Prevention and management of postcataract cystoid macular edema

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Purpose of review
This review highlights treatment options, both under investigation and currently available, for treating postcataract macular edema. An update on current clinical studies for postcataract macular edema has been summarized.

Recent findings
Pseudophakic cystoid macular edema (PCME) is a common complication of cataract surgery leading to decreased visual acuity due to inflammation promoting vascular permeability and macular edema. There is no gold standard protocol for treatment with physicians choosing topical NSAIDs and corticosteroids most commonly. Recent developments in a therapeutic approach to PCME include improved delivery methods via implantation and improved drugs and combinational therapies.

Summary
While PCME treatments are poorly studied due to their common sudden resolution without medical intervention, chronic PCME is debilitating for patients. Clinical studies show hope for improved drug delivery methods, practices to prevent potential PCME, and improved therapeutics.

Keywords
Irvine-Gass syndrome, post cataract macular edema, pseudophakic cystoid macular edema

INTRODUCTION

Pseudophakic cystoid macular edema (PCME), also known as Irvine-Gass syndrome, is one of the most common causes of decreased visual acuity after cataract surgery [1] and develops in up to 22% of patients following surgery [2]. This condition develops postsurgery due to an upregulation of inflammatory mediators, which break down the blood-aqueous and blood-retinal barriers [3–5]. This increases the vascular permeability of the retina and consequently promotes fluid accumulation in the macula [3–5]. The effects are compounded in the highly vascular and metabolically demanding macula [6]. After cataract surgery, inflammatory mediators released in the eye such as vascular endothelial growth factor (VEGF) and TNF-alpha, increase vascular permeability, and the macula most susceptible to these blood-retinal barrier changes [6,7]. Risk factors correlated with PCME onset include increased preoperative central macular thickness [8], high-grade preoperative idiopathic epiretinal membrane [9▪], diabetes mellitus [10,11], prostaglandin analog (PGA) use [4], and pupil expansion devices [12].

Current treatment of PCME includes a combination of anti-inflammatory drops such as NSAIDs, corticosteroids (topical, periocular and intraocular), carbonic anhydrase inhibitors, anti-VEGF injections, and various immunomodulators. Surgical approaches include laser vitreolysis and pars plana vitrectomy, in select cases which are attributed to vitreous prolapse [13,14]. Recent advances have ventured into promising treatments including new drug developments and drug delivery systems. In this review, we will discuss a few treatments for PCME.

THERAPEUTICS

NSAIDS

A study by Jukić *et al.* [15] aimed to compare the effects of topical bromfenac and dexamethasone on

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KEY POINTS

- There is conflicting research to address whether preoperative intervention can adequately address the risk for developing PCME.
- Interventions under investigation for the management of PCME include OCS-01 which is a steroid drop which may address inflammation without having a significant increase in IOP.
- Many approaches are being studied and utilized to manage PCME including postoperative NSAID and steroid eye drops, intravitreal injections of steroids and anti-VEGFs, and discontinuation of prostaglandin analogs.

intraocular interleukin-6 (IL-6) concentrations and the incidence of PCME after cataract surgery in patients with nonproliferative diabetic retinopathy (NPDR). Ninety eyes with mild-to-moderate NPDR were divided into three groups: bromfenac, dexamethasone, and placebo [15]. IL-6 levels in the aqueous humor and central foveal subfield thickness (CFT) were measured pre and postsurgery [15]. Results showed no significant difference in IL-6 levels between groups, but postoperative CFT was lower in the dexamethasone group compared to placebo [15]. A significant correlation between IL-6 and CFT was found in the dexamethasone group [15]. No cases of PCME or adverse events occurred [15]. The study concluded that neither bromfenac nor dexamethasone significantly affected IL-6 concentrations, and bromfenac was not more effective than dexamethasone in reducing CFT postoperatively] [15].

Extensive research has been done on the NSAID nepafenac, which is notable for effective penetration and bioavailability in the eye [16]. In a study by El Gharbawy, 100 patients underwent cataract surgery (20 eyes of diabetic patients, 20 uveitic eyes, 20 traumatic cataracts, 20 glaucomatous eyes on topical PGAs, and 20 eyes with posterior capsular rupture during phacoemulsification) [17]. They were randomly assigned either postoperative topical steroids alone (Group A) or both steroidal and nonsteroidal anti-inflammatory eye drops (Group B) [17]. Results show that there was an increase in postoperative central foveal thickness compared to preoperative ($60.9 \pm 87.95\,\mu$ in group A and $25.52 \pm 57.26\,\mu$ in group B) and a significant difference in postoperative macular thickness between the two groups ($280.1 \pm 86.0\,\mu$ and $246.80 \pm 57.73\,\mu$, respectively, in groups A and B), which was used to evaluate the incidence of PCME [17]. However, no difference was reported preoperatively or postoperatively in corrected distance visual acuity and intraocular pressure (IOP). The researchers suggest that the addition of nepafenac reduces the incidence of PCME [17].

Another study by Sarkar *et al.* [18] researched whether prednisolone acetate is needed adjunctive with nepafenac to treat inflammation postoperatively. Group A received 0.1% nepafenac eye drops 4 times/day for 4 weeks, and group B received 1% prednisolone acetate eye drops in tapering doses for 4 weeks after surgery [18]. The researchers found that the difference in mean central macular thickness between the groups was significant (205.713 ± 17.14 vs. 220.984 ± 32.83 in groups A and B, respectively, $P \leq 0.001$) at 1 month [18]. Also, the mean pain score was significantly lower ($P = 0.018$) in the nepafenac group on day 7 of surgery [18]. This suggests that nepafenac is an adequate treatment for postsurgical inflammation and does not require prednisolone [18]. This study and the previous study both show that NSAIDs could reduce the CMT and potentially aid in the prevention of PCME [18].

Furthermore, in a study by Danni *et al.* [19], patients ($n = 103$) were randomly assigned either no preoperative anti-inflammatory medication or to receive preoperative topical anti-inflammatory medication with a combination of prednisolone acetate and nepafenac. However, both groups received 3 weeks of the combination drops postoperatively [19]. This study found that a lack of preoperative anti-inflammatory treatment does not impact the recovery of diabetic patients with an increased risk of PCME if they are treated postoperatively with prednisolone acetate and nepafenac [19]. At day 28 and month 3, patients from both study groups did not differ significantly in central subfield macular thickness (CSMT) and none of the eyes developed PCME [19]. This suggests that preventive measures are not necessary if treating patients postoperatively with this therapeutic combo [19].

While typical postoperative care includes an NSAID or corticosteroid, there is less research done on the combination of these therapeutics to prevent PCME. In a published phase 4 clinical trial [20], patients were randomized to one of the following treatment plans: prednisolone and ketorolac drops given either 3 days preoperatively (control) or the day of surgery, ketorolac alone pre or postoperatively, or a drop-less regimen where an intraoperative sub-tenon injection of dexamethasone was administered. The results found that flare increased significantly in the drop-less group compared to the control group, but the other study groups lacked a significant difference from the control group [20]. IOP decreased in all groups, but there was a significantly less decrease in the IOP within the

Pred+NSAID-Pre and Pred+NSAID-Post groups when compared to the NSAID monotherapy and drop-less groups [20]. There were no differences in visual acuity when compared to the control group [20]. These results suggest that a combination of monotherapy with NSAIDs may be preferred, and the preoperative interventions are not as significant [20]. These results are corroborated by findings from a study by Wielders *et al.* [21] where patients treated with topical bromfenac and dexamethasone had a lower risk of developing PCME after cataract surgery than patients treated with a single drug. While NSAIDs are currently known to help treat PCME [22,23], these findings suggest NSAIDs can also be used to prevent PCME. Table 1 summarizes NSAID therapeutics as well as other interventions and clinical trials that are discussed below.

CORTICOSTEROIDS

While postoperative topical corticosteroids like prednisolone acetate 1% are considered standard care for cataract surgery to prevent PCME, research is being done to improve current steroids as well as minimize the amount of patient compliance necessary [24]. For instance, a study by Lindholm in 2019 compared outcomes between 50 patients treated with dexamethasone eye drops (DEX) and 51 patients that received a perioperative subconjunctival injection of triamcinolone acetonide [25]. In DEX-treated eyes at 7, 28, and 90 days, CRT increased while CRT did not increase in the triamcinolone acetonide treatment group [25]. At 90 days, aqueous flare increases were persistently higher in the DEX group [25]. Changes in CDVA and IOP were comparable and well tolerated [25]. Three eyes in the DEX group developed PCME by the 90-day follow up compared to zero in the triamcinolone acetonide group, however, this was not found to be significant ($P = 0.118$) [25]. This suggests that the perioperative triamcinolone acetonide injection was just as effective in preventing postcataract macular edema as the routine dexamethasone eye drops [25]. Transitioning from eye drops to injections postsurgery can help minimize the necessity of patient compliance and assist those patients who are unable to use droppers.

Another improvement with current treatments is the potential to treat chronic and persistent PCME. In a study by Marques *et al.* [24], patients with recurrent PCME previously treated with two intravitreal injections of a corticosteroid (triamcinolone and/or dexamethasone implant) before the study were administered a single intravitreal injection of fluocinolone acetonide (FAc) intravitreal implant. They found that in the five eyes included in the study, BCVA with FAc implant either remained stable or improved [24]. The CMT decreased in all eyes until 24 months and was maintained in three of the five eyes with mild IOP elevations controlled with drops [24]. This suggests that in chronic PCME, intravenous fluorescein angiography (FA) can be used as an alternative treatment [24]. However, as the sample size was five eyes and retrospective, the authors do state that further research is required in using FAc implants [24]. A study by Momenaei *et al.* [26] found that suprachoroidal triamcinolone acetonide (XIPERE) for PCME significantly decreased central foveal thickness and improved visual acuity.

Wong *et al.* [27] studied a liposomal drug delivery system as a single subconjunctival injection or liposomal prednisolone phosphate intraoperatively. Laser flare photometry showed inflammation peaking at 1-week post cataract surgery and resolved to baseline within 2 months [27]. However, the sample size was small ($n = 5$), none of the patients developed PCME, and there was no comparison group to accurately determine if this is an effective system to replace eye drops [27].

Rodrigues *et al.* [28▪] recently compared BCVA and CFT at 12 months in nondiabetic and diabetic patients without diabetic retinopathy (DR) treated with a DEX implant for PCME after being previously treated with topical nepafenac 0.1% and prednisolone 1%. Researchers saw an increase in BCVA by 6 months and maintained until 12 months of the study and a reduction in CFT in both groups [28▪]. This study shows that nondiabetic and diabetic patients benefit from a dexamethasone implant for 12 months after topical treatments for refractory PCME.

Investigators of the LEOPARD clinical trial are recruiting patients to evaluate the safety and efficacy for OCS-01, a new steroid drop, which may treat PCME and/or uveitic macular edema [29]. A similar study is evaluating OCS-01 against placebo [30]. Compared to the placebo group, the OCS-01 treated group experienced significantly less inflammation (57.2 vs. 24%) and pain (75.5 vs. 52%) with a negligible amount of change in IOP [31]. However, further studies need to be conducted on the incidence of PCME for these drops.

ANTI-VASCULAR ENDOTHELIAL GROWTH FACTOR MEDICATIONS

Anti-VEGF therapeutics have been recognized and introduced for PCME care [32,33] and have also been shown to be effective at preventing refractory and recalcitrant PCME [34,35▪]. However, further research must be done on the safety and efficacy. In the PROMISE trial, patients with diabetic retinopathy were administered intravitreal aflibercept (IVA) to prevent PCME [36]. The results revealed the group that

Table 1. Treatment modalities for PCME

Therapeutic class	Treatment	Study name/Clinical study number	Description	Route
NSAIDs	Bromfenac	Jukić *et al.* [15] Wielders *et al.* [21]	Compared with dexamethasone, did not significantly affect IL-6 concentrations or reduce CFT.	Topical
	Nepafenac	El Gharbawy *et al.* [17] Sarkar *et al.* [18] Danni *et al.* [19]	Effective in reducing incidence of PCME and CMT postoperatively, better when combined with steroids.	Topical
	Ketorolac	Erichsen *et al.* [20]	Combination with prednisolone postsurgery is effective to prevent PCME.	Topical
Corticosteroids	Dexamethasone	Lindholm *et al.* [25] Marques *et al.* [24] Rodrigues *et al.* [28▪]	Commonly used postoperatively; newer methods of administration like implants are being studied.	Topical/ Subconjunctival
	Triamcinolone acetonide (TA)	Lindholm *et al.* [25] Momenaei *et al.* [26]	Effective in preventing PCME when administered perioperatively via injection.	Subconjunctival
	Fluocinolone acetonide (FAc)	Marques *et al.* [24]	Used as an intravitreal implant for chronic PCME.	Intravitreal
	Prednisolone acetate	Sarkar *et al.* [18]	Used in combination with NSAIDs; also studied for monotherapy postoperatively.	Topical
	Liposomal prednisolone phosphate	Wong *et al.* [27]	Studied as a single subconjunctival injection; further research needed.	Subconjunctival
	OCS-01	LEOPARD NCT05147233	New steroid being studied for efficacy but found reduced inflammation and pain.	Topical
Anti-VEGFs	Aflibercept	Song *et al.* [36]	Effective in preventing refractory PCME; similar incidences of AEs compared to control in trials.	Intravitreal
	Bevacizumab	Karasu *et al.* [38▪▪] Akay [39]	Found to resolve CME faster than other VEGFs in some studies.	Intravitreal
	Ranibizumab	Karasu *et al.* [38▪▪] Akay [39]	Used in studies comparing its efficacy against other VEGFs and dexamethasone implant.	Intravitreal
Prostaglandins	Latanoprost	Fakhraie *et al.* [42] Niyadurupola *et al.* [40]	Impact on PCME is minimal as CMT changes were not statistically significant.	Topical
Other investigational Therapeutics	Interferon α2b	Kawali *et al.* [45] Aghaei *et al.* [46]	Studied for treating PCME resistant to NSAIDs and steroids.	Topical; Subconjunctival
	Acetazolamide	Jorge *et al.* [47]	Implant studied for PCME in cases where oral administration failed.	Intravitreal
Laser	Subthreshold micropulse yellow laser	Verdina *et al.* [49]	Laser with higher absorption rate in O_2 hemoglobin that prevents foveal damage	Laser

received the intravitreal aflibercept injection showed less instances of macular edema (ME) at day 14, but differences were less prominent by day 90 [36]. Similarly, the control group had a more noticeable increase in CST in the first 60 days, but the difference was not significant by day 90 [36]. For visual acuity, both groups resulted with similar improvements in BCVA by the end of the study [36]. A clinical trial was registered to study subconjunctival aflibercept for macular edema treatment after cataract surgery [37], but this study was terminated.

In a study comparing IVA, bevacizumab (IVB), ranibizumab (IVR), and dexamethasone implant (IVDI) in the treatment of serous retinal detachment (SRD) caused by Irvine-Gass syndrome (IGS) in non-diabetic patients, all agents were effective, but eyes treated with IVA and IVDI required fewer injections [38▪▪]. SRD entirely resolved in all eyes in the IVA group at the final follow-up visit [38▪▪]. They also found that IVA had the fastest resolution of CME ($P = 0.032$) [38▪▪]. Another study showed that between bevacizumab, ranibizumab, and aflibercept,

the resolution of CME was the fastest in the bevacizumab group, $P = 0.031$ [39].

DISCONTINUING PROSTAGLANDIN ANALOGS

Prostaglandin analogs are a vital treatment for glaucoma but can have side effects including macular edema and uveitis. A clinical trial completed and published by Niyadurupola *et al.* [40] looked at 62 eyes of 62 participants with ocular hypertension or primary open-angle glaucoma (POAG) treated with PGAs prior to cataract surgery [41]. These patients were randomly assigned to either continue the PGA therapy or discontinue the therapy [40]. The PCME incidence was identical in both groups at 12.9% (four of 31 eyes) at the 1-month postoperative visit [odds ratio (OR) 1.000; 95% confidence interval (95% CI) 0.227–4.415] [40]. Another study by Fakhraie *et al.* [42] studied glaucoma patients who either discontinued or continued latanoprost. The latanoprost group had a temporary increase in the central macular thickness (CMT) at 1 month that returned to normal by 3 months [42]. The CMT changes between the groups were not statistically different at either time point [42]. At postoperative month 1, both groups had the same incidence of CME and lower IOP in the CPGA group than baseline IOP [42]. These findings suggest that PGA therapy has a minimal impact on PCME. However, there is much more research on PGAs increasing the chance of PCME [4,43,44].

OTHER INVESTIGATIONAL TREATMENTS

In a prospective, interventional case series of patients with PCME, eight patients were treated with topical interferon (IFN) therapy with regular monitoring for resolution [45]. This took about 5 weeks on average, though three patients had recurrences that improved with restarting treatment. One patient did experience papillary conjunctivitis, but there were no other adverse effects reported in relationship to topical IFN [45]. In a different case study, a 60-year-old patient with PCME unresponsive to multiple courses of topical NSAIDs and steroids was treated with subconjunctival interferon α2b injections [46]. After four injections, the CME resolved without evidence of recurrence or reported side effects in 6 months [46].

In a case study of a 54-year-old woman with PCME, oral acetazolamide was unable to treat the condition [47]. However, there was complete resolution of her symptoms 4 weeks after a single intravitreal injection of an acetazolamide implant (260 μg), suggesting the acetazolamide implants could be a potential therapeutic in cases of unsuccessful treatment [47].

A recent case study series found that two patients were diagnosed with a late presentation of bilateral PCME and a common correlation is the use of tyrosine kinase inhibitors, suggesting more research is needed on the effects of these medications on PCME [48▪].

LASER AND SURGICAL APPROACHES

A retrospective study by Verdina *et al.* [49] looked at subthreshold micropulse yellow laser (SMYL) as a potential therapeutic for refractory PCME in patients with complicated cataract surgery ($n = 2$), uncomplicated cataract surgery ($n = 5$), and retinal detachment ($n = 3$). SMYL has been studied for diabetic macular edema and retinal vein occlusion, due to a hypothetical better yellow laser absorption rate for O_2 Hb that prevents foveal damage; however, this procedure not been studied thoroughly for PCME [50]. After one session, all but two patients (one complicated and one retinal detachment) had significant improvements in best corrected visual acuity and central macular thickness with the best improvements in the retinal detachment group [49]. The two patients required two or three laser sessions for full resolution of PCME. Further larger studies are needed to better understand the role of SMYL in PCME. Current surgical treatments for PCME include laser vitreolysis [13] and pars plana vitrectomy [14], to release vitreous incarceration in the cataract incision wound or address any vitreous prolapse or traction that may be contributing to PCME.

CONCLUSION

Current and ongoing trials investigating the prevention and treatment of PCME focus on novel drugs, improved drug combinations, and advanced delivery systems. These trials reflect a significant shift toward minimizing the burden of postoperative drops, emphasizing preoperative and intraoperative strategies to reduce inflammation. The exploration of anti-inflammatory agents, VEGF inhibitors, and corticosteroid-sparing regimens demonstrates progress in achieving effective, long-lasting management with fewer side effects. Additionally, sustained-release implants and innovative drug delivery systems hold promises for simplifying treatment regimens.

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Conflicts of interest

Jessica Randolph has received consultant fees from Abbvie, Regeneron, Appellis, and Advarra.

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