

# Silicone oil induced glaucoma: A review

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## Abstract

**Background** Silicone oil has been an important adjunct for internal tamponade in the treatment of complicated retinal detachment for the past 4 decades. A known complication of its use has been the development of secondary glaucoma. **Methods** This article reviews the current body of literature documenting the different pathogeneses, predisposing factors and management of silicone oil induced pressure elevation and optic neuropathy. Categorization is clarified for the different types of secondary glaucomas due to silicone oil.

**Results** Four different mechanisms have been proposed for the pathogenesis of glaucoma that require different therapeutic strategies: (1) overfill with total anterior chamber fill leads to an open-angle glaucoma due to mechanical obstruction of outflow, (2) pupillary block with silicone oil incites angle closure glaucoma, (3) denaturation of silicone oil into microdroplets may sweep into the trabecular meshwork with the development of secondary open angle glaucoma, or (4) finally, inflammation or exacerbation of pre-existing glaucoma.

**Conclusion** Understanding the risk factors and the pathogenesis of secondary glaucoma when using silicone oil helps guide the timely and appropriate course of treatment.

**Keywords** Silicone oil · Glaucoma · Emulsification

**Conflict of interests** The authors have no conflict of interests or financial disclosures.

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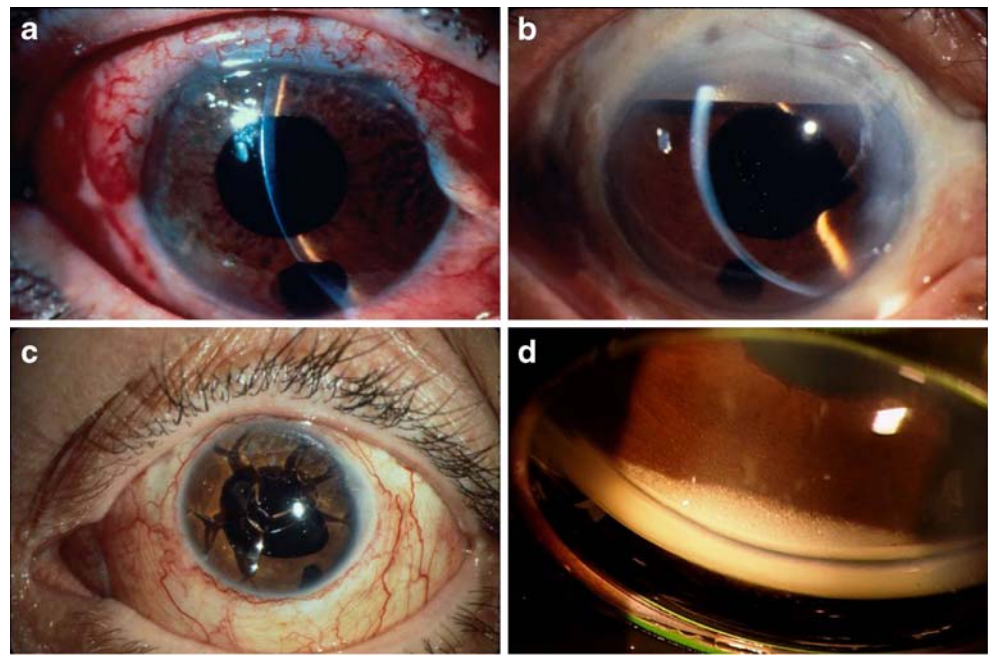
## Introduction

Silicone oil (polydimethylsiloxane) is a linear synthetic polymer made of repetitive Si-O units and is chemically similar to silicone rubber, except that silicone oil polymer chains are not crosslinked and are shorter than those of silicone rubber [1]. Silicone oil was introduced by Cibis for vitreoretinal surgery [2]. It has been shown to be beneficial especially in eyes with marked proliferative vitreoretinopathy after pars plana vitrectomy (PPV) with intraocular gas tamponade, in traumatic hemophthalmos with secondary retinal detachment, and in eyes with advanced proliferative diabetic retinopathy with widespread tractional retinal detachment. It is used for intraocular tamponade owing to its buoyant force and high surface tension. It has been associated with complications including cataract, keratopathy, anterior chamber oil emulsification and glaucoma [3–12]. Secondary glaucoma can occur at any time in the post-operative period, and may range from mild and transient [10, 13] to severe and sustained intraocular pressure (IOP) spikes, resulting in loss of vision [14]. There are four different pathogeneses for intraocular pressure elevation with silicone oil that may require different therapies.

## Pathogenesis

Glaucoma may develop after PPV, scleral buckling and endophotocoagulation, with or without the use of silicone oil [15, 16]. However, several mechanisms of secondary glaucoma following the use of silicone oil with retinal surgery have been described. An early postoperative rise in intraocular pressure can be due to: (1) pupillary block (Fig. 1a,b) [5–7, 14, 17–24], (2) inflammation [18, 25], (3) pre-existing glaucoma, and/or (4) migration of silicone oil

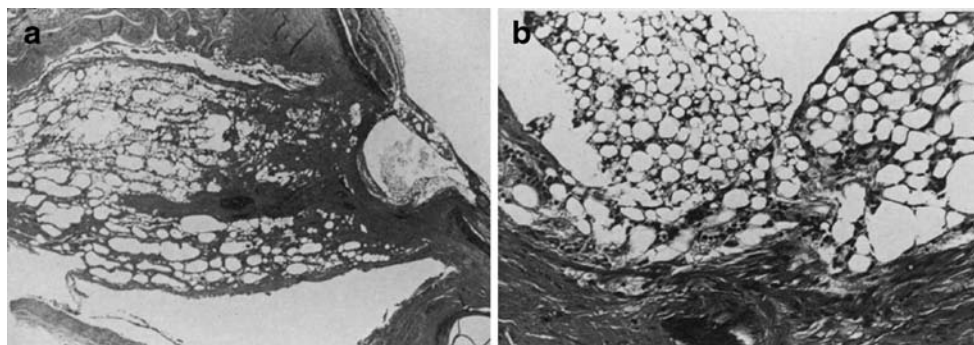
**Fig. 1** Mechanisms of secondary glaucoma following the silicone oil tamponade. **a** Slit-lamp photograph showing silicone oil leading to pupillary block (closed angle). **b** Slitlamp photograph showing mechanical obstruction due to silicone oil filling the total anterior chamber (open angle), with associated keratopathy superiorly. **c** Emulsified silicone oil in anterior chamber. **d** Goniophotograph of emulsified silicone oil causing mechanical impediment to aqueous outflow



into the anterior chamber (Fig. 1c,d) with consequent mechanical impediment to filtration. In the presence of pupillary block, the aqueous accumulates behind the iris diaphragm at the gravitationally lowest part of the posterior segment. Aqueous pressure build-up in the posterior segment forces silicone oil through the pupil into the anterior chamber.

For intermediate- and late-onset glaucoma, possible mechanisms include: (1) infiltration of the trabecular meshwork by silicone bubbles [26], (2) chronic inflammation [18], (3) synechial angle closure, [5, 20, 22, 25], (4) rubeosis iridis [17, 19, 24, 25, 27], (5) migration of emulsified and nonemulsified silicone oil into the anterior chamber [5, 9, 14, 19, 20, 22, 24, 25, 27–30], or (6) idiopathic open-angle glaucoma [5, 22, 24]. The relative contribution of each of these factors in the pathogenesis of glaucoma is unclear.

**Silicone oil neuropathy** In addition to glaucomatous pressure-dependent optic neuropathy, there may also be a direct toxic effect of silicone oil on the optic nerve. Silicone oil can be found in the optic nerve as early as 1 month after instillation in eyes with elevated intraocular pressure (pseudocavernous optic atrophy (Fig. 2a, b). Silicone oil may enter the optic nerve after longstanding silicone oil endotamponade and elevated intraocular pressure [26, 31–33]. The mechanism may be similar to the migration of acid mucopolysaccharides from the vitreous into the optic nerve in nonvitrectomized eyes with markedly elevated intraocular pressure (Schnabel cavernous optic nerve atrophy). Silicone oil may replace up to 40% of the cross-sectional area of the retrolaminar optic nerve, and may penetrate deep into it [34]. The question of damage to the contralateral optic nerve arises, if silicone oil reaches the optic chiasm.



**Fig. 2 a** The optic disc is deeply cupped. Behind the posteriorly bowed lamina cribrosa sclerae, numerous silicone vacuoles, which coalesce focally, forming cavernous spaces, totally replace the parenchyma of the optic nerve (hematoxylin-eosin, original magnifi-

cation  $\times 5$ ). **b** Mass of macrophages laden with silicone oil rests on floor of deeply excavated optic disc (hematoxylin-eosin, original magnification  $\times 50$ ) [31]

Granulomatous inflammatory reactions surrounding retrolaminar silicone oil may add to the optic nerve damage in some eyes (Fig. 3a, b) [35].

### Incidence and risk factors for elevated intraocular pressure

The true incidence of glaucoma after silicone oil injection is difficult to ascertain from the literature (Table 1). First reported by Cibis [36], the rates of intraocular pressure range from 2.2% in 6 months [13] to 56% in 8 months [25]. Reports in the 1970s and 1980s [5–7, 25, 26, 37, 39, 40]

suggested a higher rate of development of glaucoma, while recent reports [9, 10, 40, 41] have documented a somewhat lower prevalence due to several recent developments and change in concepts [10].

The prognostic factors predictive for IOP elevation remain unclear. Several studies have evaluated several risk factors for developing an elevated IOP after silicone oil injection.

#### Pre-existing pressure problems

Nguyen et al. [9] found that eyes with pre-existing glaucoma were more likely to have postoperative pressure problems, while Burk et al. [17] found no correlation with a history of glaucoma.

#### Diabetes mellitus

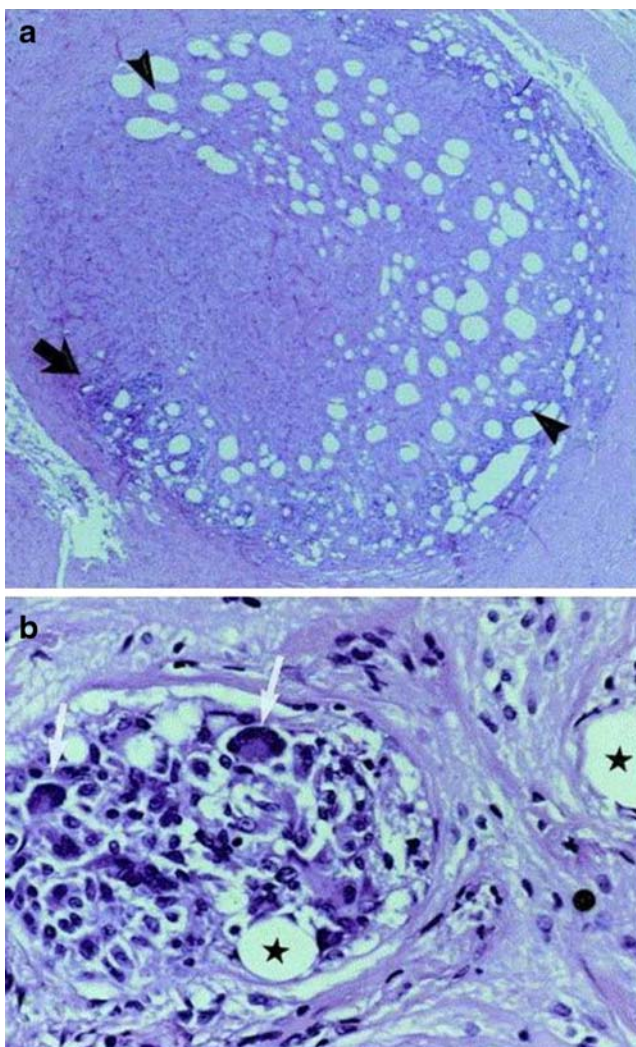
de Corral et al. [25] found that diabetes mellitus was not associated with pressure problems, but Ando [42] found that diabetic patients with aphakia were likely to have a postoperative pressure rise. Henderer et al. found that diabetic patients with isolated proliferative diabetic retinopathy (PDR) detachments had a higher risk of an elevated IOP than those with isolated proliferative vitreoretinopathy (PVR) detachments [20]. Pre-operative and post-operative PVR has been shown to be a risk factor for poor anatomic outcome and a higher incidence of hypotony [43].

#### Silicone oil in the anterior chamber

Emulsification of the silicone oil in the microglobules has been associated with a rise in the IOP [13, 18, 29, 44, 45]. It has been hypothesised that the microglobules mechanically obstruct the trabecular meshwork or are toxic to the outflow apparatus. However, normal pressures have been documented, despite the presence of oil globules in the angle [7, 9, 13, 46, 47]. It has also been observed that glaucoma can develop prior to emulsification of silicone oil [4].

#### Physiochemical properties of silicone oil

Emulsified silicone oil droplets which migrate to the trabecular meshwork may cause inflammation of the trabecular meshwork, leading to decreased outflow facility and secondary open-angle glaucoma. The propensity of emulsification is affected by the purity and the viscosity of the silicone oil. Viscosity ratings roughly correlate with the molecular weight composition of the polymeric oil (1000 cps=15000 molecular weight; 5000 cps=30000 molecular weight). Low molecular weight contaminants are more common in less viscous commercial preparations. These have a lower vapourisation pressure, which may



**Fig. 3** **a** Retrolaminar cross section of an optic nerve. The eye was enucleated 4 years after instillation of silicone oil. Note silicone oil vacuoles of different sizes (*arrowheads*). The *arrow* denotes location of image on bottom (hematoxylin–eosin,  $\times 50$ ). **b** Detail from image 4a. Note multinucleated giant cells (*white arrows*), epithelioid cells, and silicone oil vacuoles (*stars*) ( $\times 400$ ) [34]

**Table 1** Incidence of intraocular pressure elevation after injection of silicone oil and heavy tamponade agents

Author/year	Reference	No. of eyes	IOP criteria	Follow-up	Incidence (%)
Leaver et al./1979	5	93	>22 mmHg	1–4 years	15
Billington et al./1986	39	53	>22 mmHg	6 mths–1.5 years	29
Chan and Okun/1986	28	407	Not stated	3 mths–23 years	16.8
de Corral et al./1987	25	48	>10 mmHg over preoperative IOP	3–8 mths	56
Laqua et al. /1987	29	500	Not stated	-	11
Burk et al./1988	17	100	Not stated	1 year	50
Federman and Schubert/1988	7	150	Not stated	6 mths–5 years	10
Punnonen et al./1989	27	25	>25 mmHg	1–4 years	48
Riedel et al./1990	18	415	>30 mmHg	6 mths–2.5 years	13
Nguyen et al./1992	9	50	>25 mmHg and >10 mmHg over preoperative IOP	2 mths–4 years	48
Valone and Mc Carthy/1994	40	48	>21 mmHg	>1 year	23
Barr et al./1999	10	120	>25 mmHg	6 mths–3 years	8
Fisk and Cairns/1999	41	127	Not stated	3 years	43
Honavar et al./1999	19	150	>25 mmHg and > 10 mmHg over preoperative IOP	>6 mthszaaf et al	40
Henderer et al./1999	20	532	>25 mmHg or glaucoma surgery	1–5 years	29.3
Flaxel et al./2000	89	62	Not stated	1 year	18
Jonas et al./2001	24	198	>21 mmHg	3 mths–5.5 years	20
La Heij et al./2001	45	58	>25 mmHg or > 20 mmHg with anti-glaucoma medication	3 mths–2 years	12
Wolf S/2003	59	33	>21 mmHg	12–16 months	18
Theelen T/2004	60	19	>21 mmHg	3 months	0
Tognetto D/2005	63	26	-	1 year	30.7
Al-Jazaaf et al./2005	73	450	>21 mmHg	1 year	11
Rizzo S/2007	57	32	>21 mmHg	6 months	3.1

facilitate the migration of oil molecules into tissues at physiological temperatures [49]. Petersen and Ritzau-Tondrow [49] found that 1000 centistoke of oil was more likely than 5000 centistoke of oil to cause an elevated IOP, but Stinson and Small [50] found no demonstrable difference. Clinically, some degree of emulsification may be detected at the slit lamp in up to 100% of eyes with silicone oil left in place for 1 year [7].

DJ deSilva et al. suggested two important factors in the process of emulsification of silicone oil: the amount of the silicone oil fill in the eye chamber, and the effect of indentation from the encircling band. It follows that with an increased silicone oil fill, the total area of contact between silicone oil and the surrounding aqueous layer is increased. With an increased silicone oil fill, the increased size of the silicone oil bubble results in reduced movement of the silicone oil and aqueous layers. This results in an increased stability, a reduction of shearing forces, and hence oil emulsification [51]. Encircling scleral buckle procedure induces a geometric alteration in the anatomy of the eye, and hence leads to reduced emulsification by reducing silicone oil/aqueous movement and resulting shearing

forces [51]. Silicone oil emulsification may develop earlier than expected in patients with nystagmus who undergo pars plana vitrectomy combined with silicone oil injection [52].

#### Cytomegalovirus (CMV) retinitis

Among patients with CMV retinitis, an elevated IOP after silicone oil injection has been shown to be rare [53]. Stinson and Small also found a low incidence of 0% at 6 months and 5.9% at 1 year in their patients with CMV retinitis (all of the patients with CMV retinitis in this study were positive for the human immunodeficiency virus [HIV]) [50]. Low incidence of intraocular pressure rise in patients with CMV retinitis can be possibly explained by the diminished inflammatory response due to compromised immune system of HIV-positive patients [53].

#### Heavy tamponades

Various heavy tamponade agents with a specific gravity higher than water have been tried in cases of retinal breaks situated in the inferior retina, especially in the presence of PVR.

Fluorinated silicone oil [54], perfluorocarbon liquids [55] and partially fluorinated alkanes [56] formed the first generation of heavy tamponades. The second generation of heavy tamponades currently consists of three different prefabricated mixtures: Oxane HD, Densiron 68, and HWS 46-3000 [57]. The substances differ in their silicone oil and PFA components. Oxane HD (Bausch & Lomb, Toulouse, France) is a mixture of silicone oil (Oxane 5700; Bausch & Lomb) and a mixed fluorinated and hydrocarbonated olefin (RMN3) [58]. Significant rates of emulsification and a rise in IOP have been noticed with Oxane HD [59, 60]. Densiron 68 (Fluoron Co, Neu-Ulm, Germany) is a solution of perfluorohexyloctane (F6H8) and 5000 cSt silicone oil. In some series, heavy-oil emulsification has been reported to be low, with little or no clinical impact [59, 61]. However, Sandner and Engelmann reported emulsification of Densiron in seven out of 48 patients with pseudo-hypopyon formation in four patients [62]. Emulsification, when it does occur, is likely to lead to partitioning of heavy oils into their component parts, and increases the likelihood of oil-related problems. HWS 46-3000 is the latest, heaviest and most viscous of the three new tamponades; a clinically significant emulsification has not been observed in the first clinical application of HWS 46-3000 when left in situ for 1–3 months, and the initial report, with low complication and high success rates, are encouraging [57]. Acute pupillary block type of elevation of the IOP has been noted with various heavy tamponades. Tognetto et al. reported three cases of IOP rise caused by an overfilling with a mixture of F6H8 and silicone oil [63]. A mixture of silicone oil and F6H8 was associated with an IOP rise in 31–39% of cases [58, 64]. In larger series with a longer follow-up after Oxane HD, a rise in IOP could be seen in 14–18% [59, 64]. Short-term elevations in IOP were reported by Cheung et al. [65] in 42%; in another series, no significant IOP rise was noted following Oxane HD application [60]. A higher rate of rise in IOP could be noted with Densiron 68 in 8–19% [61, 63]. Intraocular pressure elevation seemed comparable to the ones seen with conventional silicone oil [66].

#### Duration of silicone oil tamponade

Gonvers and Andenmatten [67] reported that the increase in IOP was independent of the duration of silicone oil endotamponade, and that the risk of developing ocular hypertension after a temporary silicone oil tamponade is moderate. It has been suggested that many complications may be prevented or their progression arrested when the silicone oil is removed a few weeks or months after surgery [68, 69], but there are few reports detailing the effects of removing it either as a prophylactic measure or as part of the management of established complications [68–72].

## Management

### Medical therapy

Medical therapy is initiated with cycloplegics and corticosteroids to decrease inflammation. Aqueous suppressants are generally used to reduce the pressure. Use of topical and systemic antiglaucoma medications controlled IOP in 30% of eyes (18 of 60) in a study by Honavar et al. [19].

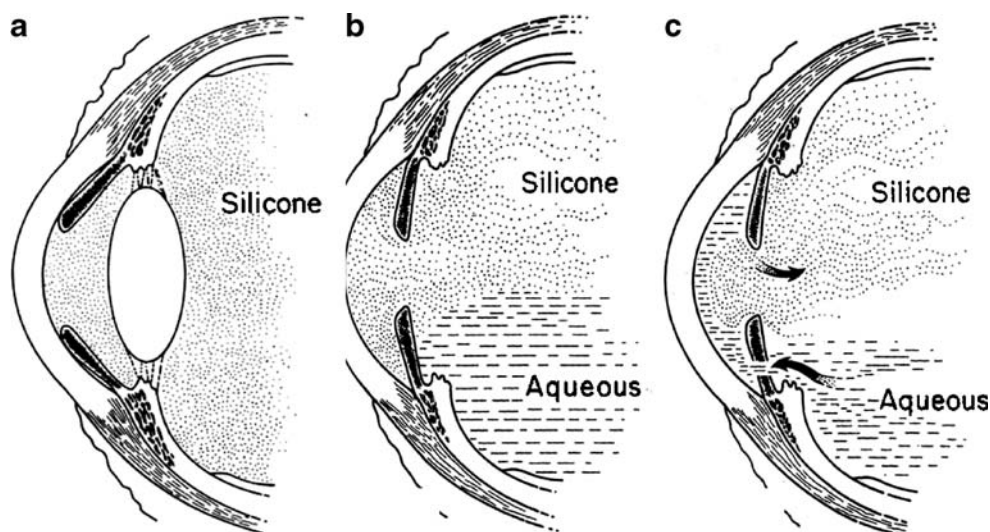
In a study by Al-Jazaaf et al., 78% of patients (40 of 51) with glaucoma after PPV and silicone oil injection were medically treated [73]. The intraocular pressure was controlled in most eyes with topical beta-blockers and prostaglandin analogues. The small proportion of patients requiring surgical therapy may be due to availability of effective topical antiglaucoma medications, or reduced severity of glaucoma due to improved silicone oil and surgical techniques [52, 73].

### Prophylactic peripheral iridectomy

Ando [46] introduced the concept of inferior peripheral iridectomy (PI) in aphakic and pseudophakic eyes to prevent forward migration of silicone oil and reduce the incidence of pupillary block glaucoma. Since silicone oil floats superiorly (specific gravity of 0.97), hence when the patient is upright, an iridectomy at the 6-o'clock position prevents pupillary block and ensures aqueous passage from the posterior to the anterior chamber (Fig. 4a–c). An iridectomy at the 12-o'clock position is unlikely to solve the problem, because the silicone oil bubble is directly behind the iris diaphragm superiorly and thus may block the PI [74–76]. In addition, aqueous accumulated inferiorly may not get access to the PI located superiorly. However, a superior PI has also been reported to be successful [77]. Superior Iridectomy is done when heavy silicone oil is used for tamponade, and is particularly suitable in patients requiring intracapsular cataract extraction after previous vitrectomy and silicone oil surgery.

Theoretically, a 15-micrometer diameter iridotomy should be large enough to prevent angle-closure glaucoma due to pupillary block, but practically an iridotomy should be at least 150 to 200 micrometers in diameter if acute angle-closure glaucoma is to be reliably prevented [78]. A very large PI may allow forward migration of the oil [74]. Unfortunately, postoperative closure of the PI occurs in approximately one third of eyes undergoing silicone oil surgery (particularly proliferative diabetic retinopathy), and is highly correlated with forward oil migration [72]. Face-down positioning may reverse the pupillary block in some cases [72].

**Fig. 4** **a** A large quantity of silicone oil in posterior chamber causing anterior displacement of iris. **b** A pool of aqueous in the lower vitreal space and posterior chamber. **c** An inferior iridectomy allows alleviation of the pupillary and direct trabecular block. Aqueous flow towards the anterior chamber and the oil bubble retracts [15]



### Laser therapy

Failure to perform an adequate inferior peripheral iridectomy or its closure increases the risk of developing pupillary block. Despite this precaution, inferior surgical iridectomies close in 11% to 32% of cases [7, 18, 19, 75, 76]. Reddy and Aylward [79] reported that the Nd: YAG laser failed to reopen 78% of closed inferior surgical iridectomies, and recommended a large surgical iridectomy if removal of SO is not desired. Zalta AH [80] has reported a short-term failure rate (92%) of laser iridotomy consistent with previous reports (89%–100%), [79, 81], while their long-term failure rate (38%) was much lower (78%–100%) [79, 81].

Transcleral cyclophotocoagulation has been used to treat glaucoma secondary to silicone oil. Successful IOP control has been reported in 66% to 82% of patients at 1 year [82–84].

Sivagnanavel [85] had a lower success rate for IOP reduction (56% treatment failure), despite the use of higher mean laser energies and a higher re-treatment rate. Multiple treatments may be required to attain intraocular pressure control, and since there is a risk of visual loss it is reserved for eyes with poor visual potential. Cyclodiode treatment does however carry a lower incidence of complications compared to cyclocryotherapy [86]. An additional, although extremely rare, complication of the cyclophotocoagulation is sympathetic ophthalmia [87, 88].

### Silicone oil removal

With increasing duration of contact between the emulsified silicone bubbles and the trabecular meshwork, organic changes in the trabecular endothelium and the collagen component of the trabecular meshwork are logical sequelae, resulting in sclerosis and collapse of the trabecular meshwork. At this stage, silicone oil removal may have no role in controlling IOP. Early removal of the emulsified

oil probably causes reversal of mechanical trabecular blockage by the oil particles, and/or contains damage to the filtration channels and thus helps better control of IOP. Early silicone oil removal with or without concurrent glaucoma surgery has been performed to lower IOP, but it carries some risk of retinal detachment.

Budenz et al. retrospectively reviewed the outcomes of surgical intervention for secondary glaucoma in 43 eyes that had pars plana vitrectomy with silicone oil injection [22]. Success was achieved in 69%, 60%, 56%, and 48% of eyes at 6, 12, 24, and 36 months respectively. Surgical treatment consisted of silicone oil removal alone in 74% patients; glaucoma surgery (trabeculectomy with or without antifibrotic agents, glaucoma drainage implant surgery and/or modified Schocket procedure) was performed with silicone oil removal in 19% of patients. The authors found that patients who underwent silicone oil removal alone to control IOP were more likely to have persistent IOP elevation, and possibly required reoperation for glaucoma, while patients who underwent concurrent silicone oil removal and glaucoma surgery were more likely to develop hypotony. Jonas et al. found that 93.4% (185 out of 198) patients with a secondary increase in IOP after silicone oil endotamponade had normalization of IOP after removal of the oil [24]. Flaxel et al. reported that elevated IOP persisted in all eyes (62 eyes) after silicone oil removal [89].

A study by Moisseiev et al. also demonstrated improvement in IOP control in only one of 11 eyes after removal of emulsified oil [90]. There are several major factors underlying post silicone oil removal rise in intraocular pressure. First, there is edema in the trabecular meshwork as a result of postoperative inflammation. Second, the mechanical impact of balanced salt solution during silicone oil removal may split the silicone oil droplets into much smaller drops, which are more likely to obstruct the trabecular meshwork [88]. It has been confirmed by

pathologic examination that emulsified silicone oil drops or macrophage endocytosis with silicone oil drops can block the trabecular meshwork [90, 91].

### Glaucoma surgery

Patients with complete synechial angle closure would not be expected to have normalization of IOP with silicone oil removal alone, in the absence of retinal redetachment. Glaucoma surgery may be indicated in such cases, and the decision as to whether to concomitantly remove the silicone oil would depend on an assessment of the relative risk of redetachment with oil removal [91]. When emulsified or nonemulsified oil blocks the trabecular meshwork directly, then silicone oil removal alone can help, if the retina is completely attached. Conventional filtration surgery has a limited role and success rate in the management of glaucoma after pars plana vitrectomy and silicone oil injection [9]. Trabeculectomy is technically difficult because of conjunctival scarring from the vitreoretinal surgery, and carries a poor prognosis. An inferior trabeculectomy is avoided because of the high risk of complications such as endophthalmitis.

### Glaucoma drainage devices

Glaucoma drainage implants offer a good surgical option in cases of refractory glaucoma associated with silicone oil [9]. But there is a possibility of silicone oil escape via the glaucoma drainage tube [92]. Al Jazaf et al. performed Ahmed Glaucoma shunt implantation (with inferotemporal placement of the plate and the tube) to reduce the chance of silicone oil from flowing into the tube [73]. They found a cumulative probability of success of 86% at 6 months and 76% at 1 year after implantation of an Ahmed Glaucoma shunt.

### Summary

The use of intravitreal silicone oil in conjunction with pars plana vitrectomy may lead to secondary open or closed angle glaucoma. Understanding the etiology of the pressure elevation is vital to control its level effectively. Patients with suspected risk factors for the development of glaucoma need to be more closely monitored. Surgical options, including Silicone oil removal, glaucoma drainage implants, and cyclodestructive procedures, may be successful in lowering IOP in complicated eyes. Treatment options should be directed at the underlying mechanism of secondary glaucoma for each patient balancing the risk of the intervention with the expected benefit.

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