

# Incidence of and Risk Factors for Silicone Oil–Associated Cystoid Macular Edema After Pars Plana Vitrectomy

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## Abstract

**Purpose:** To observe the incidence of and risk factors for cystoid macular edema (CME) after silicone oil (SO) implantation after pars plana vitrectomy (PPV) for retinal detachment (RD) repair. **Methods:** This retrospective analysis used the TriNetX database to identify patients who received SO tamponade after PPV for RD between March 2014 and March 2023. The onset and regression of CME were identified using spectral-domain optical coherence tomography. The demographics, intraoperative parameters, and postoperative disease course of patients with and patients without CME were compared using  $\chi^2$  tests, Student *t* tests, and logistic regression models. **Results:** Twenty (25.3%) of 79 eyes developed CME after intraocular insertion of SO. The use of 1000 cs SO (*n* = 50) vs 5000 cs SO (*n* = 29) was significantly associated with CME onset (odds ratio, 4.46; *P* < .05). The mean ( $\pm$  SD) SO tamponade duration was  $199.0 \pm 125.5$  days. The mean time from SO implantation to detection of CME was  $82.6 \pm 57.9$  days. Disease regression occurred in 15 (75.0%) of the 20 eyes with CME and was recorded a mean of  $218.2 \pm 256.2$  days after SO removal. Compared with untreated groups, the frequency of CME regression was not influenced by the administration of sub-Tenon triamcinolone acetate (75.0% vs 75.0%; *P* = 1.00), prednisolone acetate eyedrops (75.0% vs 75.0%; *P* = 1.00), or ketorolac eyedrops (71.4% vs 76.9%; *P* = .79). **Conclusions:** The viscosity of the SO used for vitreous tamponade in RD repair may play a role in the development of CME, with lighter grade oil increasing the risk for disease. Furthermore, SO removal alone potentially leads to a prominent reduction in CME in most cases.

## Keywords

cystoid macular edema, optical coherence tomography, pars plana vitrectomy, retinal detachment, silicone oil

## Introduction

Cystoid macular edema (CME) is a common ophthalmic pathology marked by the presence of cystic intramembranous fluid within the retina that results from disruptions in the blood–retina barrier, inducing leakage and subsequent accumulation of intraretinal fluid.<sup>1–4</sup> The resulting anatomic changes caused by CME can be effectively monitored by detailed cross-sectional images provided by spectral-domain optical coherence tomography (OCT).<sup>2,3</sup>

Although typically inflammatory etiologies, diabetic retinopathy (DR), and various other macular conditions have been implicated in the development of CME, retina surgeons have also observed that it commonly occurs after the implantation of intraocular silicone oil (SO).<sup>1,4,5</sup> SO is injected during pars plana vitrectomies (PPVs) to provide a tamponade in the vitreous cavity, stabilizing the retina during vital stages of retina attachment and laser maturation.<sup>5,6</sup> While its influence on ocular recovery is primarily therapeutic, SO has also been shown to possess inflammatory properties that are theorized to induce ME.<sup>5–7</sup>

Because postsurgical CME can lead to long-term visual impairments, adding to the body of literature with regard to its incidence as well as identifying risk factors for SO-related CME could be valuable for retina surgeons.<sup>8,9</sup> The purpose of the current study was to observe the incidence of and risk factors for CME after SO tamponade for PPV in retinal detachment (RD) repair.

## Methods

This retrospective study was approved by the Institutional Review Board, Medical College of Wisconsin, and adhered to the tenets of the Declaration of Helsinki. Medical information

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was extracted from the TriNetX data warehouse to identify patients who had PPV for RD between March 2014 and March 2023. Procedures were performed by multiple surgeons on the retina service at a single Midwestern academic medical center. Exclusion criteria were a history of CME (before SO implantation), DR, uveitis, central retinal artery or vein occlusions, pigmentary retinal dystrophy, and other inflammatory retinal diseases. The remaining medical charts were reviewed to exclude eyes that had vitrectomies without SO tamponade or lacked OCT imaging after SO implantation.

A comprehensive review of OCT reports taken before, during, and after the SO tamponade phase was conducted to ascertain the onset and regression of CME. The presence of CME, defined as an observable increase in macular thickness and intraretinal cysts, was identified and recorded by retina specialists responsible for the care of the patients. Eyes were categorized into 2 groups based on their CME status; that is, CME positive or CME negative. Those assigned to the CME-positive group had findings of CME on OCTs obtained during SO tamponade, while those assigned to the CME-negative group did not display such edema.

Demographic information included race, age, sex, and laterality of the involved eye. Intraoperative information included the macular status of the RD, SO viscosity (1000 cs or 5000 cs), nature of the RD, concurrent retina procedures, scleral buckle history, SO tamponade history (before the studied procedure), lens status during tamponade, and duration of SO implantation. The duration of tamponade was defined as the time between SO injection and SO removal. In eyes that did not have SO removed, the duration of tamponade was recorded as the interval between SO injection and the latest OCT scan within 1 year of surgery. Last, information on the course of CME recovery and the targeted treatments was documented. Regression of CME was defined as a prominent decrease in the appearance of both cystoid spaces and foveal thickness on OCT.

Categorical data from the CME-positive group and CME-negative group were analyzed using  $\chi^2$  tests, while continuous variables were evaluated using Student *t* tests. Logistic regression models were then used to identify significant risk factors for CME after SO implantation. Univariate logistic regression models were used for each independent variable. Multivariate logistic regression was performed on variables obtained after a stepwise-forward selection process to account for potential confounders. Statistical significance was set at  $P < .05$ . All mean values are  $\pm$  SD.

## Results

The initial extraction of patient charts and application of inclusion and exclusion criteria yielded 256 individuals. After a thorough review of the medical records, 177 patients were excluded for insufficient OCT imaging or loss to follow-up after SO implantation. The final group comprised 79 eyes of 79 patients.

Twenty (25.3%) of 79 eyes had positive findings for CME on OCT during SO tamponade and were assigned to the CME-positive cohort; the remaining 59 eyes were assigned to the

CME-negative cohort. The mean duration of SO implantation for all patients was  $199.0 \pm 125.5$  days. The mean duration of tamponade in the CME-positive cohort and CME-negative cohort was  $179.4 \pm 122.6$  days and  $205.5 \pm 125.9$  days, respectively ( $P = .420$ ); the mean duration of tamponade in eyes that received 1000 cs SO ( $n = 50$ ) and in eyes that received 5000 cs SO ( $n = 29$ ) was  $188.8 \pm 135.0$  days and  $216.2 \pm 118.2$  days, respectively ( $P = .314$ ).

Table 1 summarizes the baseline demographic characteristics of patients enrolled in the study. There was no statistically significant difference in race ( $P = .341$ ), age ( $P = .207$ ), sex ( $P = .818$ ), or laterality ( $P = .563$ ) between the CME-positive cohort and the CME-negative cohort.

Table 2 compares the intraoperative data during PPV between the CME-positive cohort and the CME-negative cohort. SO viscosity was the only statistically significant predictor for CME onset. The use of 1000 cs oil rather than 5000 cs oil was associated with CME-positive eyes ( $P = .020$ ). There was no statistically significant difference between the groups in combined retina procedures, including membranectomy ( $P = .599$ ), laser treatment ( $P = .304$ ), retinectomy ( $P = .254$ ), or triamcinolone acetonide injection ( $P = .359$ ). In addition, the macular status ( $P = .893$ ), history of scleral buckling ( $P = .373$ ), SO history ( $P = .428$ ), lens status ( $P = .276$ ), and nature of the RD ( $P = .675$ ) were not significantly associated with CME outcomes.

After analysis of variables with univariate logistic regression, the use of 1000 cs SO was the only statistically significant risk factor for CME (odds ratio [OR], 4.46; 95% CI, 1.18-16.89;  $P = .028$ ). In contrast, demographic background, macular status, combined retinal procedures, history of scleral buckling, SO history, lens status, and RD nature were not statistically significant. Multivariate analysis with the stepwise-forward selection method found SO viscosity to be the only statistically significant risk factor for CME.

All patients were treated with a postoperative course of prednisolone acetate eyedrops for the insertion and removal of the SO. For disease management, patients in the CME-positive cohort ( $n = 20$ ) may have received a combination of postoperative sub-Tenon triamcinolone acetonide ( $n = 12$ ), additional prednisolone acetate eyedrops ( $n = 12$ ), or ketorolac eyedrops ( $n = 7$ ). The duration of topical therapy with prednisolone acetate and ketorolac ranged from 4 to 6 weeks. In patients who received sub-Tenon triamcinolone acetonide, a mean of  $1.33 \pm 0.471$  injections were administered. These therapies were given after the CME diagnosis during the SO tamponade phase or after SO removal. Table 3 summarizes the disease course and treatments received by the CME-positive cohort.

Eighteen (90.0%) of the 20 eyes in the CME-positive cohort had SO removal; the 2 patients without SO removal did not experience disease regression. Fifteen (75.0%) of the 20 eyes in the CME-positive cohort had CME regression (Figure 1B and Figure 2B), with all instances taking place after SO removal. Regression was recorded a mean of  $218.2 \pm 256.2$  days after SO removal. Eyes that received sub-Tenon triamcinolone acetonide (75.0% vs 75.0%;  $P = 1.00$ ), prednisolone acetate eyedrops (75.0% vs 75.0%;  $P = 1.00$ ), or ketorolac eyedrops (71.4% vs

**Table 1.** Comparison of Demographics Between Cohorts.

Characteristic	Cohort		P Value <sup>a</sup>
	CME Positive	CME Negative	
Eyes, n	20	59	
Race, n (%)			.341
White	19 (95.0)	46 (78.0)	
Black	1 (5.0)	6 (10.2)	
Hispanic	0	2 (3.4)	
Asian	0	5 (8.5)	
Mean age (y) $\pm$ SD	62.8 $\pm$ 8.7	56.6 $\pm$ 21.0	.207
Sex, n (%)			.818
Male	13 (65.0)	40 (67.8)	
Female	7 (35.0)	19 (32.2)	
Laterality of RD, n (%)			.563
Right eye	8 (40.0)	28 (47.5)	
Left eye	12 (60.0)	31 (52.5)	

Abbreviations: CME, cystoid macular edema; RD, retinal detachment.

<sup>a</sup>Variables between groups were compared with  $\chi^2$  tests and Student *t* tests.**Table 2.** Comparison of Intraoperative Data Between Cohorts.

Parameter	Cohort		P Value <sup>a</sup>
	CME Positive	CME Negative	
Eyes, n	20	59	
Macular status, n (%)			.893
Attached	4 (20.0)	11 (18.6)	
Detached	16 (80.0)	48 (81.4)	
SO viscosity, n (%)			.020 <sup>b</sup>
1000 cs	17 (85.0)	33 (55.9)	
5000 cs	3 (15.0)	26 (44.1)	
Combined procedures, n (%)			.599
Membranectomy	17 (85.0)	47 (79.7)	
Laser	20 (100.0)	56 (94.9)	.304
Retinectomy	10 (50.0)	21 (35.6)	.254
TA injection	11 (55.0)	39 (66.1)	.359
History of SB, n (%)			.373
Yes	11 (55.0)	39 (66.1)	
No	9 (45.0)	20 (33.9)	
History of SO, n (%)			.428
Yes	5 (25.0)	10 (16.9)	
No	15 (75.0)	49 (83.1)	
Lens status, n (%)			.276
Phakic	7 (35.0)	23 (39.0)	
Pseudophakic	11 (55.0)	22 (37.3)	
Aphakic	2 (10.0)	14 (23.7)	
Nature of RD, n (%)			.675
Rhegmatogenous	18 (90.0)	56 (94.9)	
Tractional	1 (5.0)	1 (1.7)	
Hemorrhagic	1 (5.0)	2 (3.4)	

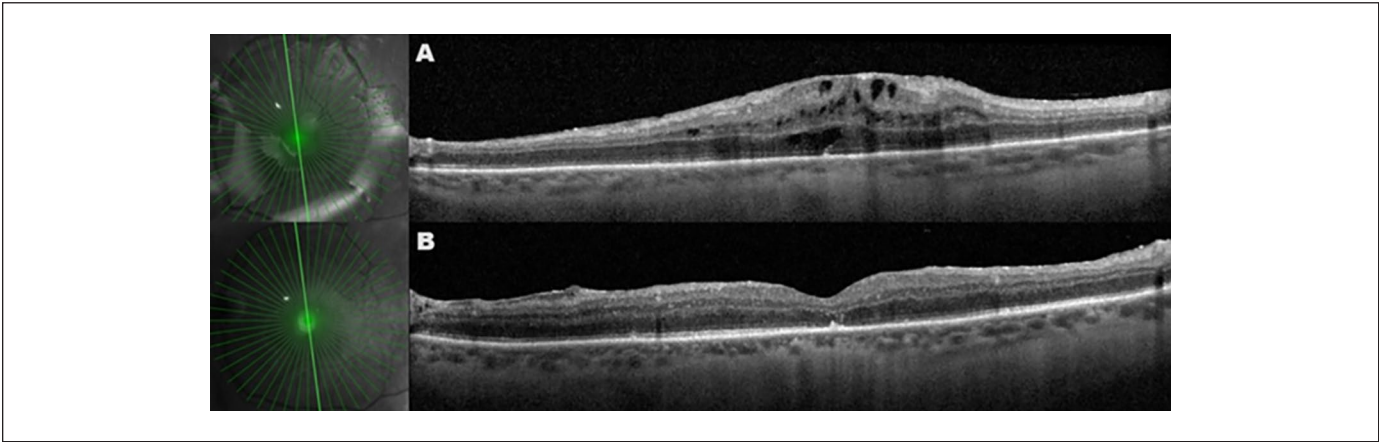
Abbreviations: CME, cystoid macular edema; RD, retinal detachment; SB, scleral buckle; SO, silicone oil; TA, triamcinolone acetonide.

<sup>a</sup>Variables between groups were compared with  $\chi^2$  tests and Student *t* tests.<sup>b</sup>Statistically significant.

**Table 3.** Postoperative Course of Eyes in the CME-Positive Cohort.<sup>a</sup>

Metric	n (%)	Mean Duration (d)±SD
Eyes	20	
Time to CME detection after SO implantation	20	82.6 ± 57.9
Eyes given 1000 cs SO	17	70.4 ± 39.4
Eyes given 5000 cs SO	3	152.0 ± 104.5
Disease regression		
Eyes with regression	15 (75.0)	—
Time to regression after SO removal	15	218.2 ± 256.2
Postoperative sub-Tenon TA		
Eyes treated with sub-Tenon TA	12	—
Treated eyes with regression	9 (75.0)	—
Eyes not treated with sub-Tenon TA	8	—
Untreated eyes with regression	6 (75.0)	—
PA eyedrops		
Eyes treated with PA eyedrops	12	—
Treated eyes with regression	9 (75.0)	—
Eyes not treated with PA eyedrops	8	—
Untreated eyes with regression	6 (75.0)	—
Ketorolac eyedrops		
Eyes treated with ketorolac eyedrops	7	—
Treated eyes with regression	5 (71.4)	—
Eyes not treated with ketorolac eyedrops	13	—
Untreated eyes with regression	10 (76.9)	—

Abbreviations: CME, cystoid macular edema; PA, prednisolone acetate; SO, silicone oil; TA, triamcinolone acetonide.  
<sup>a</sup>All treatments were administered after CME diagnosis during the SO tamponade period or after SO extraction.



**Figure 1.** (A) Spectral-domain optical coherence tomography shows cystoid macular edema after implantation with 1000 cs silicone oil (SO). (B) Disease regression is seen 120 days after SO removal.

76.9%; *P* = .787) did not have significantly increased regression rates compared with untreated groups.

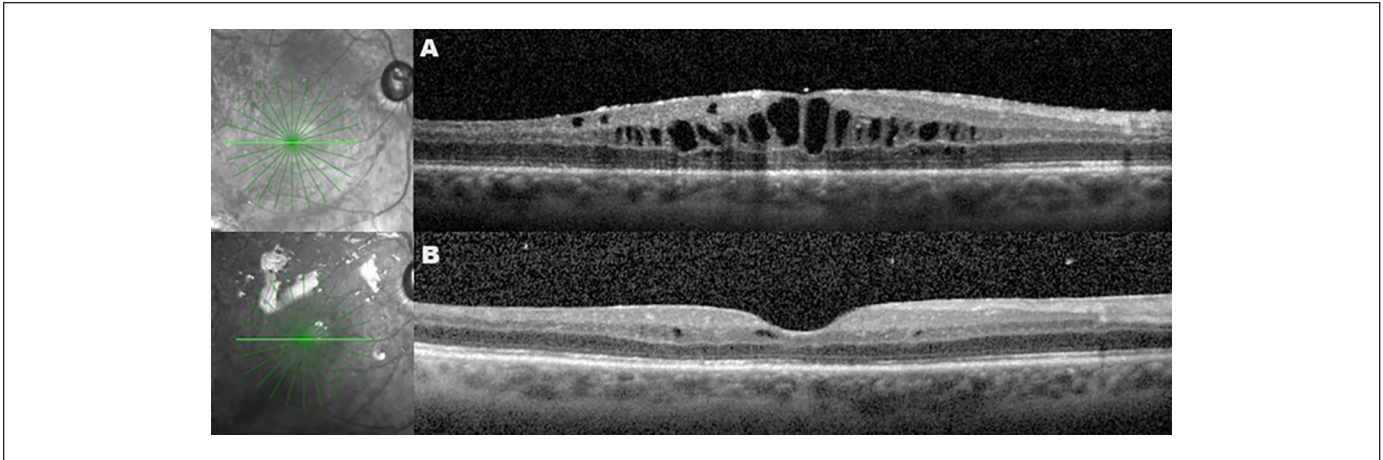
**Conclusions**

Current research on the risk factors for CME after SO tamponade for PPV is relatively limited, with the recent body of literature mostly comprising small series. To our knowledge, the current study represents the largest reported series of

vitrectomy cases evaluated for SO-related CME outcomes in a Western population.

In this study, the onset of CME after SO implantation occurred in 20 (25.3%) of 79 eyes. This incidence falls within the middle to higher range of that documented in past similar studies (3.5% to 36.2%).<sup>2,10–15</sup> This broad range likely stems from the diverse methodologies and clinical standards across different studies. For instance, Yang et al<sup>10</sup> reported the highest CME rate (36.2%) among 58 patients; however, those patients





**Figure 2.** (A) Spectral-domain optical coherence tomography shows cystoid macular edema after implantation with 5000 cs silicone oil (SO). (B) Disease regression is seen 71 days after SO removal.

also had the longest average SO tamponade duration (274 days). Most other studies recorded tamponade durations between 105 days and 270 days.<sup>11–13</sup> Rashad et al<sup>2</sup> reported the next highest incidence (27.5%) in a group of 51 eyes. However, one half of their patients with CME had diabetic ME, an exclusion criterion in the current study and other studies.<sup>13,14</sup>

On the opposite end of the spectrum, Shaheen et al<sup>14</sup> reported the lowest CME incidence (3.5%); however, unlike other investigations, including ours, patients without SO removal were excluded. Shah et al<sup>15</sup> studied the frequency of CME after various types of PPVs, with SO-involving PPVs accounting for roughly 3% of 708 total cases. A low SO-related CME incidence of 4.8% (1 of 21 cases) was reported. This low rate may have resulted from the limited sample size of PPVs with SO tamponade.

In the current study, we theorize our moderately high CME incidence resulted from the inclusion of multiple SO viscosities and a relatively long mean tamponade duration (199 days). Nevertheless, our rate appears to correspond with that of past published studies.

With specific regard to risk factors, the most notable finding in our study was a strong statistically significant association between SO viscosity and postoperative CME. Eyes that received 1000 cs SO as opposed to 5000 cs SO were significantly more likely to develop CME (OR, 4.46). The potential pathophysiology behind this outcome could be explained by the decreased stability of lighter grade SO.<sup>16,17</sup>

Kartasasmita et al<sup>18</sup> explored this phenomenon by observing in vivo emulsification rates of 1000 cs and 5000 cs intraocular SO after PPV for RD. Spectrophotometry was used to record a significantly higher absorbance and lower transmittance from 1000 cs SO than from 5000 cs SO after 8 to 12 weeks, signifying the elevated propensity for emulsification by 1000 cs SO.<sup>18</sup> In a separate study, Klettner et al<sup>19</sup> found that retinal microglia consumed particles from emulsified SO and, in response, released proinflammatory cytokines. The findings in both studies suggest that eyes receiving 1000 cs SO rather than 5000 cs

SO experienced heightened oil emulsification and inflammation, which induced CME.<sup>18,19</sup> Moreover, in our study, earlier disease detection was seen in CME-positive eyes receiving 1000 cs SO than in those receiving 5000 cs SO, further corroborating this notion.

Yet, the current opinion on the association between SO viscosity and clinical emulsification appears to be divided.<sup>20–26</sup> Valentin-Bravo et al<sup>21</sup> evaluated 5 studies on this subject and reported that differing SO viscosities did not alter clinically observed emulsification rates.<sup>22–26</sup> Four of these 5 studies, however, did not outline specific diagnostic criteria for emulsification.<sup>22–25</sup> Extremely low or high total emulsification rates were also reported, resulting in a floor or ceiling effect as follows: 0.8% (n = 393),<sup>22</sup> 2.2% (n = 325),<sup>23</sup> 6.8% (n = 44),<sup>24</sup> and 100% (n = 82).<sup>25</sup>

In contrast, Zafar et al<sup>26</sup> defined a clear emulsification criterion of pseudohypopyon presence, reporting that 1000 cs SO (n = 44 [52.3%]) had a significantly higher emulsification rate and earlier removal than 5000 cs SO (n = 41 [22.0%]). To that end, this relationship remains unclear and inconsistent and warrants further investigation. Subclinical emulsification, as studied by Kartasasmita et al,<sup>18</sup> should also be considered a potential factor for SO viscosity-related outcomes. Of note, although we suspect variable oil emulsification rates could explain our outcomes, clinical signs of emulsification, such as hyperoleon, were not explicitly reported or measured in any chart reviewed in the current study.

Supporting literature on the association between SO viscosity and CME is rare because most investigations into this subject did not involve the use of more than 1 oil viscosity. Previous studies all assessed CME outcomes for SO-filled eyes of only a single oil viscosity.<sup>2,10–13</sup> In contrast, the study by Shaheen et al<sup>14</sup> included multiple oil viscosities, specifically 1000 cs SO and 5000 cs SO, the same viscosities used in our study. In their report, 17 (89.5%) of 19 eyes with postoperative CME received 1000 cs SO, while only 1 eye received 5000 cs SO and 1 eye received an unreported viscosity. Not including the eye with an

unknown viscosity, the observed ratio of 1000 cs SO use to 5000 cs SO use (17:1) in eyes with CME onset is similar to that of our study (17:3). This potentially supports our theory on the role of SO viscosity in CME. However, the Shaheen et al<sup>14</sup> study did not evaluate patients without CME; therefore, the denominator to assess the individual incidence rates of CME in each of the SO groups is unknown.

Aside from oil viscosity, our study did not find additional predictors for CME after SO tamponade. Although few other risk factors have been reported in similar studies, previous studies by Azzolini et al<sup>27</sup> and Scheerlinck et al<sup>28</sup> reported a direct correlation between the CME incidence and SO tamponade duration. Our study, however, showed the opposite trend, with a shorter, although nonsignificant, tamponade duration in the CME-positive cohort than in the CME-negative cohort. This likely resulted from our involvement of multiple SO viscosities because most eyes in the CME-positive cohort received 1000 cs SO, which in general was removed earlier than 5000 cs SO.

Macula-off status was reported as an additional risk factor by Azzolini et al<sup>27</sup>; however, this finding was not replicated in the current study or others, including the study by Yang et al,<sup>10</sup> in which posterior staphyloma was a predisposing factor for SO-related CME,<sup>11–13</sup> a particularly unique predictor not commonly examined in other studies. Their finding was linked to the abnormal vitreous anatomy of eyes with posterior staphyloma, theorized to form a retro-oil space that fostered local inflammation.<sup>10</sup> However, this may be a more specific finding for populations in Asia because in our US cohort, posterior staphyloma was not frequently or explicitly reported in any chart.

Another important evaluation was the influence of triamcinolone acetonide on both the prevention and treatment of CME. Regarding prophylaxis, triamcinolone acetonide injections administered during SO implantation did not reduce postoperative CME outcomes. This could have occurred because triamcinolone acetonide typically loses its effectiveness after roughly 42 days.<sup>29,30</sup> In our study, the mean detection of CME was 82.6 days after SO implantation. Hence, the poor effectiveness of intraoperative triamcinolone acetonide on CME during SO tamponade may be attributed to its waning potency at the time of oil emulsification and subsequent inflammation.

Regarding the treatment of SO-related CME, triamcinolone acetonide did not appear to improve disease outcomes in affected patients. In eyes in the CME-positive cohort, the administration of postoperative sub-Tenon triamcinolone acetonide did not increase the frequency of CME regression compared with untreated eyes, nor did topical prednisolone acetate or ketorolac enhance CME regression. Shaheen et al<sup>14</sup> noted similar findings, reporting that medications, including triamcinolone acetonide, topical steroids, and topical nonsteroidal anti-inflammatory drugs, do not increase the CME recovery rate. These findings call into question the necessity of SO-related CME treatments beyond SO removal. Yang et al<sup>10</sup> reported qualitative improvement in 81.8% of untreated patients after SO removal, as seen on OCT, while a study by Bae et al<sup>12</sup> found that 88.9% of untreated patients showed improvement after SO removal.

In our study, 6 (75.0%) of 8 eyes without any CME-targeted treatments exhibited regression of CME after SO removal. It is suspected that the removal of SO redistributes inflammatory factors into the vitreous cavity that were originally concentrated on the macula, permitting macular recovery.<sup>31,32</sup> This phenomenon may be sufficient for spontaneous CME regression. Whether postoperative triamcinolone acetonide or other interventions provide additional benefits in the resolution of SO-related CME requires further investigation.

The current study faces multiple inherent limitations as a result of its retrospective design. In particular, a significant portion of screened patients was excluded as a result of insufficient OCT imaging. This limited our study size and potentially introduced sampling bias because it is plausible that patients suspected of having ocular inflammation received more frequent imaging, which would influence outcomes, such as the demographics, clinical courses, and CME incidence. Yet this appeared to be surgeon-dependent, with some using imaging more routinely than others. OCT imaging was also commonly deferred in cases in which meaningful vision was deemed unsalvageable or clinical outcomes were extremely poor. Thus, possible sampling bias could be mitigated by the fact that patients with any imaging at all, and therefore not excluded, were typically under the care of a subgroup of retina surgeons who ordered OCTs for nearly every visit.

This study was also limited by the subjectivity of CME diagnoses because they were based on interpretations of the physician reviewing the OCT images. In addition, further selection bias was possible because the SO viscosity administered to patients was not randomized. In addition, SO emulsification may be affected by factors such as interfacial tension, mechanical shear forces, surgical equipment, and surfactant compounds, which were beyond the scope of what is typically recorded in clinically oriented operative reports and encounter documentation.<sup>33</sup> Last, this project was conducted at a single hospital; therefore, applying these findings broadly is restricted by the limited diversity of the study population.

In conclusion, the evidence from our investigation suggests that eyes given lighter grade SO for intraocular tamponade may be at an increased risk for developing CME. We hope that our findings help inform decision-making by retina surgeons when SO tamponade is needed as part of vitreoretinal surgery. Although 5000 cs SO is certainly more difficult and slower to implant and explant from the eye, the authors recommend considering CME as a factor when choosing a SO viscosity, in particular in eyes with useful visual potential, predisposing risk factors for CME, or lengthier durations of SO implantation.

### Ethical Approval

This study was conducted in accordance with the Declaration of Helsinki. Ethical approval for this research project was granted from the Institutional Review Board, Medical College of Wisconsin, ensuring compliance with ethical guidelines to protect human subjects involved in this study.

## Statement of Informed Consent

Because this was a retrospective study, informed consent was not required by the Institutional Review Board, Medical College of Wisconsin. Patient confidentiality was protected throughout this investigation's data-collection process.

## Declaration of Conflicting Interests


The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of the article.

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