https://doi.org/10.5114/oku/213379 (Research Paper)

Received: 9.09.2025 Accepted: 22.09.2025

Silicone Oil–induced Secondary Glaucoma

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Summary: Secondary glaucoma induced by silicone oil is one of the most serious complications following vitrectomy with endotamponade. The pathogenesis of elevated intraocular pressure is multifactorial and includes both early mechanisms – such as pupillary block or migration of oil into the anterior chamber – and late mechanisms, associated with chronic inflammation, synechial closure of the drainage angle, and infiltration of the trabecular meshwork by emulsified oil droplets. Treatment includes standard hypotensive pharmacotherapy, prophylactic peripheral iridotomy to prevent pupillary block, and, in selected cases, removal of the silicone oil. In advanced cases, surgical methods are employed, such as trabeculectomy, drainage implants, or cyclophotocoagulation. Early identification of the mechanisms leading to secondary glaucoma and the implementation of an appropriate therapeutic strategy remain crucial for preserving visual function in patients after retinal surgery.

Key words: silicone oil, glaucoma, vitrectomy, intraocular pressure.

Silicone oil (SO) is a key component of intraocular endotamponade in vitreoretinal surgery, serving as a long-term substitute for the vitreous body in complex surgical cases. It is a colorless fluid composed of organosilicon polymers, primarily polydimethylsiloxane (PDMS), and is used as a vitreous substitute due to its transparency and chemical inertness.

SO is characterized by specific physicochemical properties that are crucial for surgical applications. Its density is 0.975 g/cm^3, making it lighter than water; consequently, it floats on the aqueous surface. The refractive index of SO is 1.4035, which is higher than that of the vitreous body (1.33). Its surface tension is high, measuring 40 dyn/cm^2, though lower than that between gas and water (70 dyn/cm^2). Silicone oil is chemically inert, non-toxic, and resistant to biological degradation [1, 2].

Various types of silicone oil, differing in viscosity, are used in vitreoretinal surgery. Standard oils, which are lighter than water, include silicone oil with a viscosity of 1000 centistokes (1000 cSt) – the most commonly used – and oil with a viscosity of 5000 cSt, which is more viscous and offers greater resistance to emulsification. The first generation of heavy tamponades included fluorinated silicone oil, perfluorocarbons, and partially fluorinated alkanes. The currently used second generation consists of ready-made mixtures such as Oxane HD and Densiron 68, which differ in composition and in the proportions of silicone oil and fluorinated components. Oxane HD (Bausch & Lomb, France) is a mixture of Oxane 5700 silicone oil and RMN3 (a partially hydrocarbonated fluorinated olefin), with a viscosity of approximately 3300 cSt. Densiron 68 (Fluoron Co, Germany), on the other hand, is a heavy silicone oil with a density exceeding 1 g/cm^3 and a viscosity of around 1400 cSt [3]. The presence of silicone oil inside the eye changes its optical properties because of the oil's refractive index. In a phakic eye (with the natural lens preserved), the concave anterior surface of silicone oil in contact with the lens causes a hyperopic shift in refraction, reaching up to +5.00 diopters. In contrast, in an aphakic eye (without the lens), the convex surface of the oil induces a myopic shift, which may reach as much as –5.00 diopters [4].

An important advantage of SO over intraocular gases is that its volume remains stable over time. As a result, it requires less restrictive postoperative positioning, making it a preferred choice, particularly for children or patients unable to maintain optimal postoperative positioning [5, 6].

The Silicone Study was a prospective, multicenter, randomized clinical trial that compared the efficacy of 1000 cSt silicone oil with 20% SF_6 and 14% C_3F_8 in patients with retinal detachment (RD) complicated by proliferative vitreoretinopathy (PVR). The results of the study demonstrated that, after one year of follow-up, the use of silicone oil led to significantly better anatomical outcomes and improved visual acuity compared to SF_6, while no significant differences in efficacy were observed between silicone oil and C_3F_8 [7–9].

A long-term follow-up report on this patient group showed that among individuals with maintained macular attachment after 36 months, there were no significant differences in anatomical outcomes or visual acuity between the groups treated with silicone oil, SF_6, or C_3F_8 over a maximum follow-up period of up to 6 years [10].

In turn, the Retinal Detachment Study conducted by the European Vitreoretinal Society (EVRS) was a retrospective analysis of treatment outcomes in complex retinal detachments, including cases of PVR, giant retinal tears, choroidal detachment, and macular holes. The study compared the failure rate (so-called level 1), defined as unsuccessful retinal reattachment regarded as inoperable at the end of follow-up, between patients treated with gas tamponade and those treated with silicone oil. No significant differences were observed between the groups in this study either [11].

Silicone oil has become the gold standard tamponade in the surgical management of complex retinal detachments, giant retinal tears, proliferative vitreoretinopathy, and ocular trauma.

Mechanisms of action of silicone oil in the eye:

- **intraocular tamponade:** due to its high surface tension, silicone oil effectively seals retinal defects across all its regions, regardless of their size [12],
- **space filling:** a stable oil bubble that does not mix with water limits the free movement of proliferative cells and chemical mediators within the vitreous cavity, compartmentalizing the eye in a way that may inhibit the development of proliferative vitreoretinopathy [13],
- **mechanical suppression of membrane contraction:** the oil bubble alters the direction of traction forces, aligning them parallel to the retinal surface and modifying traction vectors [12],
- **hemostasis:** silicone oil reduces the presence of blood and fibrin in the space between the retina and oil bubbles, dimini-

ISSN 1505-2753

shing proliferative activity and thereby limiting iris neovascularization in the course of proliferative diabetic retinopathy [12],
- **prevention of ocular atrophy:** silicone oil slows the progression of globe atrophy and helps maintain ocular volume [12].

Ocular hypertension and secondary glaucoma following silicone oil administration

An increase in intraocular pressure may occur at any time following surgery involving silicone oil – ranging from mild and transient to severe and persistent, potentially leading to vision loss. An increase in intraocular pressure (IOP) may result from various pathogenic mechanisms. Two clinical entities are distinguished: early postoperative ocular hypertension and late-onset glaucoma.

Early intraocular hypertension following vitrectomy with silicone oil tamponade may result from either open-angle or angle-closure mechanisms, and is most often secondary in nature. However, the possibility of undiagnosed preoperative primary open-angle glaucoma (POAG) or exacerbation of pre-existing intraocular hypertension should also be considered.

- Early postoperative elevation of intraocular pressure may also result from: pupillary block, inflammatory response, reaction to postoperative steroid therapy, and migration of silicone oil into the anterior chamber, which causes mechanical obstruction of aqueous outflow – either with or without pupillary block. In cases of pupillary block, aqueous humor accumulates behind the iris diaphragm, in the most gravity-dependent portion of the posterior segment of the eye. The pressure increase in this area forces silicone oil through the pupil into the anterior chamber.
- In cases of late-onset glaucoma, the described mechanisms of development include trabecular meshwork infiltration by silicone oil droplets, chronic inflammation, angle closure due to synechiae, iris neovascularization, migration of emulsified or non-emulsified silicone oil into the anterior chamber, and idiopathic open-angle glaucoma.

For diagnosing previously undetected POAG, useful indicators can include the patient's medical history, normal anterior chamber depth, open drainage angle, optic nerve head appearance, and assessment of the fellow eye.

The incidence of glaucoma following silicone oil use and the risk factors for elevated intraocular pressure are difficult to determine conclusively. Publications from the 1970s and 1980s suggested a higher risk of glaucoma development, whereas more recent reports indicate a slightly lower frequency of this complication, which may be associated with advances in surgical techniques and changes in therapeutic approaches [14]. Prognostic factors that could reliably predict the risk of intraocular pressure elevation remain unclear, although numerous authors have attempted to identify them. It has been shown, among other findings, that patients with pre-existing glaucoma are more prone to pressure-related complications after surgery, although not all analyses confirm this association. In the case of diabetes, study results are also inconclusive. Some authors found no correlation between diabetes and pressure disturbances, while others reported that patients with both diabetes and aphakia more frequently experience postoperative pressure elevation [15, 16]. It has also been demonstrated that patients with retinal detachment due to proliferative diabetic retinopathy are at higher risk of elevated intraocular pressure than those with detachment associated with proliferative vitreoretinopathy [17]. Both the presence of PVR prior to surgery and its postoperative development are risk factors for unfavorable anatomical outcomes and a higher incidence of hypotony [18]. It is worth noting that myopia is a shared risk factor for both retinal detachment and primary open-angle glaucoma [19].

The management of acute ocular hypertension following silicone oil administration depends on the clinical presentation. The mechanisms underlying early elevation of intraocular pressure are varied and not fully understood.

In all cases of ocular hypertension, prompt topical and/ or oral pharmacological treatment is required to lower IOP.

Pupillary block

Silicone oil is lighter than aqueous humor and can mechanically obstruct the pupil, preventing fluid from flowing into the anterior chamber. In such cases, pressure builds up in the posterior segment of the eye, causing the iris to shift forward. This leads to closure of the drainage angle, preventing aqueous humor from flowing through the trabecular meshwork and resulting in a further increase in intraocular pressure. Pupillary block leading to secondary angle closure occurs more frequently in aphakic eyes, although it has also been reported in phakic and pseudophakic eyes, which may be due to zonular weakness or iatrogenic injury during surgery [20, 21]. An important mechanism also involves the migration of silicone oil into the anterior chamber, resulting in mechanical obstruction of aqueous outflow. Contact between SO and a pre-damaged drainage angle may trigger an inflammatory response, initially leading to ocular hypertension, and over time progressing to fully symptomatic glaucoma. Additionally, SO induces a foreign body-like reaction – silicone oil microdroplets are phagocytosed by macrophages within the trabecular meshwork [22]. If the drainage angle becomes closed, IOP can rise to very high levels within a short time. In eyes with extensive synechial closure of the drainage angle, removal of SO alone rarely restores normal pressure. In such cases, glaucoma surgery should be considered – either before, concurrently with, or after PDMS extraction – along with simultaneous iris reconstruction to reopen the drainage angle [23].

A silicone oil droplet larger than the pupil diameter that migrates into the anterior chamber may lead to acute pupillary block and obstruction of aqueous outflow, particularly in patients who remain in a supine position for prolonged periods. In such cases, aqueous humor accumulating behind the iris may force SO through the pupil into the anterior chamber. This occurs more frequently in aphakic patients, particularly when a six o'clock iridectomy (so-called Ando iridectomy) has been improperly performed or has closed due to a fibrin reaction. Careful placement of the iridotomy at the lower edge of the SO meniscus is crucial for ensuring both safety and efficacy. Prophylactic basal iridectomy at the six o'clock position should be routinely performed in aphakic eyes, as it allows aqueous humor to flow into the anterior chamber and prevents pupillary block. An iridectomy opening with a diameter of 15 micrometers may be sufficient to prevent angle-closure glaucoma caused by pupillary block; however, in clinical practice, a diameter of at least 150–200 micrometers is recommended to effectively prevent acute cases [24]. An excessively large iridectomy may promote anterior migration of silicone oil [25]. However, the rate of postoperative closure of the Ando iridectomy has been reported to reach up to one-third of cases [26]. Silicone oil acts as a scaffold that promotes membrane formation behind the iris. Additionally, its presence in the anterior chamber may trap remnants of iris tissue, blood, and fibrin within the iridotomy, facilitating fibrin clot formation [27]. Furthermore, the formation of posterior synechiae may lead to pupillary block. If oil removal is not feasible, reopening the iridectomy with an Nd: YAG laser is recommended; however, if the procedure is unsuccessful, a more extensive surgical iridectomy may be required [28]. In patients with persistent or recurrent iridotomy occlusion due to relapsing inflammation, adjunctive treatment may include sub-conjunctival or sub-Tenon steroid injections, as well as intracameral tPA administration [27]. In cases involving heavy tamponade agents, prophylactic peripheral iridectomy at the twelve o'clock position is recommended [20, 29]. It should be noted that heavy silicone oil can induce a stronger inflammatory response, likely due to its greater tendency to emulsify compared to polydimethylsiloxane (PDMS)[30]. In parallel with mechanical procedures, treatment should include medications that inhibit aqueous humor production and intensive anti-inflammatory therapy. Additionally, a larger pupil reduces the risk of recurrent block – hence the recommendation to use mydriatic agents. If pharmacological treatment fails to lower the pressure, PDMS removal should be considered, provided the risk of retinal redetachment is low. Adopting a face-down position may promote posterior displacement of SO and facilitate aqueous humor flow into the anterior chamber. However, in cases of pupillary block mechanism, IOP typically rises sharply, and pharmacological treatment alone, as well as face-down positioning, are not sufficiently effective [31].

Another cause of early postoperative IOP elevation is overfilling with PDMS during surgery; however, this is usually easy to recognize and manage by an experienced surgeon.

Response to postoperative steroid therapy

The incidence of steroid-induced glaucoma (SIG) among all forms of secondary glaucoma is not precisely defined; however, it is known that the population's response to glucocorticosteroid administration varies. It is estimated that approximately 61–63% of healthy individuals do not exhibit a significant increase in intraocular pressure, showing a rise of less than 5 mmHg, whereas 33% of the general population demonstrate a moderate response, with pressure increasing by 6–15 mmHg. Only 4–6% of healthy individuals belong to the group of strong responders, in whom IOP increases by more than 15 mmHg. In contrast to the general population, as many as 46–92% of patients with POAG exhibit a significant and potentially dangerous rise in IOP following topical steroid administration. This highlights that patients with POAG represent a group at particular risk of developing steroid-induced glaucoma and require special caution when selecting anti-inflammatory therapy.

Ocular hypertension or SIG occurs considerably more frequently after topical than systemic administration, with as many as three quarters of SIG cases resulting from topical steroid use.

Older-generation corticosteroids (e.g. dexamethasone, prednisolone, betamethasone, fluorometholone) tend to elevate IOP more frequently and more significantly compared to newer formulations (loteprednol, difluprednate, rimexolone). IOP elevation induced by older steroids may range from 6 mmHg to as high as 22 mmHg and occurs ten times more frequently in patients treated with dexamethasone than in those receiving fluorometholone. To reduce the risk of adverse effects, newer steroids such as rimexolone, loteprednol etabonate, and difluprednate have been developed. Loteprednol causes a clinically significant rise in IOP in only a small proportion of patients (1.7–2.1%), and the onset of hypertension is generally slower than with dexamethasone or prednisolone. Difluprednate, on the other hand – despite a relatively low incidence of IOP elevation (3%) – can cause a markedly greater pressure increase than other newer steroids. Following the discontinuation of steroid eye drops, the hypertensive effect may persist for up to 18 months, necessitating continued hypotensive therapy during this period [32].

Emulsification of silicone oil

The behavior of silicone oil within the eye and its potential to cause complications are largely determined by its physicochemical properties. Emulsification is a process in which a uniform SO globule breaks down into smaller, non-coalescing bubbles. Contributing factors include the quality of the oil used, shear forces, and vibrations caused by ocular movements. It is commonly believed that the extent of emulsification is often underestimated, as most droplets measure less than 2 μm and remain invisible during standard slit-lamp examination and gonioscopy. When emulsification is detected in the anterior chamber, it is highly likely that the process has already occurred in the posterior chamber [33]. This may explain cases in which IOP increases despite the absence of visible emulsification. However, the effect of these microscopic droplets on IOP remains a matter of debate, especially since emulsification in the anterior chamber is sometimes observed without an accompanying rise in pressure.

Emulsified SO droplets may migrate into the trabecular meshwork, triggering inflammation and reducing aqueous outflow, ultimately leading to the development of secondary open-angle glaucoma. The tendency toward emulsification depends primarily on the purity of the preparation and its viscosity, which in turn correlates with the polymer's molecular weight. Some studies have suggested that SO with a viscosity of 1000 cSt is more likely to cause an increase in IOP than preparations with a viscosity of 5000 cSt. However, other authors have not confirmed this relationship [34, 35]. Clinically, signs of emulsification can be observed with a slit-lamp in nearly all patients in whom SO remains in the eye for one year [36]. It has been noted that the emulsification process also depends on the volume of chamber fill with oil and the effects of the encircling scleral buckle. With greater intraocular fill, the contact surface between the oil and aqueous humor increases, but at the same time, the mobility of both layers is reduced, which stabilizes the silicone oil bubble and decreases the shear forces responsible for the process of emulsification. A similar stabilizing effect can be achieved with the use of a scleral buckle [37]. However, some studies suggest that there is little to no correlation between the presence of emulsified SO in the anterior chamber and the development of glaucoma. According to the authors of these studies, elevated IOP may be unrelated to oil emulsification and could instead result from other pathogenic mechanisms [38].

Heavy tamponade agents, characterized by a density greater than that of water, are used particularly in cases of retinal tears located in the inferior regions of the retina, especially in the presence of PVR. Earlier studies had already demonstrated that their use is associated with a risk of increased intraocular pressure, with the incidence varying depending on the type of agent used, the duration of follow-up, and the diagnostic criteria applied. The incidence of IOP elevation following the administration of silicone oil or heavy tamponade agents ranged from a few to several dozen percent across various studies, with particularly high values observed in long-term follow-ups [39–41].

It should be noted, however, that elevated intraocular pressure following the use of heavy tamponades may manifest in various forms. Acute pupillary block leading to a rapid rise in IOP has been reported, particularly in cases of excessive chamber fill with a mixture of F6H8 and silicone oil. In such situations, IOP elevation was observed in as many as 31–39% of patients [42]. In studies involving Oxane HD, the incidence of pressure elevation ranged from 14% to 18%, while transient pressure spikes occurred in up to 42% of cases [43, 44]. For Densiron 68, IOP elevation was noted in 8–19% of patients, with a frequency comparable to that observed following the use of conventional silicone oil [40, 45, 46].

Role of inflammation

Prolonged contact of silicone oil with the trabecular meshwork may trigger a delayed type IV hypersensitivity reaction, leading to an influx of inflammatory cells, particularly macrophages [47].

It as been demonstrated that patients with silicone oil tamponade exhibit elevated levels of inflammatory mediators in the aqueous humor, including Interleukin-17 (IL-17), Interleukin-6 (IL-6), and tumor necrosis factor-α (TNF-α) [48].

Treatment of ocular hypertension and secondary glaucoma

In cases of elevated IOP caused by the presence of silicone oil, the primary approach is pharmacological management aimed at lowering the pressure. Treatment is usually based on medications that inhibit aqueous humor secretion. Their effectiveness can be enhanced by combining them with topical anti-inflammatory agents. In most cases, this therapy enables effective pressure control both in eyes filled with SO and after its removal. Prostaglandin analogs have proven effective and safe in the treatment of silicone oil-induced glaucoma, showing no significant difference in inflammation control compared to commonly used timolol [49].

Removal of silicone oil and intraocular pressure control

If pharmacological treatment proves ineffective, the first option to consider is the removal of silicone oil. Prolonged exposure of the trabecular meshwork to emulsified silicone oil particles leads to structural organic changes including fibrosis and meshwork collapse. In advanced stages of this pathology, removal of the oil alone may not be sufficient to normalize IOP. Early removal of emulsified silicone oil is crucial, as it may reverse mechanical blockage and limit progressive damage to the aqueous outflow system, thereby promoting better IOP control. Removal of silicone oil, either as a standalone procedure or in combination with glaucoma surgery, is aimed at lowering IOP; however, it carries a potential risk of retinal detachment. Thorough removal of emulsified oil is crucial to minimize the inflammatory response. Repeated fluid–air exchanges in the vitreous cavity and careful aspiration of silicone oil droplets from the anterior chamber are essential to minimize the risk of residual oil remaining in the eye.

Clinical studies on the effectiveness of silicone oil removal are inconclusive, likely due to the diversity of clinical observations. For example, Budenz et al., in their retrospective analysis, demonstrated that the effectiveness of the procedure in IOP control gradually declined from 69% at 6 months to 48% at 36 months. They observed that patients who underwent silicone oil removal alone were more likely to experience persistent ocular hypertension and required additional surgeries, whereas those who simultaneously underwent glaucoma surgery were more prone to developing hypotony [50]. Conversely, Jonas et al. reported markedly better outcomes, observing IOP normalization in 93.4% of patients following oil removal. The authors concluded that a clinically significant increase in IOP after vitrectomy with silicone oil tamponade occurs relatively infrequently. In most cases, it can be effectively controlled with pressure-lowering medications. Therefore, in patients with elevated IOP and silicone oil tamponade, oil removal may be a preferable alternative to invasive glaucoma surgery [51]. These results are in conflict with the observations of Flaxel et al., who noted persistently elevated pressure in all studied eyes [52].

The increase in IOP after silicone oil removal is caused by several pathophysiological mechanisms. The first of these is swelling of the trabecular meshwork, resulting from a postoperative inflammatory response. The second mechanism is the mechanical breakdown of oil droplets into smaller particles under the influence of irrigation fluid. Smaller droplets show an increased tendency to obstruct aqueous outflow channels within the trabecular meshwork, as confirmed by histopathological studies revealing that both emulsified oil droplets and the macrophages that phagocytose them can physically clog the meshwork [53].

Glaucoma surgery following silicone oil tamponade

Surgical treatment of glaucoma secondary to silicone oil tamponade is complex and requires an individualized approach. In cases where the drainage angle has become completely occluded by synechiae, silicone oil removal alone will not lead to IOP normalization. In such situations, surgical intervention for glaucoma is usually necessary. The decision to remove the oil simultaneously should be made after assessing the risk of retinal redetachment associated with tamponade removal. If either emulsified or non-emulsified oil is mechanically obstructing the trabecular meshwork and the retina remains stable, oil removal as a standalone procedure may be sufficient.

Conventional surgical methods

In cases where pharmacological treatment and SO removal prove insufficient, particularly in the presence of extensive synechial angle closure, surgical intervention becomes necessary. The choice of surgical method should be individually tailored, taking into account the patient's visual function, gonioscopic findings, IOP, and conjunctival condition. It should be noted, however, that filtration surgery in post-vitrectomy patients is associated with a higher rate of failure and complications. Surgical methods used in the treatment of glaucoma associated with SO include trabeculectomy, the Ex-PRESS mini-shunt, deep sclerectomy, and glaucoma drainage devices (GDDs). There are no reports confirming the efficacy of minimally invasive glaucoma procedures in this context. Conventional filtering surgeries, such as trabeculectomy, have limited applicability and a poorer prognosis in patients who have undergone vitrectomy with silicone oil tamponade. This is due to technical challenges associated with conjunctival scarring following prior vitreoretinal surgery [54]. A prospective study compared the efficacy of four surgical methods: trabeculectomy, deep sclerectomy, Ahmed valve implantation, and the Ex-PRESS mini-shunt in the treatment of persistent glaucoma after SO removal. The highest rate of complete success was achieved with the Ex-PRESS mini-shunt [55].

The main cause of the higher failure rate of filtration procedures is believed to be the inflammatory response and pronounced fibrosis triggered by the migration of SO droplets beneath the conjunctiva.

Retrospective studies on cyclodiode therapy have shown good efficacy, but also a high rate of required reinterventions – over 50% of patients needed a second procedure within one year of follow-up. The most serious complication of these methods remains chronic hypotony. Transscleral diode treatment carries a lower risk of hypotony compared to cryotherapy, although precise dosing of the laser effect remains challenging. The advantage of endoscopic cyclophotocoagulation lies in its ability to precisely target the ciliary body, allowing for more controlled treatment. This method has demonstrated better outcomes compared to the Ahmed drainage implant in the treatment of refractory glaucoma.

Drainage implants

Chronically elevated intraocular pressure persists in a minority of patients (11%) after silicone oil tamponade. In most cases, this condition can be effectively managed with antiglaucoma medications. For patients who do not respond to pharmacological therapy, glaucoma drainage devices represent an effective surgical option and may be considered either as a first-line approach or following the failure of previous procedures. Nevertheless, surgical prognosis remains poorer than in cases of primary glaucoma.

These devices are implanted in the inferior quadrant of the eye, which minimizes the risk of silicone oil migration into the

 ISSN 1505-2753

drainage system. The efficacy of this method was confirmed in a study conducted by Al Jazzaf et al. In their analysis, implantation of the Ahmed valve in the inferotemporal quadrant resulted in a success rate of 86% at six months and 76% at one year. These findings suggest that drainage implants are a valuable tool in the management of glaucoma associated with silicone oil tamponade [38]. An alternative approach is the implantation of a drainage device into the pars plana, which offers comparable efficacy and is associated with fewer complications compared to the conventional placement in the anterior chamber, particularly in patients with corneal conditions [56].

Transscleral cyclophotocoagulation in the treatment of silicone oil–induced secondary glaucoma

In cases of refractory glaucoma, cyclodestructive procedures such as cyclocryotherapy or diode laser cyclophotocoagulation are employed to lower intraocular pressure.

Transscleral cyclophotocoagulation is a therapeutic procedure used in the management of secondary glaucoma. According to published data this method provides effective IOP control in 44% to 82% of patients [57–59]. Unfortunately, the effect is not permanent, as more than 50% of patients required a second procedure within one year of follow-up [60, 61].

Due to the risk of vision loss, this method is reserved for eyes with limited visual potential. Nevertheless, compared to cyclocryotherapy, cyclophotocoagulation is associated with a lower rate of complications [62]. An advantage of endoscopic cyclophotocoagulation is the ability to directly target the ciliary body, which provides greater precision and, additionally, higher efficacy compared to the Ahmed valve [63, 64].

Selective laser trabeculoplasty

Selective laser trabeculoplasty (SLT) may serve as an adjunctive therapy and can be effective in lowering intraocular pressure in patients with secondary open-angle glaucoma induced by the presence of silicone oil, particularly when disease control has not been achieved despite maximal pharmacological treatment [65, 66].

Conclusions

Secondary glaucoma associated with silicone oil remains one of the major complications following vitrectomy with endotamponade. The pathogenesis of elevated intraocular pressure is multifactorial, involving both early and late mechanisms. In the early postoperative period, pressure elevation is typically caused by pupillary block, inflammatory response, pre-existing glaucoma, and mechanical obstruction of aqueous outflow due to silicone oil migration into the anterior chamber. Late mechanisms include chronic inflammation, synechial angle closure, iris neovascularization, and infiltration of the trabecular meshwork by silicone oil droplets – both emulsified and non-emulsified. The coexistence of primary open-angle glaucoma cannot be excluded either.

Management of secondary glaucoma following silicone oil tamponade requires an individualized and multi-stage approach. Pharmacological treatment involves standard hypotensive agents, though these are often insufficient. An important preventive measure against pupillary block is peripheral iridectomy – most commonly in the inferior part of the iris – which allows free flow of aqueous humor and reduces the risk of oil displacement into the anterior chamber. In selected cases, removal of silicone oil is necessary, as it may help limit the progression of complications, especially when the oil remains in the eye for an extended period. In advanced stages or when conservative treatment fails, surgical interventions – such as trabeculectomy, drainage implants, and transscleral cyclophotocoagulation – are required.

Understanding the mechanisms leading to increased intraocular pressure and implementing appropriate prophylactic and therapeutic strategies are crucial for improving ophthalmic outcomes and preserving visual function in patients treated with silicone oil.

Disclosure
Conflict of interests: none declared
Funding: no external funding
Ethics approval: Not applicable.

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