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Spontaneous resolution of an inflammation-associated epiretinal membrane with previously documented posterior vitreous detachment

Although resolution of idiopathic epiretinal membranes (ERM) with a subsequent posterior vitreous detachment (PVD) has been reported, we report a rare case of resolution of an inflammation-associated ERM in a young patient that was associated with a pre-existing PVD.^{1–3}

Case report

A 16-year-old white man with a history of intermediate uveitis and negative systemic work-up was seen at the Barnes Retina Institute for marked distortion and decreased visual acuity. Visual acuity was 20/20 in the unaffected right eye, 20/70 with marked distortion in the left eye. Fundus examination of the left eye revealed a previously noted Weiss ring, and a distinct cellular epiretinal membrane (ERM) with associated retinal thickening and distortion (fig 1A–E). Fluorescein angiography showed intraretinal leakage associated with the ERM (fig 1C). Treatment options including surgery were discussed but the patient elected to observe. Five months later, visual acuity was 20/25 in the left eye and Amsler grid testing showed no distortion. The ERM had spontaneously resolved with minimal retinal folds remaining (fig 2B); there was no leakage on fluorescein angiography (fig 2C); optical coherence tomography (OCT) showed significant resolution of neurosensory retinal thickening.

Comment

Without surgical intervention, ERM is typically a stable or slowly progressive condition.⁴ Resolution typically has been linked to the development of a posterior vitreous detachment (PVD). Spontaneous regression has been noted in the three instances in the English-speaking literature but with adequate colour depiction in only one instance.^{1–3} All previously published articles pertain to idiopathic macular pucker without a pre-existing PVD. Secondary ERM, unlike idiopathic ERM, are associated with pre-existing ocular disease such as diabetes, retinal vascular occlusion, vasculitis, trauma, vitreous haemorrhage or uveitis. Although the exact pathogenesis of ERM is not completely understood, they are composed of retinal pigment epithelial (RPE) cells, fibroblasts, vascular endothelial cells, monocytes and glial cells. Glial cells may be an important component of ERM as they produce several cytokines such as vascular endothelial growth factor (VEGF), growth factors such as fibroblast growth factor (FGF) and transcription factors as NK- κ B and AP-1 that might

Figure 1 Color fundus photograph (A) and high magnification of macula (D) demonstrates the ERM. Red-free photograph (B). Late-phase fluorescein angiogram shows significant ERM-associated leakage (C). OCT with arrow pointing to ERM (E).

stimulate inflammation and propagate the growth of secondary ERM.⁵ In our patient, a PVD with Weiss ring was noted at presentation in this uveitis-related ERM. To our knowledge, we present the first photographic documentation of spontaneous resolution of an inflammatory epiretinal membrane in the presence of a pre-existing PVD.

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Figure 2 Spontaneous resolution of ERM. Color fundus photograph (A). Red-free photograph (B). Late-phase fluorescein angiogram with no leakage (C). OCT with significant reduction of retinal edema (D).

Pressure dependent stromal oedema following deep anterior lamellar keratoplasty

A 53 year old man with keratoconus and a steroid related intraocular pressure response developed acute hydrops and underwent deep anterior lamellar keratoplasty after poor spontaneous recovery of vision. Intraocular pressure rose to 40 mm Hg on postoperative administration of topical prednisolone 0.5%, resulting in a well localised area of stromal oedema. Resolution of the oedema followed reduction of intraocular pressure, but recurrence ensued if the pressure rose above 30 mm Hg. Following acute hydrops, the regenerative process involves endothelial proliferation across the break in Descemet's membrane. This localised functional deficit is demonstrated after only a moderate intraocular pressure rise.

Case report

A 53 year old man was referred with acute hydrops in the left eye. He had a history of asthma, severe atopic keratoconjunctivitis, keratoconus, primary open angle glaucoma controlled on latanoprost eye drops, and a steroid related rise in intraocular pressure. A penetrating keratoplasty had been carried out on the right eye eight years earlier, after which the patient achieved 6/9 best-corrected visual acuity (BCVA). Over the next two months the hydrops resolved, leaving apical scarring and a large visible break in Descemet's membrane. His BCVA improved from counting fingers to 6/60. He was contact lens intolerant, and therefore underwent a left deep anterior lamellar keratoplasty (by the Melles technique¹).

Postoperatively he was given 0.5% prednisolone eye drops three times daily and on review four weeks postoperatively his intraocular pressure had risen to 40 mm Hg. There was localised stromal oedema and mild epithelial oedema overlying the area of the previous break in Descemet's membrane. His intraocular pressure was lowered to 14 mm Hg with acetazolamide, and the stromal oedema cleared. Topical latanoprost was stopped to help control an exacerbation of asthma and his intraocular pressure subsequently rose to 32 mm Hg. Once again there was a localised area of stromal oedema overlying the previous break in Descemet's membrane, which cleared as the intraocular pressure was lowered to 20 mm Hg by topical dorzolamide. At three months post-operatively his topical steroid was discontinued and his intraocular pressure remained at 10 mm Hg. Three years later his corneal stroma is clear and he achieves 6/9 BCVA with a spectacle correction following suture removal.

Comment

Clarity of the normal cornea is achieved by maintaining the corneal stroma in a relatively dehydrated state. The intraocular pressure and stromal imbibition pressure promote the accumulation of fluid in the stroma. These forces are balanced by the osmotic gradient generated by active solute transport across the corneal membranes, particularly the endothelium. It has been shown that if the endothelial function is normal, then increased intraocular pressure does not cause stromal oedema.²

In advanced keratoconus, hydrops results from rupture of Descemet's membrane. Histological studies have shown that the endothelium then spreads across the break in

Descemet's membrane and secretes basement membrane, restoring the normal anatomy over three to four months. This correlates with the resolution of the stromal oedema, suggesting that there is functional as well as anatomical recovery of the endothelial layer.³ The case presented here, however, provides evidence that the function of the regenerated endothelium and basement membrane remains impaired.

The endothelium in the region of the break in Descemet's membrane was able to maintain the clarity of the overlying corneal stroma when the intraocular pressure was normal. However, when the intraocular pressure was raised, the cornea became oedematous. We could find no earlier documentation of this effect, presumably because such cases have previously undergone penetrating keratoplasty, removing the area of re-endothelialised stroma.

In summary, this case highlights the residual functional impairment of the regenerated endothelium and basement membrane at the site of a break in Descemet's membrane. Localised stromal oedema occurred overlying this region with only a moderate increase in intraocular pressure.

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