Posterior vitreous detachment

Dr Katie Williams reviews the assessment of posterior vitreous detachment and the best management options. **Module C14674, one general CET point for optometrists and DOs**

Posterior vitreous detachments account for a significant number of patient consultations for both optometrists and ophthalmologists. The condition is a normal physiological occurrence that for most patients occurs without any ocular complications. However, for some there can be significant retinal sequelae. This article aims to revise what a posterior vitreous detachment is, why it is relevant, recognise risk factors for further complications in the light of a recent clinical study, and provide advice regarding current recommended clinical practice.

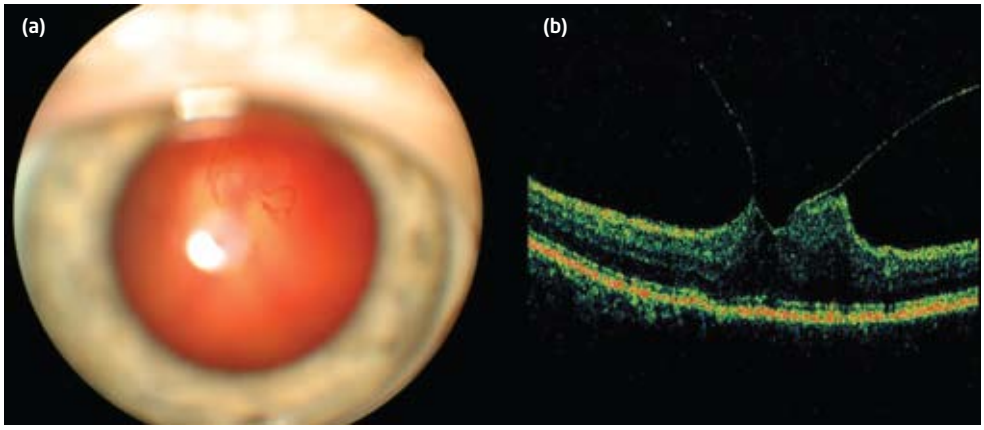

Figure 1 Posterior vitreous detachment (a) Weiss' ring (b) OCT showing posterior vitreal attachment distending the retina

What is a posterior vitreous detachment?

A posterior vitreous detachment (PVD) can be defined anatomically as the separation of cortical vitreous from the internal limiting membrane of the retina. This is the final step in progressive liquefaction and shrinkage of the vitreous gel, followed by penetration of vitreal fluid behind the posterior hyaloid face (Figure 1).

The condition is termed symptomatic if it occurs in the presence of photopsia (flashing lights), floaters or reduced vision. It is, however, important to note that there are many other causes of floaters and/or flashing lights, including both ocular causes, such as posterior uveitis, and non-ocular causes, such as migraines. Classically, patients with a PVD describe brief, monocular flashes of light in their peripheral vision that are often more noticeable at night.

Detachment of the posterior vitreous is a normal physiological change in the maturing eye, but is also known to have a higher frequency in myopes, pseudophakes, and females following the menopause.[1] The reported prevalence ranges from 24 per cent in adults, to 87 per cent in those aged 80 to 89.[2]

What is the relevance of a posterior vitreous detachment?

Although a PVD generally occurs without ocular complications, it is recognised to be the leading cause of a retinal break (ie a tear or hole in the retina). Strong adhesional forces exist between the vitreous and retina, especially at the vitreous base which occurs approximately 4mm posterior to the ora serrata. As the cortical vitreous separates from the retina these tractional forces may be sufficient to cause a tear or hole in the retina (Figure 2).

Retinal breaks can be assessed in terms of their shape, size, site and adhesions.

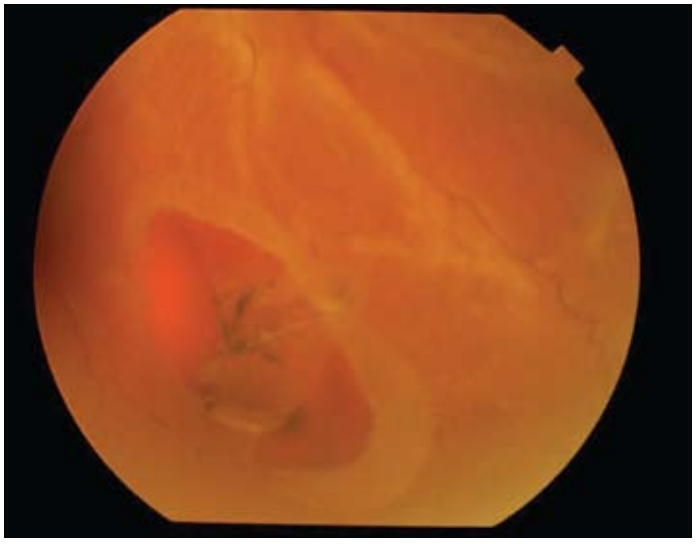

Figure 2 An inferior retinal tear with localised retinal detachment (Courtesy of Tom Williamson)

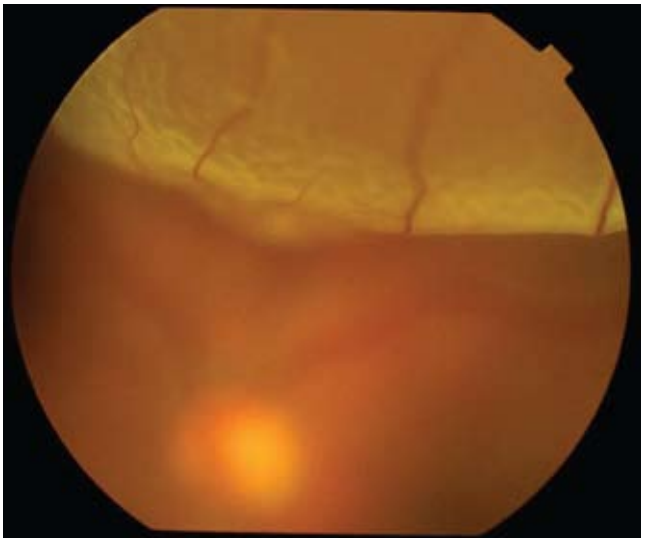

Figure 3 A superior retinal detachment (Courtesy of Tom Williamson)

This assessment can in turn be used to grade the retinal break as high risk or low risk. For example, a high risk tear could be defined as a u-shaped tear, large in size, located in the superior retina, with strong adherence to the adjacent cortical vitreous. In contrast a low risk tear could be defined as a round hole, small in size, located in the inferior retina with weak adhesions to the adjacent vitreous.

It is known that 30-50 per cent of the high risk retinal breaks associated with a PVD, in the presence of persistent vitreoretinal traction, may lead to a rhegmatogenous retinal detachment.[3] In this situation vitreal fluid penetrates to the sub-retinal space via the retinal break, causing separation of the neurosensory retina from the retinal pigment epithelium, ie a retinal detachment (Figure 3). Left untreated this will result in irreversible retinal damage and loss of vision. There is a significant disparity in both the visual outcome and financial implications in the management of a retinal detachment compared to a retinal break. Therefore the clinical ideal would be that all retinal breaks secondary to PVD are promptly diagnosed and treated with argon laser retinopexy.

What is the risk of a retinal break in a symptomatic PVD at initial assessment?

It is known that approximately 10-15 per cent of patients with a symptomatic PVD will have a retinal break at their primary assessment (Coffee *et al* 2007 case series 8.2 per cent, Coffee *et al* meta-analysis 21.7 per cent).[4] Major risk factors for retinal breaks at the initial assessment include the presence of pigmented vitreous granules (ie retinal pigment epithelial cells, commonly known as 'tobacco dust') and/or vitreous haemorrhage. Published research suggests that a retinal break will be present in 100 per cent of patients with pigmented vitreous granules and 45 per cent of patients with vitreous haemorrhage.[5] In other research the presence of pigmented vitreous cells, vitreous haemorrhage or retinal haemorrhage has been found to give a retinal break odds ratio (OR) of 9.3 (confidence interval (CI) 5.4- 497). This statistic can be interpreted as meaning that the presence of one or more of these features makes it nine times more likely that a retinal break will be present. There are a number of other known risk factors including myopia, underlying retinal pathology, trauma, pseudophakia and male gender.[6,7,8]

What happens following the posterior vitreous detachment?

It is known that a retinal break may develop in the period following the initial assessment of an acute PVD; this is defined as a delayed retinal break. This tends to occur within six weeks of symptom onset and only rarely occurs subsequently. Debate arises around the true incidence of delayed retinal breaks due to the fact that retinal breaks found subsequent to initial assessment could simply be a retinal break that was initially missed. However, published research indicates the incidence of retinal breaks to range from approximately 1-3 per cent (Coffee *et al* case series 1.5 per cent, Richardson case series 2.1 per cent, Coffee *et al* meta-analysis 1.8 per cent, Hollands *et al* meta-analysis 3.4 per cent).[4,8,9] A subsequent PVD in the other eye is highly likely to occur following the PVD in the first eye, generally within 6-24 months.[10] The natural history of a PVD is illustrated in the Figure 4 flow chart.

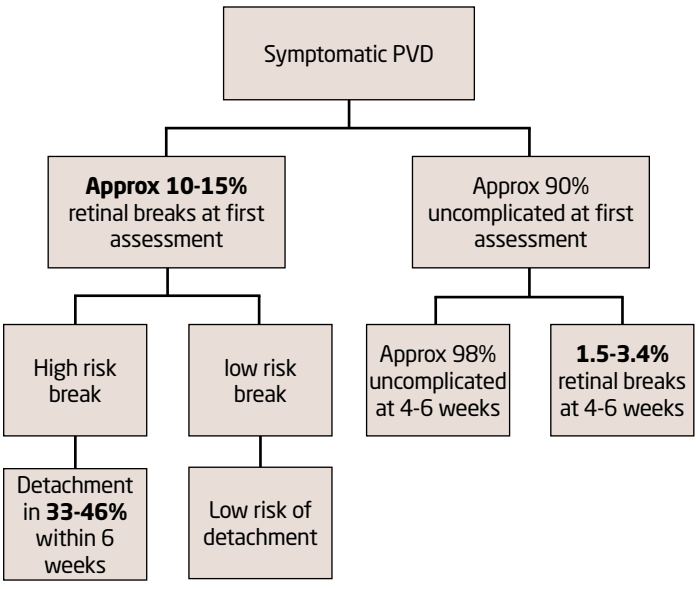

Figure 4 Flow chart illustrating the natural history of an acute PVD

Prospective study of delayed retinal breaks

At the author's practising centre it is policy to review all patients with an acute symptomatic PVD of less than four weeks' duration. This is not policy at all eye departments. In order to clarify the local incidence of delayed retinal breaks and additionally identify potential risk factors for rationalisation of follow-up the authors performed a seven-month prospective study. In the study period, 354 patients presented with an acute symptomatic PVD of less than four weeks' duration. All patients were invited for a follow-up at four to six weeks, or earlier if vitreous haemorrhage was present. Approximately 9 per cent of patients did not attend follow-up and seven were excluded on the basis that the PVD was not felt to be the cause of their symptoms.

At the study end-point (six weeks following initial assessment) 14 retinal breaks were identified, giving a delayed retinal break incidence of 3.95 per cent. The retinal breaks included two retinal detachments (macula on). Thirteen of the retinal breaks occurred in the presence of one or more of these features; vitreous haemorrhage, or retinal haemorrhage, or pseudophakia, or myopia, or symptom duration less than 14 days, but this was not statistically significant (OR 1.05; 95 per cent CI 0.13 to 8.35). The individual risk factors for delayed retinal breaks were considered separately; only vitreous haemorrhage was found to be a statistically significant risk factor (OR 11.89; 95 per cent CI 3.76 to 37.58) (Table 1).

The authors identified an incidence of delayed retinal breaks comparable to published clinical standards.[4,8,9] This confirmed the opinion of the authors that the incidence of delayed retinal breaks provides sufficient evidence for reviewing patients with an acute PVD within six weeks. The study additionally identified that only vitreous haemorrhage is a statistically

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significant risk factor for a subsequent delayed retinal break and that the study findings did not support targeted clinical follow-up.

What is the current advised clinical practice?

All patients presenting with an acute symptomatic PVD must receive a thorough examination of their fundus including the peripheral retina. History and specifically length of symptoms should be assessed. The likelihood of a retinal tear in patients with flashes and floaters, floaters alone or flashes alone appears fairly equal in published studies and is therefore not clinically useful in determining risk of retinal break.[8] Visual acuity and defects in the visual field should be recorded. A reduction in visual acuity has been found to be associated with an increased likelihood of a retinal break (likelihood ratio (LR) 5.0, 95 per cent CI 3.1-8.1).[8] Defects in the visual field are highly suggestive of a retinal detachment and can aid the examiner in locating the site of the detachment.

The IOP should be measured in both eyes; although not helpful in determining the presence of a retinal break, a low IOP may be indicative of a retinal detachment. Direct and consensual pupillary responses should be noted before then installing dilating drops into both eyes. Slit examination of the anterior vitreous should be performed to look for pigmented vitreous granules and vitreous haemorrhage; as mentioned earlier both are highly predictive of a retinal break.[5] This is best performed by asking the patient to look up, then immediately down and then immediately straight ahead with the slit lamp beam focused on the anterior vitreous. The gold standard technique for assessment of the fundus entails indirect ophthalmoscopy with indentation; this allows dynamic examination of the retina right up to the ora serrata. In practice this examination may be performed using a three-mirror or super-quad contact lens. Generally this part of the examination is preceded by assessment of the posterior pole and mid-periphery using a Volk lens. If a retinal break or retinal detachment is identified this should be promptly managed.

Referral to ophthalmology can be prioritised based on the above assessment. All patients presenting with an acute PVD require a thorough assessment of their peripheral retina. Volk lens examination alone is inadequate as this method does not allow visualisation of the peripheral retina and will therefore mean peripheral breaks are missed. The urgency with which referral to ophthalmology should be made can be guided by certain features. For example a reduction in vision, a visual field defect, vitreous haemorrhage or pigmented vitreous granules signify a high probability of retinal complications and therefore require urgent referral.

The next step in clinical management, as described earlier, will vary between eye departments. Some ophthalmic units will review patients with an uncomplicated PVD; others will discharge the patient but with clear instructions to seek further assessment if they notice an increase in floaters and/or flashing lights, a reduction in their vision or a visual field defect. It should be noted that floaters may persist for months or even years after a PVD; therefore patients should be instructed to be concerned only if their floaters increase in frequency. As described earlier, vitreous haemorrhage at the primary assessment is a statistically significant risk factor for a subsequent retinal break. Symptomatically meta-analysis findings suggest a sudden increase in the number of floaters from <10 to ≥10 and subjective visual reduction are predictive of a new retinal break (LR 8.1-36 and 2.3-17 respectively).[8]

TABLE 1

Risk factors for delayed retinal break secondary to acute, symptomatic posterior vitreous detachment

	Patients reviewed with symptomatic PVD (n)	Delayed retinal break (n)	Odds ratio (95 per cent CI)
Myopia	171	8	1.45 (0.47 to 4.54)
Pseudophakia	29	2	1.93 (0.41 to 9.08)
Retinal haemorrhages	47	3	1.82 (0.49 to 6.77)
Vitreous haemorrhages	26	6	11.89 (3.76 to 37.58)
Increase in symptoms	31	0	0.32 (0.05 to 2.12)
Symptoms <14 days	296	13	2.48 (0.32 to 19.36)

References

1 Chuo *et al.* Risk factors for PVD: A case-control study. *Am J Ophthalmol*, 2006;142:931-937.

2 Hikichi T *et al.* Comparison of the prevalence of pvd in whites and Japenese. *Ophthalmic Surg*, 1995; 26(1): 39-43.

KEY POINTS

- A PVD is the separation of cortical vitreous from the retina
- A PVD is commoner in older age, myopes, pseudophakes, females and following the menopause
- At initial assessment of a PVD 10-15 per cent of patients have an associated retinal break. The risk of a retinal break is higher in the presence of vitreous haemorrhage, pigment vitreous granules, myopia, underlying retinal pathology, trauma, pseudophakia and the male gender
- Approximately 30-50 per cent of high risk retinal breaks may lead to a retinal detachment
- In the period following an acute PVD 1-3 per cent of patients may develop a delayed retinal break, generally in the initial six weeks. The risk of a delayed retinal break is statistically significantly higher in the presence of vitreous haemorrhage
- Initial assessment of a symptomatic PVD should include:
  - Visual acuities
  - Visual fields
  - IOPs
  - RAPD
  - Slit-lamp examination of anterior vitreous
  - Peripheral fundus examination using appropriate technique
  - Consideration of urgency of referral
- Features predictive of a delayed retinal break include vitreous haemorrhage at initial assessment, a significant increase in the number of floaters and a reduction in visual acuity

3 Davis. Natural history of retinal breaks without detachment. *Arch Ophthalmol*, 1974;92:183-194.
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9 Richardson *et al*. The PVD clinic: do new retinal breaks develop in the six weeks following an isolated symptomatic PVD. *Eye*, 1999;13:237-240.
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MULTIPLE-CHOICE QUESTIONS – take part at opticianonline.net

1 Which of the following is unlikely in symptomatic PVD?
A Photopsia
B Photophobia
C Vision loss
D Floaters

2 Which of the following is not a known risk factor for PVD?
A Female gender after menopause
B Age
C Pseudophakia
D Hyperopia

3 What is the prevalence of PVD in people in their 80s?
A 24 per cent
B 42 per cent
C 72 per cent
D 87 per cent

4 What percentage of PVDs have an associated retinal break?
A 1 to 3 per cent
B 10 to 15 per cent
C 30 to 50 per cent
D 90 per cent

5 What percentage of patients presenting with pigmented vitreous cells retrolentally will have a retinal break?
A 100 per cent
B 75 per cent
C 45 per cent
D 1 to 3 per cent

6 What percentage of high risk breaks will go on to a retinal detachment?
A 100 per cent
B 70 to 80 per cent
C 30 to 50 per cent
D 1 to 3 per cent

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