Graefes Arch Clin Exp Ophthalmol (2011) 249:809–810
DOI 10.1007/s00417-011-1665-1

EDITORIAL

Is traction the cause or the effect?

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Received: 9 March 2011 / Accepted: 9 March 2011 / Published online: 12 April 2011

The role of vitreomacular traction in the pathogenesis of many macular conditions has generated much interest, not least because spectral-domain optical coherence tomography (SD-OCT) has enabled us to examine the vitreoretinal interface with higher resolution, in 3 dimensions and from different perspectives. In this issue, Martinez and Ophir add another condition, that of macular oedema following retinal vein occlusion, to the already long list of macular diseases in which vitreous plays a putative role [1] . In a retrospective and observational study, the authors found nine of 22 patients (five of 11 with CRVO and four of 11 with BRVO) had what they called extrafoveal traction.

There are many questions. When does an attachment of the vitreous to the retina become "adhesion", when does "adhesion" become "traction"? What is the significance of an association between the finding of an abnormal vitreoretinal interface and any given macular condition? Does the association imply cause or effect? Using OCT, in early idiopathic macular hole, the posterior hyaloid can be seen to be attached to the fovea, and often to the rim of the full-thickness macular hole. These findings are widely accepted as supportive evidence of the tractional aetiology of this condition [2] . In diabetic macular oedema, OCT can show vitreous attachment to the retina appearing as a taut posterior hyaloid membrane [3] . Many believe such membranes cause traction. Stefansson pointed out that "vitreoretinal traction decreases the tissue pressure, the hydrostatic pressure between the blood vessels and tissue compartment increases, this stimulates water flux from vessels to tissue and oedema formation" [4] . In a recent analysis of a series of internal limiting membranes (ILM) excised from eyes with diabetic macular oedema, we demonstrated using immunohistology the presence of glial and neural tissue on retinal and vitreal surfaces of the ILM as a clear indication of proliferative changes at the vitreoretinal interface. In the case of diabetic macular oedema, abnormalities of the vitreoretinal interface could be both the cause and the effect [5] .

The strength of the paper by Martinez and Ophir is the precise definition of traction. They used SD-OCT B-mode video clips and 3-D image reconstructions to carry out a meticulous survey of the vitreoretinal interface. They regarded it to be evidence of traction where there was "vitreous adherence to the retina or optic nerve head associated with, a) tissue elevation or thickening and deformity at the traction site, e.g., the shape of the inner retina at the exact site of traction changed its angle and thus was typically thicker than that of the adjoining oedematous retinal tissue, and b) the posterior hyaloid or vitreous strand terminates or changes its angle at that site". Conversely, vitreous adherence without traction related to eyes in which "the attachment of the vitreous was not associated with any inner retinal or papillary deformities of these tissues at that site".

The significance of their findings is two-fold. Firstly and unsurprisingly, the prevalence of vitreous traction is much higher using SD-OCT (and 3-D imaging) than previously reported using time-domain OCT [6] . Secondly and more importantly, treatment directed at removing the traction may reduce oedema and improve vision. In one study involving 26 patients with branch retinal vein occlusion, no posterior vitreous detachment (PVD) and macular oedema, Sakuma et al. injected one international unit of autologous plasmin 4 months after the onset of macular oedema [7] . They achieved a complete PVD in 23 of 26 patients, dramatic decrease in OCT thickness at the macula, and improvement of vision by 2 lines or more lines in 23 patients. Similarly, uncontrolled case series have demonstrated that vitrectomy with or without ILM peeling may improve the macular

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oedema and bring about improvement in vision [8] . Whether such improvement was entirely attributable to the removal of traction remained questionable. Williamson et al. measured the pO2 and demonstrated an increase after vitrectomy in patients with retinal vein occlusion [9] .

Again, if a parallel can be drawn with diabetic macular oedema, then the recent publication from the DRCR.net is encouraging [10]. In this prospective study of 87 eyes undergoing vitrectomy for DME associated with at least moderate visual loss and investigator-determined vitreomacular traction, the median change in visual acuity at 6 months was an improvement of 3 letters, with visual acuity improving by≥10 letters from baseline to 6 months in 38% (95% CI, 28%–49%) and worsening by≥10 letters in 22% (95% CI, 13%–31%). Reduction in OCT central subfield thickness to <250 microns occurred in almost half, and most eyes had a reduction of thickening of≥50%.

With the advent of anti-VEGF [11, 12] and sustained intravitreal steroid implant [13] , the relative indication for the release of traction is unclear. There may, however, be an important difference between macular oedema caused by diabetic retinopathy and retinal vein occlusion. Diabetic retinopathy has an insidious onset, whereas retinal vein occlusion has a clearly defined acute onset. If vitreoretinal traction is a both an effect and a cause of macular oedema, then the trend to treat retinal vein occlusions earlier using steroid or anti-VEGF might be a step in the right direction. Might earlier treatment prevent traction from developing in the first place?

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