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ORIGINAL RESEARCH

Unexplained Visual Loss After Silicone Oil Removal: A 7-Year Retrospective Study

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ABSTRACT

Introduction: Unexplained visual loss after removal of silicone oil from the eye has been described. The purpose of this study is to determine the incidence of unexplained loss of visual acuity after SO removal and to provide possible explanations for this phenomenon.

Methods: This retrospective study included patients that underwent vitreoretinal surgery, at Centro Hospitalar São João, between January of 2012 and October of 2018. Inclusion criterion was vitreoretinal surgery in which the chosen endotamponade was SO, followed by removal of SO and exchange with balanced salt solution (BSS) or air. After SO removal, patients with documented loss of best corrected visual acuity (BCVA) on two or more Snellen lines were analyzed and patients in which the cause of the visual loss was identified, namely OHT (intraocular pressure > 21 mmHg), retinal re-detachment, glaucoma, retinal proliferative membrane formation, or corneal decompensation, were excluded. All patients with unexplained visual loss underwent spectral domain optical coherence tomography (SD-OCT) to exclude causes of visual reduction such as cystoid macular edema, epiretinal membrane, or ellipsoid/interdigitation zone disruption. A p value less than 0.05 was considered statistically significant.

Results: A total of 46 eyes underwent SO tamponade and SO removal during the study period. In 34.8% of the cases (n = 16) there was visual acuity loss in at least two Snellen lines. Of 46 eyes, 23.9% (n = 11) showed vision loss due to known secondary causes. Unexplained loss of visual acuity after SO removal occurred in 10.9% of cases. OHT during silicone endotamponade (p = 0.046) and silicone emulsification (p = 0.001) were identified as factors associated with unexplained visual loss after SO removal.

Conclusion: Unexplained loss of visual acuity after SO removal occurred in 10.9% of cases. OHT during silicone endotamponade and SO emulsification were identified as important

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factors in the ethology of this phenomenon.

Keywords: Retinal detachment; Silicone oil; Unexplained visual loss; Vitrectomy

Key Summary Points
Why carry out this study?
Determine the incidence of unexplained loss of visual acuity after silicone oils (SO) removal.
Provide possible explanations for loss of visual acuity after SO removal.
What was learned from the study?
Unexplained loss of visual acuity after SO removal occurred in 10.9% of cases.
Intraocular hypertension (OHT) during silicone endotamponade and SO emulsification were identified as important factors in the ethology of this phenomenon.

INTRODUCTION

Silicone oils (SOs) are long-term, well-tolerated, chemically inert, and biocompatible vitreous substitutes [1–7]. Owing to these physical and chemical assets SOs ensure the maintenance of the adhesion between the retina and the retinal pigment epithelium and keep the pathological triggers away from the healing site [8–11]. For this reason, SOs are substances widely used in vitreoretinal surgery [12].

Indications for the use of SOs in vitreoretinal surgery include complicated retinal detachment associated with proliferative vitreoretinopathy, diabetic tractional retinal detachment, giant retinal tear, complicated full thickness macular hole, severe ocular trauma, and endophthalmitis [1, 2].

Despite their diverse attributes, SOs are not ideal intravitreal tamponade agents. Conventional SO floats, owing to its lower density compared to the vitreous humor, and as a result the inferior retina is not sufficiently supported because of incomplete filling of the vitreous cavity [4, 9]. As a consequence, pathological substances accumulate in the sub-silicone compartment, inducing an environment prone to retinal re-detachment with proliferative vitreoretinopathy. SO hydrophobicity, low viscosity, and the inadequate surface and interface tension are responsible for its emulsification and dispersion that culminate in a high incidence of long-term complications, namely inflammation, refractive changes, intraocular hypertension (OHT), cataracts, band keratopathy, glaucoma, peri-oil fibrosis, epiretinal membrane formation, metabolic changes, toxicity, and retinal re-detachment [1, 13–51].

The absence of biodegradability of SO and the repercussions in the visual function are determinants that impel the need for SO removal surgery, although small droplets can remain in the eye for at least 11 years after SO removal [13, 52–61]. In addition, the removal of SO is associated with a visual acuity improvement in approximately 30% of patients [53].

SOs have an established correlation with unexplained visual loss, but the mechanisms accountable for this phenomenon are not entirely known and many hypotheses have been proposed [56, 57, 62]. SO emulsification, exacerbated by ocular movements, promotes SO sequestration in the optic nerve and retina possibly causing retinal damage and optic neuropathy [63, 64]. Newsom et al. [54, 61] were the first to describe neuronal cell loss, particularly in the outer plexiform layer of the retina, as a result of SO tissue infiltration. Even though no typical electrophysiological pattern has been found in previous studies, generalized macular dysfunction with retinal lesions of ganglion cells and horizontal-bipolar cell synaptic processes in the outer plexiform layer have been reported as possible mechanisms [47, 58, 65, 66]. An alternative hypothesis is that SO emulsification and resulting tissue infiltration induces mechanical obstruction of the retinal vasculature and a decrease in retinal oxygen saturation that can trigger retinal hypoxia, particularly on the outer retina layers

[6, 50, 67, 68]. Besides that, other reports state that SO causes oxidative stress, by physical blockage of oxygen and metabolic exchange between the retina and the vitreous humor [69]. The ionic theory proposed that local changes in concentrations of potassium, calcium, and magnesium or in various cytokines levels can explain the visual loss. Another possible explanation may be light phototoxicity (through exposure to ambient light in the postoperative period) since SO transmits more light in the blue spectrum than vitreous and that could lead to apoptosis and macular dysfunction [47, 54, 55].

The purpose of this study is to determine the incidence of unexplained loss of visual acuity after SO removal and to provide possible explanations for this phenomenon.

METHODS

This retrospective study included patients that underwent vitreoretinal surgery, at Centro Hospitalar São João, between January of 2012 and October of 2018. Inclusion criterion was vitreoretinal surgery in which the chosen endotamponade was SO, followed by removal of SO and exchange with balanced salt solution (BSS) or air. Exclusion criteria included best corrected visual acuity (BCVA) under 2/10 (Snellen chart), retinal detachment during SO tamponade, removal SO followed by intraocular gas tamponade (instead of instillation of BSS or air) and complicated pars plana vitrectomy (PPV) during SO removal.

All medical records coded with 14.75 (vitreous substitute injection) in the ICD9-CM (International Classification of Diseases, Ninth Revision, Clinical Modification) were reviewed. Data collected included age, gender, affected eye, comorbidities, primary diagnosis (retinal detachment, diabetic retinopathy without retinal detachment, ocular traumatism, endophthalmitis, other), dates of SO injection and removal, total number of previous retinal surgeries, SO viscosity, duration of SO tamponade, complications during SO tamponade, concomitant procedure performed during SO removal, phakic state post removal of SO, BCVA with SO tamponade, BCVA at the last follow-up (after SO removal surgery), and postoperative complications.

After SO removal, patients with documented loss of BCVA on two or more Snellen lines were analyzed and all patients in which the cause of the visual loss was identified, namely OHT (intraocular pressure > 21 mmHg), retinal re-detachment, glaucoma, retinal proliferative membrane formation, or corneal decompensation, were excluded. All patients with unexplained visual loss underwent spectral domain optical coherence tomography (SD-OCT) to exclude causes of visual reduction such as cystoid macular edema, epiretinal membrane, or ellipsoid/ interdigitation zone disruption.

Statistical analysis was performed using SPSS v.22.0 (IBM Corp., Armonk, NY, USA). BCVA values of count fingers, hand motion, and light perception (LP) were assigned Snellen values of 0.014, 0.0052, and 0.0016, respectively. Patients' BCVA values were transcribed from their records and converted to a logarithm of the minimal angle of resolution (logMAR) scale for analysis. Count fingers, hand motion, and light perception were assigned logMAR values of 1.8, 2.3, and 2.8, respectively. Fisher's exact test was used to compare categorical data. Non-parametric analysis of variance (ANOVA) with Mann–Whitney test, chi-square test, and Kruskal–Wallis test were used to analyze continuous variables. A p value less than 0.05 was considered statistically significant. This study complies with the guidelines for human studies and was conducted ethically in accordance with the World Medical Association Declaration of Helsinki. Approval was provided by the Health Ethics Committee of São João Hospital Center.

RESULTS

Eyes that Underwent SO Removal

A total of 3231 eyes coded with 14.75 in the ICD9-CM classification, between January 2012 and October 2018, were selected. A total of 268 eyes underwent SO tamponade and SO removal during the study period; however, 222 eyes presented a BCVA under 2/10 (Snellen chart)

and were excluded. Hence, 46 eyes met study criteria.

Mean age was 61.39 ± 19.99 years (range 1–90 years), 67.4% were male, and in 50.0% the right eye was affected. Systemic comorbidities included diabetes mellitus (26.1%), systemic hypertension (63.0%), and dyslipidemia (41.3%) (Table 1).

The main indication to initial surgery was retinal detachment (67.4%), of which 50.0% were rhegmatogenous, 10.9% diabetic tractional, and 6.5% retinal detachment due to other causes, such as Coats disease, cytomegalovirus retinitis, and tractional retinal detachment due to proliferative membranes secondary to other vascular diseases. Other indications for initial surgery were diabetic retinopathy without retinal detachment (10.9%), ocular traumatism (6.5%), endophthalmitis (8.7%), and others, such as vitreous hemorrhage secondary to vascular diseases and macular hole (6.5%) (Table 2).

Prior to SO tamponade, 41.3% of the eyes had undergone phacoemulsification surgery and 32.6% of the eyes had been submitted to at least another one PPV.

In 41 eyes (89.1%) 5000 mPa s (millipascal seconds) SO was used as endotamponade while in 5 eyes (10.9%) 3000 mPa s SO was the choice. The mean time with SO tamponade was 7.27 ± 5.21 months (range 3–30 months) (Table 2).

During SO tamponade many complications occurred, namely OHT (47.8%), SO emulsification (30.4%), anterior chamber SO migration (17.4%), membrane proliferation (15.2%), glaucoma (10.9%), subconjunctival SO migration (8.7%), keratopathy (8.7%), and cataract (6.5%) (Table 3). In the course of SO removal, laser was performed in 52.2% of cases, internal limiting membrane (ILM) peeling in 19.6% of cases, and cataract phacoemulsification surgery in 2.2% of cases (Table 3). After SO removal, 4.3% were phakic and 95.7% were pseudophakic (Table 3). The mean logMAR BCVA prior to SO removal was 0.49 ± 0.21 (range 0.04–0.7) and subsequent to SO removal it was 0.68 ± 0.59 (range 0.0–2.8) (Table 3).

Table 1 Descriptive analysis of eyes that underwent silicone oil removal (n = 46)

Age (years)	61.39 ± 19.99 (range 1–90)	
Gender (male)	67.4%	n = 31
Eye (right eye)	50.0%	n = 23
Systemic comorbidities		
Diabetes mellitus	26.1%	n = 12
Systemic hypertension	63.0%	n = 29
Dyslipidemia	41.3%	n = 19

Best Corrected Visual Acuity After SO Removal

After SO removal, 30 eyes (65.2%) maintained or improved BCVA. However, in 34.8% of the cases (n = 16) there was visual acuity loss in at least two Snellen lines (Table 4).

Of 46 eyes, 23.9% (n = 11) showed vision loss due to known secondary causes. In nine eyes, vision loss occurred as a result of clear causes such as retinal re-detachment and proliferative vitreoretinopathy (five eyes), vitreous hemorrhage secondary to diabetic retinopathy (three eyes), and glaucoma/OHT (one eye) (Table 4). In the remaining seven eyes, SD-OCT was performed and revealed ellipsoid/ interdigitation zone disruption in two cases. In five eyes SD-OCT did not reveal any causes for the acute visual loss, such as cystoid macular edema or epiretinal membrane.

Unexplained Best Corrected Visual Acuity Reduction After SO Removal

Of the eyes included in this study, 10.9% (n = 5) presented unexplained loss of vision. Three of these patients (60.0%) were male. The mean age of this group was 56.60 ± 17.79 years (range 34–73 years). Systemic comorbidities included diabetes mellitus (20.0%), systemic hypertension (40.0%), and dyslipidemia (40.0%) (Table 5).

The main indication for initial surgery was retinal detachment (80.0%), followed by

Table 2 Descriptive analysis of eyes that underwent silicone oil removal ($n = 46$)

Indication for initial surgery		
Retinal detachment	67.4%	$n = 31$
Rhegmatogenous	50.0%	$n = 23$
Tractional	10.9%	$n = 5$
Others	6.5%	$n = 3$
Diabetic retinopathy without retinal detachment	10.9%	$n = 5$
Ocular traumatism	6.5%	$n = 3$
Endophthalmitis	8.7%	$n = 4$
Others	6.5%	$n = 3$
Silicone oil tamponade viscosity (mPa s)		
5000	89.1%	$n = 41$
3000	10.9%	$n = 5$
Mean time with silicone oil tamponade (months)	7.27 ± 5.21 (range 3–30)	

diabetic retinopathy without detachment (20.0%) (Table 5).

In four patients, 5000 mPa s SO was used as endotamponade and in one eye 3000 mPa s SO was chosen. The mean time with SO tamponade was 8.25 ± 3.11 months (Table 5).

There were no intraoperative complications during SO removal and the retina remained attached for the whole duration of the procedure. At the same surgical time, ILM peeling was performed in 20.0% of the cases, laser in 60.0% of the cases, and no cataract phacoemulsification surgery was executed (Table 5).

Eyes with unexplained visual loss and those without visual loss were compared regarding several variables including age, gender, affected eye, systemic comorbidities, indication for initial surgery, duration of SO tamponade, SO tamponade viscosity, complications during SO tamponade, phakic status after SO removal, and concomitant procedures performed during SO removal (Table 5). We observed that OHT during silicone endotamponade ($p = 0.046$) and silicone emulsification ($p = 0.001$) were factors associated with unexplained visual loss after SO removal (Table 5).

Regarding the emulsification rate of silicone oil, there were no differences between silicone 5000 and 3000 mPa s. Likewise, no difference was observed in the incidence of unexplained loss of visual acuity according to the silicone oil viscosity (Table 6).

DISCUSSION

Ever since vitreoretinal surgery techniques became widely recognized by the ophthalmologist community, the application of SO as intraocular tamponade expanded significantly [59].

SOs have an established correlation with unexplained visual loss but few explanations have been proposed to explain vision loss after SO removal.

In our retrospective study, of 46 patients that underwent SO tamponade and SO removal during the study period, 34.8% of eyes developed visual impairment. This corresponds positively with the findings reported by Roca et al., Christensen and La Cour, and Moya et al., 13%, 33%, and 50%, respectively [57, 62, 70].

Table 3 Descriptive analysis of eyes that underwent silicone oil removal ($n = 46$)

Complications during silicone oil tamponade		
Ocular hypertension	47.8%	$n = 22$
Silicone oil emulsification	30.4%	$n = 14$
Anterior chamber silicone oil migration	17.4%	$n = 8$
Membrane proliferation	15.2%	$n = 7$
Glaucoma	10.9%	$n = 5$
Subconjunctival silicone oil migration	8.7%	$n = 4$
Keratopathy	8.7%	$n = 4$
Cataract	6.5%	$n = 3$
Concomitant procedure during silicone oil removal		
Laser	52.2%	$n = 24$
Internal limiting membrane peeling	19.6%	$n = 9$
Cataract phacoemulsification surgery	2.2%	$n = 1$
Phakic status after silicone oil removal		
Phakic	4.3%	$n = 2$
Pseudophakic	95.7%	$n = 44$
Best corrected visual acuity (logMAR)		
Before silicone oil removal	0.49 ± 0.21 (0.04–0.7)	
After silicone oil removal	0.68 ± 0.59 (0.0–2.8)	

Table 4 Best corrected visual acuity changes after silicone oil removal ($n = 46$)

Reduction of best corrected visual acuity in 2 or more Snellen lines	34.8%	$n = 16$
Explained causes	23.9%	$n = 11$
Retinal re-detachment and proliferative vitreoretinopathy	10.9%	$n = 5$
Vitreous hemorrhage secondary to diabetic retinopathy	6.5%	$n = 3$
Glaucoma/ocular hypertension	2.2%	$n = 1$
OCT alterations	4.3%	$n = 2$
Unexplained causes	10.9%	$n = 5$
Maintenance or improvement of best corrected visual acuity	65.2%	$n = 30$

We observed that vision loss occurred in 23.9% of the 46 eyes and was due to known causes. This is compatible with the existing literature. Newsom et al. [61] observed that some loss of vision occurs in 26–27% of patients undergoing removal of silicone oil, due to re-

Table 5 Comparison of eyes that presented and did not present an unexplained loss of best corrected visual acuity (BCVA) following silicone oil removal

	Unexplained loss of BCVA (*n* = 5)		**No loss of BCVA (*n* = 30)**		***p***
Age (years)	56.60 ± 17.79 (34–73)		61.87 ± 22.31 (1–90)		0.785[a]
Gender (male)	60%	*n* = 3	66.7%	*n* = 20	0.637[c]
Eye (right eye)	60%	*n* = 3	43.3	*n* = 13	0.757[c]
Systemic comorbidities					
Diabetes mellitus	20%	*n* = 1	23.3%	*n* = 7	0.741[c]
Systemic hypertension	40%	*n* = 2	60.0%	*n* = 18	1.000[c]
Dyslipidemia	40%	*n* = 2	36.7%	*n* = 11	0.803[c]
Indication for initial surgery					
Retinal detachment	80%	*n* = 4	66.7%	*n* = 20	0.286[b]
Rhegmatogenous	80%	*n* = 4	63.3%	*n* = 19	
Tractional	0%	*n* = 0	3.3%	*n* = 1	
Others	0%	*n* = 0	0.0%	*n* = 0.0	–
Diabetic retinopathy without retinal detachment	20%	*n* = 1	13.3%	*n* = 4	0.559[c]
Ocular traumatism	0%	*n* = 0	6.7%	*n* = 2	0.566[c]
Endophthalmitis	0%	*n* = 0	6.7%	*n* = 2	0.566[c]
Other	0%	*n* = 0	6.7%	*n* = 2	0.566[c]
Silicone oil tamponade viscosity (mPa s)					
5000	80%	*n* = 4	86.7%	*n* = 26	0.436[c]
3000	20%	*n* = 1	13.3%	*n* = 4	0.559[c]
Mean time with silicone oil tamponade (months)	8.25 ± 3.21 (4–14)		5.91 ± 3.19 (3–17)		0.999[a]
Best corrected visual acuity (logMAR)					
Before silicone oil removal	0.32 ± 0.22 (0.1–0.5)		0.42 ± 0.13 (0.04–0.7)		–
After silicone oil removal	0.91 ± 0.19 (0.7–1.0)		0.32 ± 0.17 (0.0–0.7)		–
Complications during silicone oil tamponade					
Ocular hypertension	80%	*n* = 4	36.7%	*n* = 11	**0.046**[c]
Anterior chamber silicone oil migration	0%	*n* = 0	10.0%	*n* = 3	0.474[c]
Silicone oil emulsification	80%	*n* = 4	10.0%	*n* = 3	**0.001**[c]
Subconjunctival silicone oil migration	0%	*n* = 0	6.9%	*n* = 2	0.559[c]
Concomitant procedure during silicone oil removal					
Internal limiting membrane peeling	20%	*n* = 1	20.7%	*n* = 6	0.276[c]
Laser	60%	*n* = 3	48.3%	*n* = 14	0.641[c]

Table 5 continued

	Unexplained loss of BCVA (n = 5)		No loss of BCVA (n = 30)		p
Cataract phacoemulsification surgery	0.0%	n = 0	0.0%	n = 0	–
Phakic status after silicone oil removal					
Phakic	0%	n = 0	3.3%	n = 1	0.689[d]
Pseudophakic	100%	n = 5	96.7%	n = 29	0.415[d]

[a] Mann–Whitney U test
[b] Chi-square test
[c] Fisher's test
[d] Kruskal–Wallis test
Bold values indicate statistically significant

Table 6 Silicone oil emulsification rate and unexplained visual loss

Silicone oil (mPa s)	Patients with silicone oil tamponade		Silicone oil emulsification rate			Patients with unexplained visual loss		
	n	%	n	%	p	n	%	p
5000	41	89.1%	12	29.3%	0.432[a]	3	7.3%	0.298[a]
3000	5	10.9%	2	40%		1	20%	

[a] Fisher's test

detachment (6–25%), hypotony (16%), cystoid macular edema (12%), and epiretinal membranes (12%). Roca et al. [62] reported visual loss after SO removal secondary to identifiable causes in 7.1% of the eyes, namely retinal redetachment, proliferative vitreoretinopathy, vitreous hemorrhage, and glaucoma. Despite these findings, 10.9% of the eyes in our study developed visual loss of unexplained cause. This value is lower than in one report (29.7%) but higher (5.9% and 4.4%) than in two other studies [62, 71, 72].

In our study, SD-OCT was performed after SO removal in all eyes with no clear causes for visual acuity loss. However, no fluorescein angiograms or visual field testing was performed. In all cases in the literature, fluorescein angiograms and SD-OCT revealed no alterations that could justify alterations of visual acuity [54, 55, 58, 61]. We did not perform any electrophysiological studies, but they could be helpful to determine the location and possible nature of various dysfunctions along the visual pathway. In the literature, these studies suggest optic neuropathy, macular dysfunction, and generalized retinal dysfunction as causes of the loss of visual acuity [47, 54, 55, 58, 61, 73–76].

Some studies acknowledged young age, long-term SO tamponade, macula-on retinal detachment associated with giant retinal tear, and elevated intraocular pressure as potential risk factors for visual loss after SO removal [47, 54, 61, 62]. Our study identified OHT during silicone endotamponade (p = 0.046) as a risk factor for unexplained visual loss after SO removal as already published in the literature [72, 73]. An additional risk factor for unexplained visual loss after SO was SO emulsification (p = 0.001) during endotamponade. A possible explanation for the unexplained loss of

visual acuity that we observed in our patients lies in the combination of two factors: first, the damage associated with silicone oil emulsification due to infiltration of SO into the optic nerve and retina and consequent retinal damage and optic neuropathy; second, a possible greater susceptibility to neuronal damage that is created by OHT during silicone tamponade.

Particularly in aphakic patients, phototoxicity can be an additional explanation for the visual loss since unexplained visual loss was reported in 4.4% of eyes that underwent SO removal under direct illumination compared to only 1.3% under blocked illumination and the transmission of high energy blue light is more intense in eyes filled with SO in comparison to the vitreous humor [71, 76]. In addition, SO is responsible for dissolving fat-soluble macular pigments from the retina, particularly lutein and zeaxanthin, disrupting the protective mechanism against oxidative damage of these elements [71, 77–80]. However, in our study, there was no significant difference between phakic and pseudophakic eyes with regards to unexplained visual loss after SO removal.

Some authors defended that, particularly in aphakic patients, after SO removal, in the same way corneal edema is noted when aqueous layer comes back into contact with the corneal endothelium, a similar phenomenon may occur and contribute for the macular damage. However, if the events were analogous then macular edema would be expected and this was not observed in previous studies or in our study [54, 61].

Nevertheless, other explanations are described and proposed in the literature. The "vitreous potassium sink theory" suggests that SO tamponade impedes Müller cells from buffering extracellular potassium from the retina to the vitreous humor, and thus propitiates the rise of potassium concentration in the retro-oil fluid, an aqueous layer that results from the inability to completely fill the vitreous cavity with SO. SO removal then causes an abrupt alteration of the potassium concentration in the milieu that deregulates and triggers apoptosis of the retinal neurons and Müller cells [47, 56–58, 62, 74–76]. Another explanation for the visual loss involves the accumulation of growth factors, cytokines, and several metabolites, such as fibroblast growth factor and interleukin-6, in the retro-oil fluid. These may have a deleterious effect either during SO tamponade, as a result of the failure in the retinal buffering effect, or after SO removal, by dispersion of these substances with widespread damage to the surrounding tissues [77]. Finally, it is thought that changes in the retinal vasculature by the SO tamponade and an alteration in retinal blood perfusion, at the time of SO removal, may be a contributing factor to vision loss [58].

Although vison loss does not appear to be immediate after surgery but approximately 1–-5 months after surgery, we think it would be interesting to introduce data of intraocular pressure (medium, spikes) during the SO removal procedure, in future research [65].

The main limitations of our study include its retrospective nature, and hence the absence of a standardized research protocol with the inclusion of some complementary data. The fact that this study involved patients of a single center may not adequately reflect geographical variation. However, it included comprehensive and accurate data collection, it compares eyes with unexplained visual loss to those without visual loss, and SD-OCT was performed in eyes with apparent unexplained visual loss.

CONCLUSION

The incidence of vision loss after removal of SO is of great importance (34.8%). In about 23.9% of the eyes that underwent SO removal, the loss of visual acuity occurred as a result of an identifiable etiology, but in 10.9% of cases the cause of visual loss remains unknown. This study encountered OHT during silicone endotamponade and SO emulsification as important factors in the ethology of this phenomenon. On the basis of the evidence, close monitoring of intraocular pressure in patients that underwent SO tamponade and SO removal is of critical importance. In addition, SO removal surgery should be performed as soon as possible.

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Compliance with Ethics Guidelines. This study complies with the guidelines for human studies and was conducted ethically in accordance with the World Medical Association Declaration of Helsinki. Approval was provided by the Health Ethics Committee of São João Hospital Center.

Data Availability. The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

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