evaluation of vitreoretinal adhesions in exudative AMD using optical coherence tomography

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Posterior vitreous detachment (PVD) is one of the most striking age-related changes in the human eye.

According to autopsy studies, PVD is present in fewer than 10% of persons younger than 50 years, but has been found in at least one eye in 27% of individuals aged 60 to 69 and in 63% of subjects aged 70 and older.

PVD is not acute but insidious, so that it occurs initially in the perifovea as a focal, shallow PVD, extends slowly as subclinical PVD for years without any visual symptoms, and eventually results in complete PVD on acute release of the vitreopapillary adhesion of the posterior vitreous face.
A. Vitreoretinal interface without PVD.

B. Posterior vitreous detachment initially occurs in the perifovea, with a predilection for the superior quadrant.

C. Detachment extends widely in the perifovea, with persistent attachment to the fovea and optic nerve head.

D. Detachment occurs in the fovea, with persistent attachment of the posterior vitreous face to the optic nerve head.

E. Detachment is completed in association with release of the vitreopapillary adhesion.
Stage 0, no PVD.

Stage 1, incomplete PVD in the temporal perifovea, with attachment to the fovea, optic nerve head, and midperipheral retina.

Stage 2, incomplete PVD in the temporal and nasal perifovea, with attachment to the fovea.

Stage 3, incomplete PVD over the posterior pole, with persistent attachment to the optic nerve head.

Stage 4 defined as complete PVD, with Weiss ring.

Anomalous PVD

Sebag recently proposed the concept of anomalous PVD where gel vitreous liquefaction without concurrent vitreoretinal dehiscence exerts traction on the retina. There are two distinct steps that occur: liquefaction, or synesis, and separation of the interface, or syneresis, and if that two-step process does not occur in proper synchrony and completion, anomalous PVD can result, leading to VMA.

This pathogenic mechanism is the initiating event in diseases such as retinal tears and detachments, macular holes and pucker, advanced proliferative diabetic vitreoretinopathy and exudative AMD.
EXUDATIVE AMD

The cause of exudative AMD is not fully understood. Genetic factors, oxidative stress, ischemia, aging of the retinal pigment epithelium, and inflammation are proposed etiologic factors.

Although vitreous has been studied extensively for other macular diseases like macular holes, and although the profound molecular and structural changes within the aging vitreous are known, the role of the vitreoretinal interface has not yet been examined sufficiently in the context of AMD.
A normal eye of an elder patient: the hyaloid membrane is visible and completely detached from the fovea, though there is a persistent adhesion to the optic nerve (right frame).

Eye with dry AMD and drusen: the hyaloid is attached over the entire macula, including the fovea. "no traction" configuration since no distortion is visible on the retinal surface and the angle of insertion of the hyaloid onto the retina is not steep.

Eye with choroidal neovascularization (CNV). The persistence of hyaloid adhesion causes VMT over the CNV complex: a focal distortion of the retinal profile is visible at the site of hyaloid attachment.

Shallow detachment of the posterior vitreous cortex in front of the lesion without any adhesion to the retina surface in a case of nonexudative AMD.

The posterior vitreous cortex (arrows) is attached in the area of the neovascular lesion surrounded by a shallow vitreoretinal separation.
Large amount of intraretinal fluid with cystic formation that persisted after three monthly intravitreal injections of avastin.

Same view after five months. A spontaneous release of the central VMT is detected by Spectral OCT.

The purpose of our study was to determine the frequency of persistent posterior hyaloid adherence to the posterior pole in patients with exudative AMD. A consecutive case series of 40 eyes with exudative AMD patients was included. The average age of patients was 72.12 years (SD ± 7.48).
Stage 0: No PVD. 15/40 (37.5%)

Stage 1: Beginning PVD. 7/40 (17.5%)
Stage 2 : VMT.10/40(25%)
Stage 3: detachment from the fovea. 3/40 (7.5%)

Stage 4: complete PVD. 5/40 (12.5%)
There may be more similarities between the vitreoretinopathy of diabetes and AMD than appreciated previously.

It is known that complete PVD is associated with a very low incidence of neovascularization in diabetes, whereas partial PVD is a significant risk factor for vascular proliferation.

The OCT findings suggests that a localized vitreous adhesion is associated with a high incidence of choroidal neovascularization.

A high incidence of posterior vitreous attachment was recently observed intra-operatively in patients with exudative AMD.

There is also some evidence that the treatment outcomes for exudative AMD are affected by posterior hyaloid adhesion. Chronic tractional forces might antagonize the effect of anti-VEGF treatment, resulting in a poor response to anti-VEGF treatment in patients with VMA.

The persistent adhesion of the hyaloid to the posterior pole, with or without VMT, is not able to induce AMD. It is more likely that the fundamental mechanism of AMD formation must have already begun for tractional forces to achieve a change for the worse.
Vitreomacular adhesion (VMA) would facilitate in fact the progression from nonexudative to exudative forms of AMD. The hypotheses are multiple:
- persistent vitreomacular adhesion might induce chronic low-grade inflammation,
- prevent normal oxygen and nutrients diffusion to the macula,
- and/or confine proangiogenic cytokines in the macula.

PVD may be protective against AMD, whereas VMA may promote exudative AMD.

The question that remains, however, is if CNV is already present, would inducing vitreous separation alleviate symptoms or enhance the effect of an anti-VEGF agent to decrease the number of injections needed?

Chemical vitreous manipulation may represent new frontier in the treatment of exudative AMD.