Resolution of cystoid macular edema by topical dorzolamide in a case of central serous chorioretinopathy: a case report

Debmalya Das and Eesh Nigam

Introduction
Central serous chorioretinopathy (CSCR) is a posterior segment disease characterized by localized serous detachments of the neurosensory retina often associated with focal detachments of an altered retinal pigment epithelium (RPE) and having multifactorial etiology and complex pathogenesis. In chronic CSCR, intraretinal cysts and cystoid macular edema (CME) can form. Both topical and systemic carbonic anhydrase inhibitors (CAIs) have been tried as means of treatment of both CSCR and CME caused by CSCR. We present a case of chronic CSCR in which the resolution of CME occurred with topical CAI.

Case report
A 45-year-old male presented with painless, gradual and progressive diminution of vision of both eyes for 2 years. On examination, his best corrected distant visual acuity (BCVA) was 20/120 in the right eye and 20/200 in the left eye. Posterior segment examination revealed extensive areas of RPE atrophy in both eyes with the presence of subretinal fluid (SRF) in the right eye. Fundus fluorescein angiography corroborated the clinical findings showing extensive areas of RPE atrophy in both eyes with ink blot leak in the right eye. Optical coherence tomography (OCT) of the right eye showed sub- and juxtafoveal RPE atrophy with SRF and the left eye revealed juxtafoveal cystic changes with RPE atrophy (Figure 1). Focal laser to the area of leak in the right eye was done and the patient was asked to review after 2 months. On his next visit, BCVA of right eye increased to 20/80 with no change in the left eye BCVA. OCT showed resolution of SRF in the right eye and an increase in CME and schisis in the left eye (Figure 2). The patient was started on topical dorzolamide (2%) thrice a day in his left eye. On his next visit, after 1 month, OCT showed complete resolution of CME though the visual acuity remained the same (Figure 3).

Discussion
One of the characteristics of acute CSCR is that despite SRF, the morphology of retinal layers generally remains unchanged. However, in chronic cases, intraretinal cysts and CME may develop. These may disappear or fluctuate slowly over time, suggesting fluid passage through a compromised RPE which contributes to their formation. However, in our case, CME resolution occurred within a month, suggesting a possible role of dorzolamide in its resolution.

Investigations of the ability of CAIs to enhance SRF absorption based on animal models have shown acidification of the subretinal space, a decrease in standing potential, and an increase in retinal adhesiveness. This acidification of the subretinal space is responsible for the increased fluid resorption from the retina through the RPE to the choroid resulting from modulation of carbonic anhydrase IV in RPE. RPE loses normal polarity in the presence of macular edema, and treatment with CAIs re-establishes normal polarization in RPE. Another possible explanation for the effect of CAIs on inflammation-related macular edema is its ability to inhibit γ glutamyl transpeptidase.

Figure 1. OCT through the fovea of the left eye showed juxtafoveal cystic changes (white arrow) with areas of RPE atrophy (white arrow head).
peptidase activity in ocular tissues. This facilitates cellular adhesion, neutrophil chemotaxis and degradation through elevation of leukotriene D4 concentration. A similar cellular mechanism may influence CSCR-related CME and may contribute to its resolution, as was seen in our case. Reports have also shown that CAI has no effect on the final BCVA as was also seen in our case.

One major drawback of our case is that it is a single case report with no long-term follow-up. Also, to date, only one case series, showing the efficacy of systemic CAI in CSCR, has been published. Although small case reports on topical CAIs in CSCR have been reported, few have highlighted the effect of CAIs on CME due to CSCR. Our case report adds to the knowledge that topical CAIs are as effective as oral CAIs in the resolution of CME due to CSCR, but has no effect on the final visual acuity.

References


Figure 2. OCT showed increased cystic and schitic changes (black arrow with white border).

Figure 3. OCT showed resolution of cystoid macular edema (white arrow with black border) with foveal thinning and RPE atrophy.