Residual triamcinolone acetonide at macular hole after vitreous surgery

Dear Editor,
We read with interest the article, “Residual triamcinolone acetonide at macular hole after vitreous surgery” by Kumar
et al., We would like to congratulate the authors and share our experience and views regarding this issue.

A six-year-old child who presented to us with complaints of decreased visual acuity in his right eye following trauma with a ball, was found to have a full thickness macular hole (MH). His best corrected visual acuity (BCVA) was 20/200, with no other signs of blunt trauma. The patient underwent standard three port pars plana vitrectomy and internal limiting membrane (ILM) peeling under general anesthesia. After posterior vitreous detachment (PVD) induction with the help of preservative-free triamcinolone acetonide (TA) subretinal migration of TA was noted [Fig 1a]. We aspirated the excess TA over the retinal surface and proceeded with peeling of the ILM after staining it with brilliant blue dye [Fig 1a]. Fluid air exchange was performed and 20% SF6 gas was injected into the vitreous cavity. The patient was advised prone positioning for three days. At two weeks of follow up, the MH failed to close and showed a large area of retinal pigment epithelial (RPE) atrophy around it [Fig 1b]. The visual acuity did not improve beyond the preoperative level. The MH persisted till four months of follow up.

Subretinal migration of TA during MH surgery and deposition in the area of the hole postoperatively has been described in literature. Even as most reports conclude that this does not adversely affect the anatomical and functional outcome of the surgery,[1-3] there is a single case report suggesting that the presence of TA crystals in a hole may prevent its complete closure.[4] Our case demonstrates that subretinal migration of TA during MH surgery may interfere with hole closure despite successful ILM peeling and result in RPE atrophy. We did not try to remove the residual TA in the MH completely during surgery by any active surgical maneuvers because of the risk of damaging the RPE on the floor of the MH, which is known to hinder visual recovery after hole closure.[3]

The above-mentioned issues can be dealt with by using autologous heparinized whole blood to cover the macular area prior to administering TA. Whole blood acts as a mechanical barrier, preventing the migration or deposition of TA in the hole and ameliorates any effect of TA on RPE function or closure of the hole. Autologous whole blood and serum have been used previously for MH closure with varied functional results. Thus, in addition, whole blood can act as an adjuvant agent or biological glue. It can be easily obtained during surgery from a peripheral vein and is an economic and non-toxic intraoperative tool.[5] This technique can also prevent the potential toxicity of other dyes used to stain the ILM on the bare RPE in the area of the hole.

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Dear Editor,

I read with great interest, the article by Venkatakrishnan et al., 'Peribulbar anesthesia for cataract surgery: Effect of lidocaine warming and alkalinization on injection pain, motor, and sensory nerve blockade.' They observed that alkalinization of lidocaine results in the least painful injection in peribulbar anesthesia and could be the best option for anesthesia in cataract surgery. I agree with the author's observations.

I would like to share my experience regarding alkalinization of lidocaine with sodium bicarbonate, which I have been practicing for the last few years when performing surgery on an inflamed eye. Few common indications in adults are evisceration, enucleation, and therapeutic keratoplasty, with or without anterior vitrectomy, when the eye is significantly inflamed because of invading infection. Generally, these surgeries are preferably performed under general anesthesia or under local anesthesia with good sedation.

Earlier when the procedure was being performed with local anesthesia plus sedation, many of my patients became uncomfortable and non-cooperative within the first few minutes due to pain and more often needed supplemental anesthesia. Postoperative headache, nausea, and vomiting were also often noticed.

Later, for giving blocks, I used a mixture containing lidocaine with adrenaline (5 cc) plus bupivacaine (4cc) plus hyaluronidase and sodium bicarbonate (1cc), with pH maintained between 6.5 and 7. I noticed that the duration of anesthesia lasted for more than one hour in most of our patients, and there was no need for supplemental sedation. This was particularly useful in those who were unfit or unwilling for general anesthesia. Therefore, alkalinization of the anesthetic solution sounds more beneficial for surgeries of the inflamed eye, to avoid unwanted effects of excessive sedation or possible conversion to general anesthesia.

It may be logically stated that highly inflamed tissue has acidic pH due to secretion of arachidonic acid and its derivatives. Alkalinization of injectable solution neutralizes the pH of the tissue. In alkaline pH, the efficacy of hyaluronidase also increases.

However, I must admit that our experience is limited, with fewer surgeries as compared to other institutions, and would like the experts to share their thoughts. Further studies are required to validate our observation of using alkalinization in inflamed eye surgery.

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