

# Differential diagnosis of PVD and retinal detachment

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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

Mydriasis is obtained by instilling an anti-muscarinic such as 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 the fundus. Enhance dilation is obtained by also instilling a sympathomimetic such as phenylephrine 2.5% (not 10%) which acts on the dilator muscle.

Precautions to be taken prior to dilation include a routine evaluation of the anterior chamber angle. 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.

Sympathomimetics must be used with caution in any patient with a history of cardiovascular disease, with pregnant women, with patients with advanced diabetic disease and with patients taking medication such as MAOI. Ask women of childbearing age if they are pregnant and record their answer. It is advisable to check the systemic blood pressure of any patient over the age of 40 years before instilling phenylephrine.

### Instrumentation

As previously discussed, most tears occur anterior to the equator (Jones, 1998). Direct ophthalmoscopy will only provide a view of the fundus up to the equator **with a dilated pupil** (Elliott, 1997). Thus, most retinal tears will not be visible with a direct ophthalmoscope even if the pupil is dilated.

The optimum method of examining the peripheral retina is with the head set BIO with scleral depressor. Head set BIO takes a lot of practise and according to the College of Optometrists *Clinical Practice Survey* (2001) only 15% of respondent optometrists used the Head set BIO in practice. Scleral depression or indentation further enhances the view of the peripheral retina and aids detection and analysis of retinal breaks. However, this takes further skill and it is likely that even fewer optometrists in the UK use this techniques.

Another useful method of viewing the peripheral retina is with a slit lamp microscope with a mirrored contact lens. Once more this is a technique that is probably currently not used by many optometrists in the UK. Indeed, the College of Optometrists survey did not even enquire after its use.

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**

The **retina** composed of inner neural or sensory layers and outer pigment epithelium.

The normal retina is transparent except for pigment in the blood. The sensory retina thin and weak and is susceptible to full thickness breaks.

The **RPE** is a uni-layer of polygonal cells, uniform in size and pigment except at macular and vitreous base (vitreo-retinal symphysis). The inner surface has microvilli giving loose attachment or bond to sensory retina.

The RPE is attached to the basement membrane which forms the innermost layer of basal lamina (Bruch's membrane). Bruch's membrane bonds to the choroid.

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

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

The vitreous fills 2/3rds of the globe and provides structural and metabolic support for retina. It consists of collagen type II.

There is a depression posterior to lens called the patellar fossa and Cloquet's canal traverses anterior/posterior.

The vitreous base is a 3-4 mm zone straddling the pars plana.

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

## **PVD**

PVD is the condition in which the 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

It is rare in the under 30s and aphakic eyes have a higher incidence as do high myopes. It is reportedly more common in females (postmenopausal changes ?). Trauma to the eye increases incidence e.g., boxers. There have reportedly been 2 cases of PVD following NCT.

### **Clinical appearance**

Ophthalmoscopically it can be seen as very thin and transparent membrane and can sometimes be seen with the direct ophthalmoscope. With biomicroscope it appears as a more dense transparent membrane in central portion of vitreous cavity. The posterior face of the vitreous cortex contains particulates which scatter light and these move on eye movements.

The presence of an avulsed pre-papillary gliotic ring (Weiss) is pathognomnic of PVD. It may appear as a 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 the patient is asked to move his eye around then the glial tissue will move especially in the presence of saccades. This is sometimes known as the ascension-descension phenomenon.

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

## **Pathophysiology**

PVD usually commences over posterior pole. Synchysis (liquefaction) leads to formation of pockets of fluid called lacunae. Syneresis (physical contraction or shrinkage) occurs and the 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 the 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 a frequent symptom of PVD and occur through mechanical stimulation of the retina by the 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 indicated 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 published papers 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 the presence of lattice and snail track degenerations.

### ***Flap or Horseshoe Retinal Tear***

A flap 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 or horseshoe shape.

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

### ***Operculated tear***

Here, traction causes the removal of plug of sensory retina (operculum). The commonest cause of an operculated tear is PVD. The operculum shrinks with time (x5) and the size of this plug of retina compared to its associated hole will give an indication of how recent the lesion occurred.

There is a 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 the hole that represents a very localized detachment (less than 1 DD from the edge of the break).

These tears are most often found between ora serrata and equator where the retina is thinner than in posterior region.

## **Retinal detachment (RD)**

**Rhegmatogenous** detachment, derived from *rhegma* meaning break is 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, for example, choroidal tumours.

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

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

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

The detached retina 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.

## References

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

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