Commentary

Cystoid macular edema (CME) continues to be an important complication after cataract surgery and other ophthalmological interventions. Indeed, this collateral effect represents the main cause of decreased visual acuity after phacoemulsification, even in uneventful surgeries and in patients without additional risks factors [1]. Although the frequency of clinically significant CME may vary from 0.1% to 3% among patients without risk factors, some clinical trials reported up to 9% of increased mean foveal thickness, as measured by Optical Coherence Tomography (OCT) and Angiographic macular edema [1,2]. Surgical complications like posterior capsule rupture with vitreous loss and retained lens material, as well as systemic comorbidities with high vasoactive profile (diabetic retinopathy, uveitis) may increase the incidence of clinically significant CME up to 20%.2 Albeit both steroids and non-steroidal anti-inflammatory drugs (NSAIDs) have been proposed to treat and prevent the occurrence of CME after phacoemulsification, their actual benefit remains to be fully elucidated. Therefore, an effective strategy to prevent CME after phacoemulsification with IOL implantation is required.

Although the exact pathogenesis of CME remains unclear, it is known that it represents the end of the pathway of the inflammatory cascade triggered by any intraocular surgery [3]. In particular, phacoemulsification acts as a thermal, chemical and mechanical stimulus into the eye, generating a wide range of inflammatory responses with the formation of prostaglandins (PG) from arachidonic acid, leading to the blood aqueous barrier breakdown and cystic accumulation of extracellular intraretinal fluid [1,3].

The control of inflammation within the eye is mandatory to keep the ocular integrity and function. The inflammatory cascade can be blocked at different locations using different therapeutic agents. Corticosteroids can effectively reduce PG and leukotriene synthesis by inhibiting the phospholipase A2 activity and blocking both the cyclooxygenase (COX) and lipooxygenase pathways. On the other hand, NSAIDs inhibit the 2 forms of the cyclooxygenase (COX) enzymes, COX-1 and COX-2 and prevent the production of endoperoxides, mainly prostaglandins (i.e., G2, COX-H2) and their downstream inflammatory effects [4]. The synergistic effect of both steroids and NSAID has been widely demonstrated in the literature, making this option the best way to prevent and treat postoperative CME.

There is no general consensus about the routine use of topical medication for preventing CME after cataract surgery. Although the perioperative administration of NSAIDs has been shown to reduce the incidence of CME after routine cataract surgery with IOL implantation, the prophylactic use of this medication among uneventful surgeries and in patients without risk factors is still under debate [4,5]. Opponents of this therapy stand out the relatively low incidence of clinically significant CME and the transient duration of the visual impairment. Even more, recent studies suggest that final best corrected visual acuity (BCVA) at 3 months or more after cataract surgery is similar among patients who received NSAIDs and those who did not received it [1]. Nevertheless, despite there are no significant differences in BCVA at 3 months, patients who received prophylactic NSAIDs showed faster visual acuity recovery after surgery, a key element considering that rapid recovery of the level of vision needed for early return to society is a common and significant interest of all patients having cataract surgery nowadays [5].

Patients with risk factors for CME can benefit of the prophylactic use of the prophylactic use of NSAIDs before and immediately after surgery. Main risk factors for clinically significant CME include previous uveitis, posterior capsule rupture with vitreous loss, retained lens material, diabetic retinopathy, venous occlusive disease, epiiretinal membrane, prior vitreoretinal surgery, nanophthalmos,
retinitis pigmentosa, radiation retinopathy, male gender, older age, and a history of pseudophakic CME in the fellow eye [2]. Thus, patients scheduled for cataract surgery that present any of these conditions should be listed to receive a prophylactic scheme with NSAIDs before and after surgery. Although there is no unique prophylactic scheme for these cases, it seems reasonable start with NSAIDs the day before surgery and for 4 weeks after the procedure. It is also advisable to administer one drop every 15 min in the hour prior to surgery so as to obtain better anti-inflammatory efficacy [4].

Preventing retinal swelling after cataract surgery in diabetic patients is a challenging situation. It has been demonstrated that up to 55% of diabetic patients develop significant increases in central retinal thickness measured by OCT after uncomplicated phacoemulsification [6]. This can be observed even in patients with stable mild diabetic retinopathy without macular edema prior to surgery. In such cases, new strategies are being tested in addition to the routine use of perioperative steroids and NSAIDs. Intraoperative injections of anti-vascular endothelial growth factor (anti-VEGF) with both Ranibizumab and Bevacizumab demonstrated to effectively reduce the incidence of CME after cataract surgery in diabetic patients. Even more, diabetic patients who developed macular edema following surgery showed significant decrease in retinal thickness after receiving intravitreal anti-VEGF injections at 1 and 3 months post-operatively [7,8]. Anti-VEGF injections including Ranibizumab, Bevacizumab and Aflibercept had also been considered as a novel treatment option for recurrent Irvine-Gass syndrome [9,10]. In addition, intravitreal dexamethasone implants also demonstrated efficacy for treating refractory pseudophakic macular edema with meaningful improvements in visual acuity that persist through 6 months. Further comparative longer-term trials are needed to evaluate the efficacy and safety of this alternative treatment option [11].

In conclusion, perioperative use of NSAIDs associated with corticosteroids can effectively reduce de incidence of CME after uneventful cataract surgery and should be administered as part of the routine therapeutic scheme. An additional benefit of NSAIDs use is observed in patients with risk factors for CME and in complicated cataract surgery. Special considerations should be made in patients with diabetic retinopathy, since encouraging results have been recently observed with the use of intravitreal anti-VEGF to prevent and treat macular edema after phacoemulsification. However, further studies are needed to determine their true usefulness and possible applications.

References