanisms.

In the case of secondary glaucoma in aphakia, its pathomechanism should always be established, as it determines further therapeutic management [19].

SECONDARY GLAUCOMA IN APHAKIA – TREATMENT

The treatment of secondary glaucoma in aphakic eyes, both pharmacological and surgical, is often more difficult than in the primary glaucoma [4, 14]. A whole range of treatments is available.

Pharmacological treatment of this type of glaucoma includes all available groups of antiglaucoma drugs [14]. However, it should be remembered that in the case of secondary angle-closure glaucoma in the aphakic eye, a careful decision should be made when choosing between mydriatics/cycloplegics and miotics, taking into account the most likely pathomechanism in a patient. Agarwal *et al.* [14] indicate in their study that in clinical practice the choice is not always obvious. Nevertheless, the pupillary block and the ciliovitreal block are typical situations in which mydriatics/cycloplegics are recommended, while miotics are contraindicated [17].

Among the methods of laser therapy, iridotomy and hyaloidotomy are used in both types of block [17, 27, 35]. Moreover, in the ciliovitreal block, the posterior capsulotomy is also effective [17, 28].

The gold standard in glaucoma surgery is trabeculectomy. However, in secondary glaucoma in aphakic patients, the success rate of trabeculectomy differs depending on the study: some show its effectiveness, others show no effectiveness [4, 36, 37]. The reasons for the failure of filtration surgery in aphakic eyes are not fully understood, but it is suggested that conjunctival scarring and biochemical changes in aqueous humor may play a role [38].

It seems, however, that glaucoma drainage devices (GDDs, glaucoma drainage implants – GDIs, aqueous shunts, setons) can be successfully used [4]. GDDs are traditionally reserved for treatment-resistant glaucoma (although their use is also more and more often suggested at earlier stages of therapy), and therefore in the case of failure of trabeculectomy or with a high risk of such failure. The high risk of trabeculectomy failure in adults is estimated, among others, in aphakic eyes, but also in eyes after surgical interventions within the conjunctiva (including previous: trabeculectomy, ECCE, *pars plana* vitrectomy, penetrating keratoplasty), with conjunctival diseases with scarring, after trauma, and also in cases of treatment-resistant neovascular glaucoma, uveitic glaucoma, or in the iridocorneal endothelial syndrome [39].

The effectiveness of GDDs in certain cases of high risk of trabeculectomy failure is demonstrated by the study by Fuller *et al.* [40]. Researchers analyzed the 10-year success rate of treatment with a Molteno implant for posttraumatic glaucoma in 38 eyes, 79% of which were aphakic or pseudophakic. Researchers report that they achieved IOP control in 76% of cases (some of them required treatment with antiglaucoma eye drops in addition to surgery).

Several GDDs are available, e.g. Molteno, Baerveldt, Ahmed. Over the last 25 years, there has been a huge evolution in the field of their sizes, shapes, biomaterials, and surgical implantation techniques. Nevertheless, all modern episcleral implants share a common basic design and mechanism of action. They consist of a silicone tube inserted into the anterior chamber (also possibly the ciliary sulcus or vitreous cavity) through a scleral fistula. From there they shunt the aqueous humor into the end-plate placed in the sub-Tenon's space at the equator of the globe, usually in the superotemporal quadrant, between two adjacent rectus muscles. The homeostatic formation of fibrous tissue around the episcleral endplate forms a reservoir in which the aqueous humor initially accumulates. Then, aqueous humor passes through the capsule of the reservoir (via passive diffusion) and is absorbed by the periocular and lymphatic capillaries [39].

One of the complications of GDDs is improper tube placement when it contacts the corneal endothelium, which can lead to corneal decompensation. It is possible to reduce the risk of this complication in some patients, including aphakic, by placing the tube in the ciliary sulcus or in the vitreous cavity [39, 41]. If the complication occurs, there are various ways to fix it. Usually, the tube is cut or repositioned, either by removing it from the original sclera fistula and placing it in the new one, or by sclera fixation or transcameral suture [42, 43].

Unfortunately, especially in aphakic eyes, there is an increased risk of another complication, i.e. blockage of the tube by the vitreous (even during the procedure during the supramid stent removal) and, as a result, filtration failure with increased IOP [39].

Another very serious postoperative complication – suprachoroidal hemorrhage – is especially common in patients with aphakia [44]. Dreyer *et al.* [45] reported two cases of suprachoroidal hemorrhage in aphakic patients after simultaneous implantation of the Ex-PRESS implant and an artificial intraocular lens. The Ex-PRESS implant is a small filtering device implanted under the scleral flap to ensure an efficient outflow of aqueous humor from the anterior chamber of the eye [46]. Dreyer *et al.* emphasized that in their practice they had performed the described procedure dozens of times in patients with various risk factors for failure, without ever encountering a suprachoroidal hemorrhage again. The researchers suggested that special care should be taken during the implantation of the Ex-PRESS implant into aphakic eyes.

In addition to GDDs for refractory glaucoma treatment, another traditionally reserved procedure is transscleral diode laser cyclophotocoagulation (TSCPC) [47]. However, studies have shown that the effectiveness of TSCPC in the treatment of glaucoma in aphakic eyes is moderate and the method is more effective in the elderly than in the young [48].

Recently, a newer version of this technique has been popular – micropulse TSCPC (MP-TSCPC) – which is indicated for the treatment of non-resistant glaucoma [47]. There are literature reports describing the treatment of glaucoma in aphakic eyes with MP-TSCPC [49, 50, 51]. Elhefney *et al.* [49] report the relative effectiveness of this new method in the treatment of various types of glaucoma in children, including glaucoma in aphakia.

It seems important that the anatomical differences of aphakic eyes widen the range of possible different therapeutic methods, such as those discussed above for GDDs, but also in the case of cyclodestructive procedures. Gonioprism-assisted diode cyclophotocoagulation (GADC) is procedure that requires sequentially: enlargement of the iris with hooks to expose the posterior chamber of the eyeball, visualization of ciliary processes by Swan-Jacob gonioprism, and then insertion of a diode laser probe through the peripheral incision of the cornea and performing cyclophotocoagulation. Therefore, an endoscopic probe is redundant in this technique, which makes it a more accessible and cheaper alternative to endoscopic cyclophotocoagulation, which has been described as possible for aphakic (but also pseudophakic) patients [52].

Nevertheless, the existence of so many different methods proves the therapeutic difficulties of secondary glaucoma in aphakic patients [4]. Therefore, there is a need for further development, both to improve the existing techniques, and to search for new alternative solutions.

SUMMARY

Among the most common pathomechanisms leading to the development of secondary glaucoma in aphakia, the following are distinguished: peripheral anterior synechiae, pupillary block, and the presence of a loose vitreous body in the anterior chamber and the angle region. In the case of secondary glaucoma in aphakic patients, its pathomechanism should always be established, because it determines further therapeutic management. Treatment includes a wide range of methods: pharmacological, laser and surgical. The success of trabeculectomy is not satisfactory [4, 36, 37]. It seems, however, that glaucoma drainage devices can be used with acceptable success. Aphakia carries a risk of various possible postoperative complications, including suprachoroidal hemorrhage.

There is still a need for more research to expand our knowledge on this topic.

DISCLOSURE

The authors declare no conflict of interest.

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