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Sir,

Resolution of cystoid macular oedema after retinal detachment repair: is intravitreal triamcinolone useful?

Poor functional visual outcome after successful retinal detachment (RD) repair is mainly owing to photoreceptor cell apoptosis.¹ Clinical appearance of the macula in the majority of these patients is normal, but a proportion have anatomical changes observable clinically.² More recently, ocular coherence tomography (OCT) has helped in revealing residual subfoveal fluid accumulation not visible clinically or on fluorescein angiography,³ more frequent after scleral surgery.⁴ OCT has also been helpful in showing postoperative cystoid macular oedema (CMO), responsive for the limited visual improvement in a small number of patients. The exact etiopathogenic mechanism of CMO after RD is unclear, but ocular inflammation may play a role, especially after the trauma of cryotherapy, scleral buckle, and subretinal fluid (SRF) drainage. CMO incidence, in phakic eyes, has been reported around 25–30% after cryotherapy and scleral explant,⁵ and 8% after pneumatic retinopexy.⁶ It has not been related to preoperative macular status or duration of the RD.⁷ CMO spontaneous resolution has been described in up to 76% of cases,⁵ within 2 years postoperatively. Other series, though, report a more bleak evolution of the condition.⁸ Different treatments have been described, but none with great success. Topical and systemic non-steroidal anti-inflammatory drugs and steroids have been used. There are anecdotic reports of response to acetazolamide.⁹

A 78-year-old male presented to our Primary Care Department with complaints of floaters for the past week, and decreased central vision for the last 48 h. He underwent uneventful phacoemulsification and posterior chamber intraocular lens implant 3 years ago, but otherwise he had no other past ocular history. On examination, visual acuity was 6/36 and fundoscopy showed superotemporal bullous RD involving his macula, for which he underwent pars plana vitrectomy, cryoretinopexy, and 12% C₃F₈ gas endotamponade.

Immediate postoperative period was anodyne, with complete flattening of the retina. At 4 months after successful repair, though, best-corrected visual acuity (BCVA) was 6/18. Anterior segment was quiet with pseudophakia and fundoscopy revealed cystoid macular changes. OCT confirmed this clinical finding and also showed no presence of residual SRF (Figure 1). The patient was started on topical ketorolac and prednisolone, and 1 month later received 4 mg intravitreal triamcinolone acetate (IVTA). OCT performed 2, 6, and 12 months later, still showed cystoid spaces in the macula and BCVA remained unchanged (Figure 2). At 15 months after IVTA, OCT showed total resolution of the CMO (Figure 3). Visual acuity, at this stage, had returned to pre-RD levels, with BCVA of 6/6.

There are several reports of successful treatment of uveitic, pseudophakic, and diabetic CMO with IVTA. However, no reference to CMO after RD treated with IVTA could be found in a PubMed literature search. There is a rationale for using steroids in its management, as increased prostaglandin levels in the retina and uvea and increased prostacyclin and thromboxane A₂ derivatives in the SRF have been reported.¹⁰ All these mediators increase perifoveal microvascular permeability by disrupting capillary tight junctions,

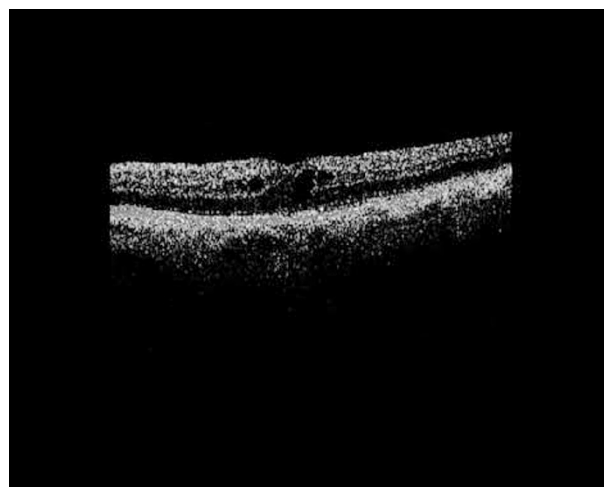


Figure 1 OCT scan 4 months after successful retinal detachment repair, showing cystoid macular oedema.

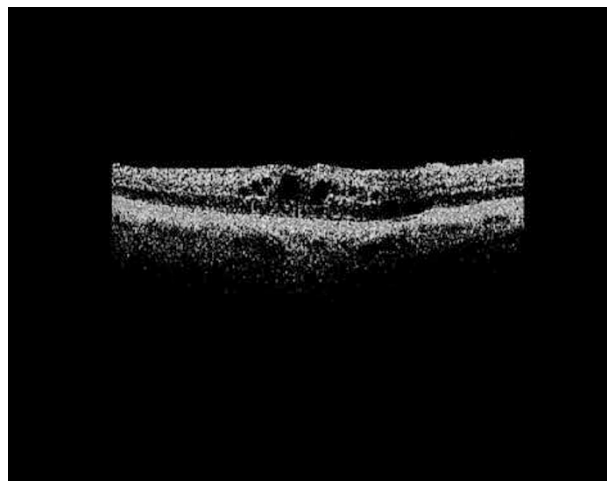


Figure 2 At 2 months after intravitreal triamcinolone injection, OCT still reveals cystoid changes.

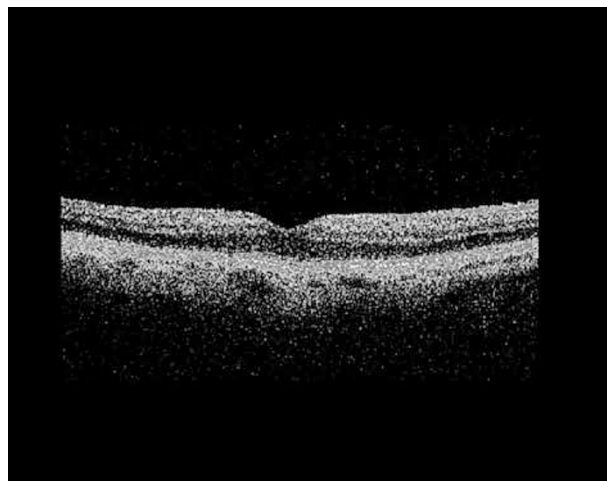


Figure 3 Resolution of the cystoid oedema and restoration of normal foveal morphology, 15 months after IVTA.

producing the breakdown of the blood–retinal barrier (BRB), and subsequent oedema.¹¹ Steroids inhibit phospholipase A₂ and reduce levels of proinflammatory cytokines, increasing BRB function.¹²

The late visual improvement observed in our patient is unlikely to be explained by IVTA effect, with an expected action duration of 3 months or less in the vitrectomised eye.¹³ With the current increase in use of IVTA for different eye conditions, many unresolving CMO after RD will be treated with IVTA. A randomised control trial (RCT) would have, therefore, its role in elucidating the usefulness of the drug in such cases. Potential side effects and complications of IVTA, such as endophthalmitis, cataract formation, and ocular hypertension, should be then weighed up in a treatment arm of a RCT for a condition that might, like in the case we present, resolve spontaneously in the long term.

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