What's Detached? A Guide to the Management of the Patient with Flashes or Floaters

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Introduction

Flashes of light and/or floaters or spots are common presenting symptoms seen in optometric practice.

Adequate investigation and management is essential because although posterior vitreous detachment is common and commonly uncomplicated, it is also frequently associated with retinal breaks some of which lead to retinal detachment.

Direct ophthalmoscopy is not adequate to view the peripheral retina **with or without** a dilated pupil. As the vast majority of retinal breaks occur beyond the retinal equator it is essential to dilate the pupil and use indirect ophthalmoscopy whenever a patient presents with flashes or floaters.

Techniques for examining the vitreous and retina

Mydriasis

Use Tropicamide 0.5% or 1%. This will not only dilate the pupil but also abolish the light pupil reflex, essential when using bright sources of light to view fundus. Acute glaucoma is rare but one need to proceed with caution if the angle is so narrow as to predict an attack of acute glaucoma if the pupil is dilated.

Instrumentation

Optimum methods of examining the peripheral retina include head set BIO with scleral depressor or slit lamp microscope with mirrored contact lens. Neither technique is widely used by optometrists in the UK.

Whilst not as adequate as a head set BIO with scleral depression, the slit lamp biomicroscope in conjunction with BIO lens such as the Volk 90 D Superfield affords vastly better views of the peripheral retina than direct ophthalmoscopy. Further, according to the College of Optometrists *Clinical Practice Survey* (2001), 80% of respondent optometrists stated that they use slit lamp BIO in practice.

Anatomy of the peripheral fundus

Retina composed of inner neural (sensory layers) and outer pigment epithelium. Normal retina is transparent except for pigment in blood. Sensory retina thin and weak and susceptible to full thickness breaks.

RPE is a uni-layer of polygonal cells, uniform in size and pigment except at macular and vitreous base (vitreoretinal symphisis). Inner surface has microvilli giving loose attachment/bond to sensory retina. RPE is attached to basement

membrane which forms innermost layer of basal lamina (Bruch's membrane). Bruch's membrane bonds to choroid.

Equator of fundus is situated approximately 14 mm from limbus and is located ophthalmoscopically by finding vortex veins which drain blood from the region.

Ora serrata demarcates anterior limit of neural retina and has scalloped appearance. 2mm wide nasally and 1 mm temporally; situated 7-8 mm from limbus. Rounded extensions of pars plana into ora called *ora bays*.

Vitreous fills 2/3rds of globe and provides structural and metabolic support for retina. It consists of collagen type II. There is a depression posterior to lens called *patellar fossa* and Cloquet's canal traverses anterior/posterior. Vitreous base is 3-4 mm zone straddling pars plana.

There are vitreoretinal adhesions in normal eye. Cortical vitreous is loosely attached to ILM of sensory retina, strongly attached to vitreous base, fairly strongly attached to optic disc margin, weakly attached around the fovea, and weakly attached along peripheral blood vessels.

PVD

PVD is condition in which vitreous cortex separates from posterior retina and optic disc. If it extends to ora serrata it is termed a complete PVD and if only posterior region, it is termed an incomplete or partial PVD.

Prevalence

Statistics for prevalence vary considerably. Jones (1998) discusses this extensively. Examples of prevalence statistics are:

For adults of all ages is 2% incomplete : 12% complete
Prevalence > 65 years is 3% incomplete : 31% complete

Rare in under 30s. Aphakic eyes have a higher incidence as do high myopes. It is reportedly more common in females .
Trauma to the eye increases incidence e.g., boxers.

Clinical appearance

Seen as very thin and transparent membrane.. With biomicroscope it appears as a more dense transparent membrane in central portion of vitreous cavity. Posterior face of vitreous cortex contains particulates which scatter light and these move on eye movements.

Presence of avulsed pre-papillary gliotic ring (Weiss) is pathognomnic of PVD. It may appear as complete annulus or be broken. Because it is close to visual axis it is frequently seen by patient. Glial strands may be attached and seen by patients as spikes, threads or spider web. If patient is asked to move eye around then the glial tissue will move especially in presence of synchisis. This is sometimes known as ascension-descension phenomenon.

Other signs associated with PVD include “tobacco dust” (Shafer’s sign) which consist of tiny brown dots -pigment cells from the RPE. In presence of tobacco dust one must assume retinal tear until proven otherwise.

Pathophysiology

PVD usually commences over posterior pole. Synchisis (liquefaction) leads to formation of pockets of fluid called lacunae. Syneresis (physical contraction or shrinkage) occurs and vitreous cortex may collapse inferiorly.

Symptoms of PVD

Floaters

Floaters are most common and are variously described as cobwebs; hairnet; strings and come in many shapes and sizes. Floaters may be due to condensation of vitreous fibrils in cortex, glial tissue torn from epipapillary region or intravitreal blood from superficial vessels. They move about freely with detached vitreous. One or two longstanding floaters indicate vitreous condensation or a PVD annulus. These are generally benign symptom with no significant associated risk.

However, the report of a sudden onset of floaters calls for a dilated fundus examination without undue delay. 95% of patients > 50 years with sudden onset floaters have a PVD. Even a single floater may indicate a PVD annulus with a small possibility of a retinal tear.

Photopsia

Photopsia is also frequent symptom of PVD and occur through mechanical stimulation of retina by traction produced by detaching vitreous cortex. They are usually bright white and usually occur with significant traction (e.g., on eye movements), and usually occur during active process of vitreous detachment.

The flashes of light persist if areas of vitreoretinal traction remain.

An arc of light is usually associated with traction at the vitreous base whereas a . flash bulb type of photopsia repeatedly in the same position indicates localised traction.

Although photopsia is a symptom of vitreoretinal traction, patients with photopsia have no higher incidence of retinal tears than those without the symptom.

Very few eyes with asymptomatic PVD have gone on to produce a retinal tear.

Complications of acute PVD

Retinal tear

Various studies have suggested that between 8 – 46% of eyes with PVD develop retinal tears due to traction at sites of strong vitreoretinal adhesions. Although they usually develop at time of PVD but can occur weeks or months later.

Tears are usually, but not always, symptomatic (photopsia and/ **or floaters**)

There are various predisposing factors for PVD producing tears including presence of lattice and snail track degenerations.

Flap or Horseshoe Retinal Tear

Results from vitreous traction which pulls a tear of sensory retina that almost always remains attached at the anterior margin of the break. This gives the flap a horseshoe shape.

Asymptomatic flap tears have been reported to have 25% -90% incidence of progressing to detachment. Therefore, essentially all flap tears are treated

Operculated tear

Here, traction causes the removal of plug of sensory retina (operculum). Commonest cause of operculated tear is PVD. Operculum shrinks with time (x5) Size of plug of retina compared to associated hole will give indication of how recent the lesion occurred.

There is 10 -20% risk of retinal detachment with an operculated tear. Scleral depression enhances the view. Generally, operculated tears are not treated unless there is significant RD.

Sometimes there may be white collar around hole that represents very localized detachment (less that 1 DD from the edge of the break).These tears are most often found between ora serrata and equator where retina in thinner than in posterior region.

Retinal detachment (RD)

Rhegmatogenous detachment (*rhegma* = break)most commonly associated with PVD or trauma.

Tractional retinal detachment is associated with conditions such as proliferative diabetic retinopathy, Eales disease and CRV occlusion.

Exudative retinal detachment is associated with, e.g., choroidal tumours.

RD is separation of sensory retina from RPE (outer segments of the photoreceptors from microvilli of RPE).

Appearance in fresh detachment is of white membrane, with tiny folds and blood vessels, in vitreous cavity that moves (undulates) on eye movements

Because the RD is separated from RPE by fluid reservoir, underlying choroidal detail will be obscured (same as retinoschisis).

RD will be mobile and to differentiate it from retinoschisis, the patient should be asked to move his eye and then re-fixate. The appearance of the folds in an RD will be different on each occasion. In contrast to this, a retinoschisis will appear unchanged.

Final conclusions

If a patient reports flashes of light and or spots/floaters:

1. Record details including duration/association
2. Dilate and view vitreous for “tobacco dust” using slit lamp and retina using indirect method
3. Record your findings
4. If you are unable to dilate or use BIO method then refer the patient to an optometrist or ophthalmologist who can
5. If you diagnose uncomplicated PVD then discuss with patient, inform that patient should see advice if symptoms change or worsen in any way and reschedule for six week review including dilation.
6. Record in the record this advice
7. Retinal tears or detachments should be urgently referred to an ophthalmologist

References

Elliott, (1997) *Clinical Procedures in Primary Eye Care.* p.109, Butterworh Heinemann, Oxford

Jones W.L. (1998) *Atlas of the Peripheral Ocular Fundus,* 2nd Edition, Butterworth Heinemann, Boston