Posterior Vitreous Detachment

4

Contents

4.1 Introduction

Posterior vitreous detachment is the commonest and most important event that occurs in the vitreous. As the vitreous ages, the normal architectural features apparent in childhood gradually disappear as degeneration causes syneresis, lacuna (cavity) formation and collapse of the vitreous gel. The collagen fibrils disintegrate and aggregate giving rise to symptomatic floaters (Bishop et al. 2004; Akiba et al. 1993). There is loss of sodium hyaluronate (Larsson and Osterlin 1985) and an increase in vitreous mobility with age (Walton et al. 2002). Most individuals will develop posterior vitreous detachment (separation of the posterior hyaloid membrane from the internal limiting membrane), without symptoms or pathological sequelae, usually between the ages of 40–80 years. 27 % of patients in their seventh decade and 63 % in their eighth decade have PVD (Foos and Wheeler 1982). This may occur at a younger age (less than 40 years old) in myopia, diabetes, retinal vascular disorders, trauma and retinitis pigmentosa (Hikichi et al. 1995b, c; Sebag 1993; Morita et al. 1995; Yonemoto et al. 1994; Akiba 1993). Presentation with symptomatic PVD (flashes and floaters) may be more common in females than males and in myopia (Chuo et al. 2006). The detached posterior hyaloid membrane becomes wrinkled and usually separates completely from the retina up to the posterior border of the vitreous base (or up to the posterior aspect of any other vitreoretinal adhesions, which may be present). Acute ischaemic events such as retinal vein occlusion may induce PVD with an increased prevalence of PVD 1 year after onset of the vein occlusion (Kado et al. 1990). No racial differences in the rates of PVD have been found between whites and oriental peoples with disagreement whether black races have less PVD (Hikichi et al. 1995a; Weiss and Tasman 1978; Foos et al. 1983). The fellow eye has evidence of PVD in 90 % in 3 years (Hikichi and Yoshida 2004) with 11 % developing symptomatic PVD in the other eye in 2 years often demonstrating similar problems to the first eye such as tears or vitreous haemorrhage (Novak and Welch 1984).

PVD is the primary process in the development of most rhegmatogenous retinal detachments because of its role in retinal tear formation. It is also implicated in common macular disorders, such as macular pucker and macular hole formation. Separation of the gel may also tear blood vessels in the retina or in neovascular complexes causing haemorrhaging into the vitreous cavity. The importance of PVD has led many investigators to try methods for the artificial inducement

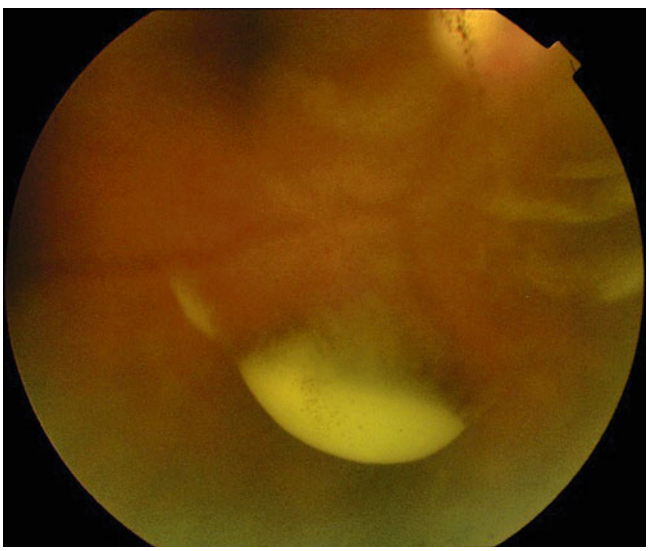

Fig. 4.1 Syneresis of the vitreous causes lacuna formation; in this figure, the presence of a lacuna is demonstrated by the fluid level of old altered blood which is trapped inside a lacuna cavity

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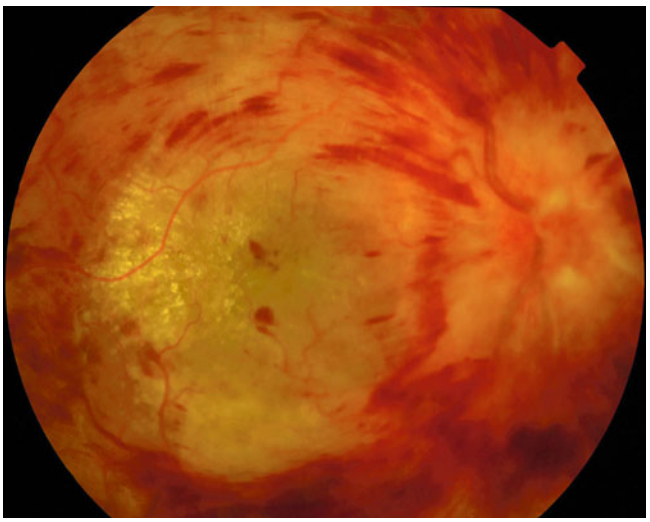

Fig. 4.2 A 13-year-old girl with CRVO in whom the onset was associated with a bout of diarrhoea on ski trip and a short-haul plane journey. She had a familial cholesterolaemia

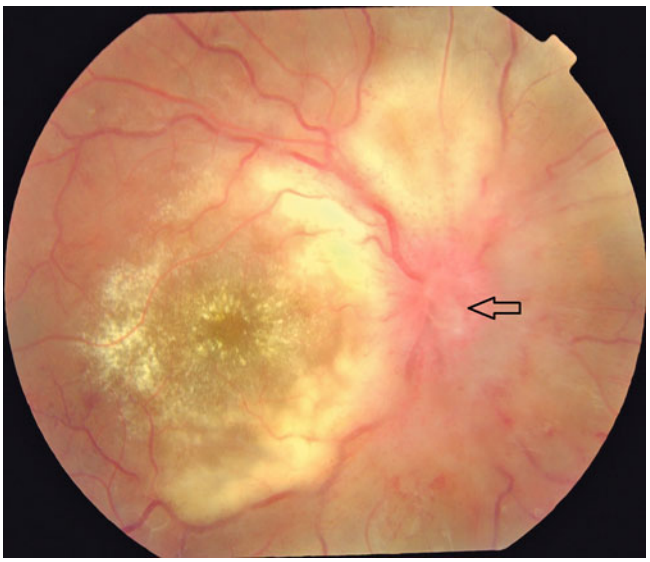

Fig. 4.3 At 2.5 months, there is a Weiss ring (*arrow*) visible in front of the disc demonstrating induction of a PVD by the CRVO

Table 4.1 Vitreoretinal conditions and the vitreous

Vitreoretinal conditions and the vitreous	
Caused by age-related posterior vitreous detachment	Retinal breaks
	Rhegmatogenous retinal detachment
	Macular epiretinal membrane and vitreomacular traction syndrome
	Macular hole
	Vitreous haemorrhage
Associated with pathological vitreous separation	Diabetic tractional retinal detachment
	Complications of posterior uveitis
	Trauma

of PVD (Unal and Peyman 2000; Hesse et al. 2000; Kakehashi et al. 1994; Hikichi et al. 1999; Verstraeten et al. 1993; Harooni et al. 1998; Tezel et al. 1998; Kang et al. 1995) such as plasmin injection as a proteolytic acting on the vitreoretinal interface.

1. The posterior hyaloid membrane consists of type IV collagen (Snead et al. 2002).
2. The vitreoretinal adhesions at the vitreous base move gradually posteriorly as the eye ages (Wang et al. 2003).
3. Epipapillary glial tissue can become avulsed from the disc margin during vitreous detachment and may be seen ophthalmoscopy as a small ring of tissue in front of the optic disc (Weiss ring). The ring is often incomplete and is absent in 13 % of PVDs (Akiba et al. 2001; Kakehashi et al. 1998).

Patients often notice a floater in their vision describing it as a ‘cobweb’ or ‘spider’ or ‘fly’ which moves with eye movements. OCT has revealed that many adults have an incomplete PVD not visible on biomicroscopy but with separation of the posterior hyaloid membrane from the retina with residual attachments at the optic disc or the fovea (Uchino et al. 2001).

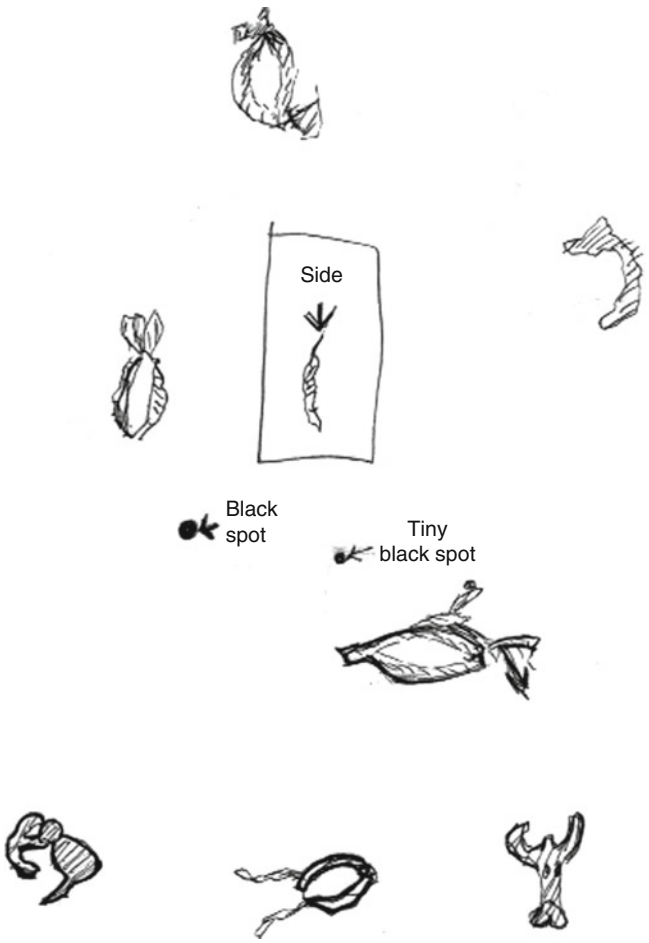

Fig. 4.4 One patient’s drawing of his floaters from posterior vitreous detachment showing the Weiss ring moving around in the eye

4.1.1 Symptoms

4.1.1.1 Floaters

Floaters must be discriminated from paracentral scotomata. Ask the patient to describe the floater which should have momentum as the eye moves, that is, the floater will move with the eye but will continue to move when the eye stops before finally returning to its original position and resting there. In contrast, a scotoma remains in the same position (relative to fixation) in all positions of gaze. The patient may also describe the floater as something in front of the vision or 'in the way' of the vision. Floaters can be characterised by cobweb, veils, a ring, spot or multiple spots. These come from thickened posterior hyaloid membrane, Weiss ring, or cells that have been dispersed into the vitreous. Floaters that occur before the age of 40 years and are chronic in presentation are most often due to vitreous degeneration without posterior vitreous detachment. However, it may only take a single floater of recent onset to indicate the development of a posterior vitreous detachment.

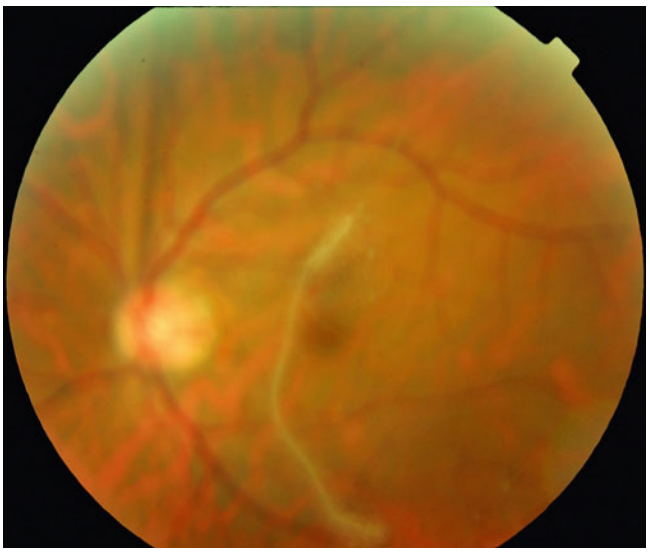

Fig. 4.5 Large 'pipe-cleaner'-shaped vitreous opacities can be seen in moderate or highly myopic eyes without the presence of PVD. If you operate to remove these for symptomatic reduction of floaters, be aware that a vitreoschisis must be looked for and removed; otherwise, the posterior hyaloid may separate later on and cause RRD (see Chap. 7)

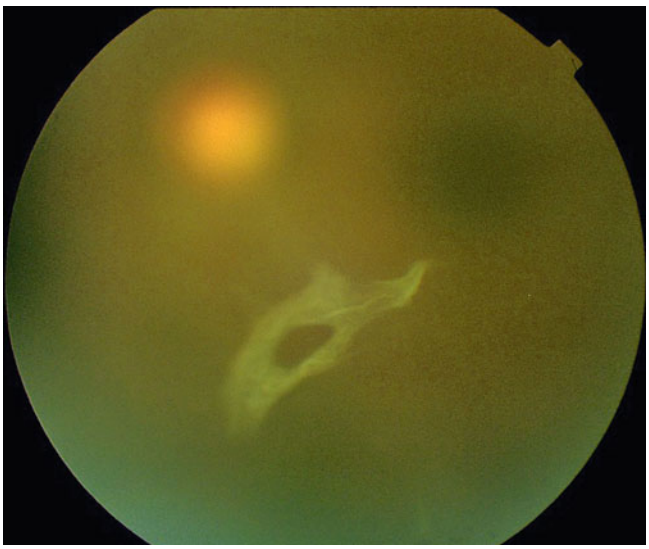

Fig. 4.7 A Weiss ring is apparent in this picture. A Weiss ring may take various forms in different eyes, and as the Weiss ring moves more anteriorly in the eye, it becomes less in focus and less noticeable by the patient

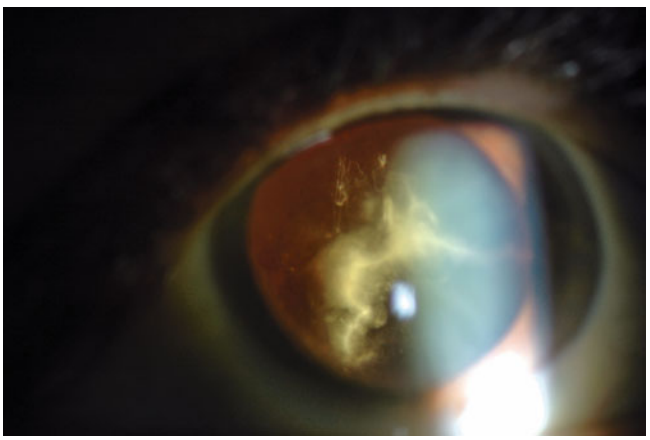

Fig. 4.6 If vitreous opacities are very symptomatic, these can be removed by vitrectomy. Beware of the high myope with apparent PVD who in fact has a vitreoschisis (see Chap. 6)

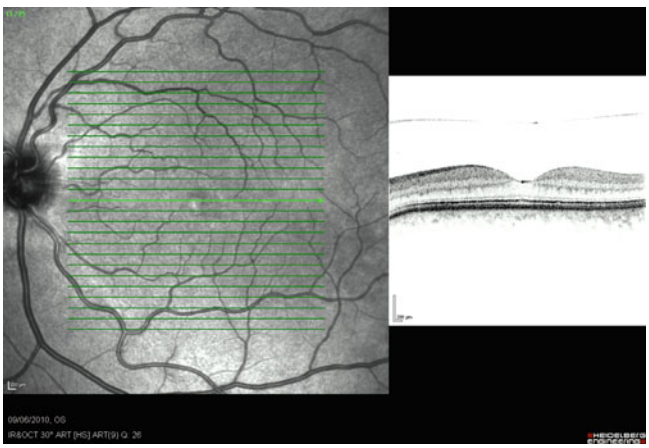

Fig. 4.8 An occult separation of the posterior hyaloid membrane from the macula, only visible on OCT

4.1.1.2 Flashes

Introduction

Photopsia is the experience of light from non-photic stimulation; the first description in the modern era was from Purkinje in 1819 (Purkinje 1819), who attributed them to traction. In 1935 and 1940, Moore described 'lightning streaks' (Moore 1935) with a 'flash-like appearance of the lights; their position, sometimes slanting but usually vertical, and almost always to the outer side of the eyes, persisting for periods of up to 3 months; and their association with the sudden development of muscae volitantes, or the presence of visible vitreous opacities'. In 1940, he added more cases using the term lightning flashes (Moore 1940) and developed streaks in his own eye in 1947 (Moore 1947). He initially thought the phenomenon to be wholly benign, although he commented that 'I used systematically to dilate the pupils and to take the visual fields in fear lest they might indicate some early organic retinal lesion, such as a commencing detachment, vascular disease, or perhaps an early neoplasm' (Moore 1935). The flashes were attributed to posterior vitreous detachment (PVD) by Verhoeff (1941) in 1941 and the risk of retinal detachment associated with lightning flashes noted by Berens et al. (1954) in 1954. In 2008, 'black flashes' or negative flashes were described at the commencement of the PVD (Williamson et al. 2008) which have been attributed to traction of the vitreous on the optic nerve head, see below.

Clinical Characteristics

Patients experience 'lightning flashes' in the temporal periphery of their visual field that typically last a second at a time. Their exact pathogenesis is obscure but may be due to depolarisation of the receptors from tugging of the vitreous base on the retina. They may occur on eye movements. If the patient produces repeated eye movements over a short time, the flashes gradually reduce in severity. This may be because the retina loses the ability to respond due to repeated depolarisation of the receptors. Sometimes they are better seen at night. In some patients, black temporal flashes are seen for a few hours before the lightning flashes and floaters occur (Williamson et al. 2008). These are thought to be produced by the Weiss ring pulling on the optic nerve head before it separates. The forces applied in this way to the surface of the nerve head block axoplasmic flow in the superficial nerve fibres, thereby inducing a negative peripheral visual phenomenon. As soon as the Weiss

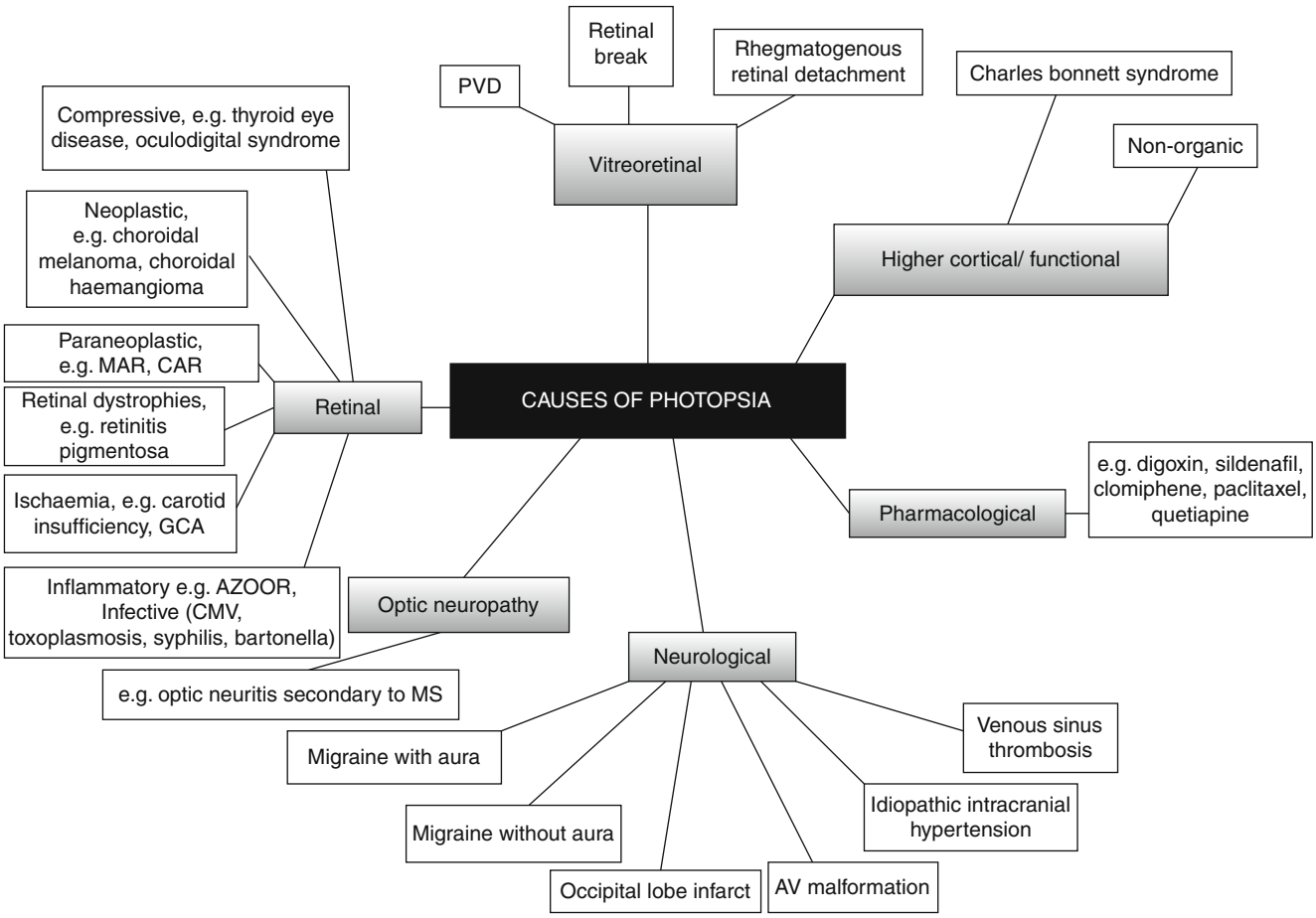

Fig. 4.9 There are multiple causes of photopsia

Table 4.2 Different presentations of flashes

Flashes							
Diagnosis	Duration	Colour	Location	Shape	Stimulus	Other symptoms	Flickering
PVD	Seconds or less	White	Temporal periphery	Crescentic vertical	Eye movements	Floaters	No
Migraine	20–30 min	Not typically	Paracentral	Arcuate Zigzag	Stress, food	Scotoma headache nausea	Yes
Occipital ischemia	Minutes	Nil	Central	Petaloid	Neck movements exertion		Yes
Cystoid macular oedema	Constant	Variable	Central	Pinpricks	Nil	Poor vision	Yes
Outer retinal or RPE abnormality	Minutes	Blue Purple	Paracentral	Blobs Spirals	Nil	Scotoma	No
Retinal detachment	Seconds	Golden	Central/paracentral	Comet oblique horizontal	Eye movements	Visual field loss	No

ring separates, the symptoms change to floaters and lightning flashes. Typically, the lightning flashes of PVD are vertical, temporally placed and instantaneous flashes. If the flashes in a patient with PVD are oblique or horizontally orientated, not in the temporal visual field or not typical instantaneous flashes, the patient is more likely to have a PVD with a retinal tear or rhegmatogenous retinal detachment (Goodfellow et al. 2009).

These visual phenomena should be discriminated from the flashes that occur with other disorders, such as the zigzag lights of migraine, flickering stars associated with occipital ischaemia and the rare coloured lights of the acute zonal outer occult retinopathy (AZOOR) syndromes. Mostly, this photopsia is centrally placed in the visual field and therefore can be easily discriminated. Slower more mid-peripheral flashes are produced by the leading edge of some retinal detachments often shaped like a comet's tail.

Patients who experience symptoms during posterior vitreous separation have a 10 % risk of developing a retinal tear (van Overdam et al. 2001; Sharma et al. 1999; Hikichi and Trempe 1994). Flashes from PVD usually subside in a few months, whilst floaters get less but may not disappear entirely (Serpetopoulos 1997). The floaters lessen as the opacities on the posterior surface of the vitreous sink lower in the eye but also because they move anteriorly and are less in focus than when they were nearer the inner surface of the retina. Severe floaters can be bothersome, and in a few patients, PPV is required to clear the vision. Rarely flashes will persist for years more often associated with vitreous attached young myope RRD (i.e. flashes associated with RRD rather than PVD) and very rarely after PVD. Occasionally, patients will have flashes after PPV.

4.1.2 Signs

4.1.2.1 Detection of PVD

A posterior vitreous detachment can be diagnosed by examining the eye with a 90-dioptre lens. Definitively, if a Weiss ring is present, then a PVD has occurred. Sometimes this is not obvious, but it is possible to see the posterior hyaloid membrane. This is more subjective, but it can be reassuring to observe the space behind the membrane; if this is optically clear, it suggests that there is no vitreous gel at this location. A partial posterior vitreous detachment is a diagnosis that should be made only with care. It can be extremely difficult to determine whether there are remaining vitreous attachments. Likely residual attachments are at the optic disc, chorioretinal scars and epiretinal membranes at the macula and neovascular tissue. Usually, a posterior vitreous detachment occurs completely, soon after the onset of symptoms probably in a few hours. There remain a few patients in whom the posterior vitreous detachment progresses over a few weeks as evidenced by the formation of new retinal breaks in the first 6 weeks after onset of symptoms in 1.8–3.4 % (Hollands et al. 2009).

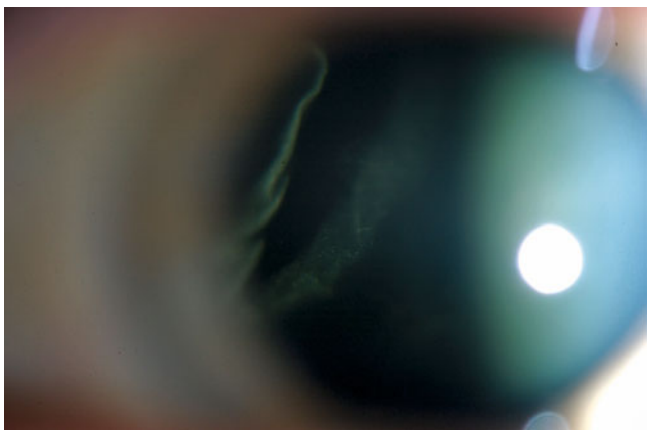

Fig. 4.10 In an eye with forward displacement of a PVD, the posterior hyaloid membrane can be seen in the anterior vitreous cavity

4.1.2.2 Shafer's Sign

In most patients with tears, retinal pigment epithelial cells released through the tear will be visible in the anterior vitreous (Shafer's sign). This is highly predictive of a retinal tear

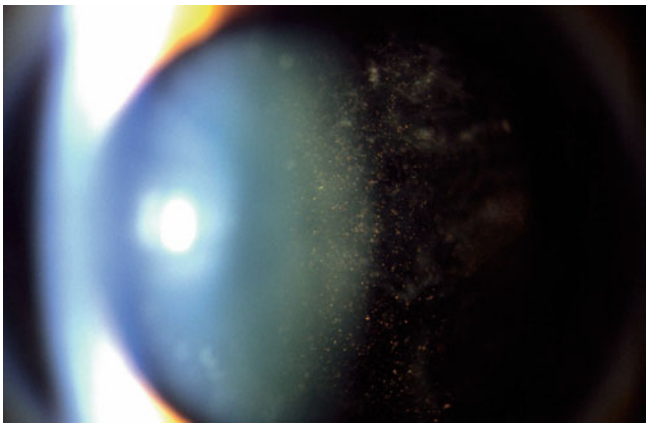

Fig. 4.11 Pigment granules in the vitreous are good indicators of the presence of a retinal tear from PVD

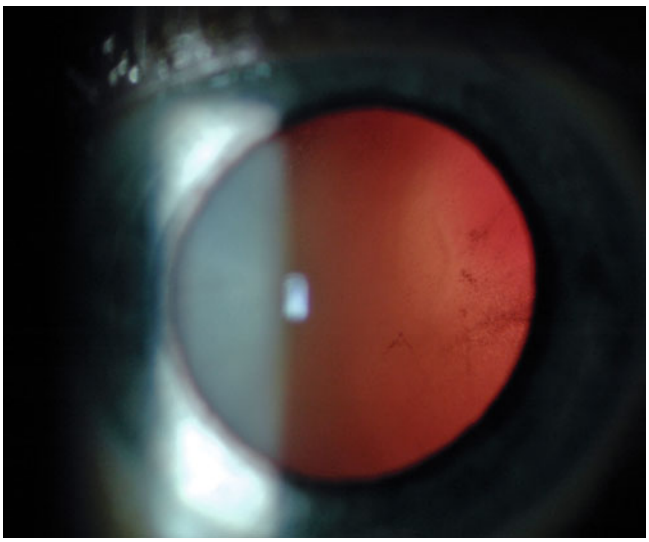

Fig. 4.12 Erythrocytes in the vitreous indicate a 50 % chance of finding and retinal break in an acute PVD

(approximately 90 %) (Tanner et al. 2000; Dayan et al. 1996; Byer 1994; Brod et al. 1991; Sharma et al. 1999). In symptomatic PVD, 10 % of patients will develop retinal tears. Most tears are present when the patient presents; however, 10 % of tears are be detected at 6 weeks from onset of symptoms (constituting theoretically 1 % of all cases with symptomatic PVD) (Richardson et al. 1999; Dayan et al. 1996). Breaks found in asymptomatic eyes are less likely to lead to retinal detachment (Byer 1998).

The pigment granules in Shafer's sign are relatively large (diameter of 30–50 μm), pigmented and are seen in the anterior vitreous especially often inferiorly. The patient, therefore, should be examined during eye movements, allowing inferior vitreous to present itself for examination in the pupil. Only one granule is required to make the diagnosis of Shafer's sign and indicate the risk of a retinal tear.

4.1.2.3 Vitreous Haemorrhage

Red blood cells (RBCs) which are smaller, 6–8 μm, may also be seen and should also raise suspicion of pathology although this is less indicative than pigment granules with 50 % of patients with RBCs in vitreous having retinal tears. Sometimes the haemorrhage is severe, preventing visualisation of all or part of the retina. This should be investigated by ultrasound, and a PPV is performed urgently to allow detection of breaks. In some patients, a superior break might be seen, but the inferior retina might be obscured by inferior vitreous haemorrhage. The surgeon may be tempted to laser the superior break and observe; however, because of the chance of multiple breaks (approximately 50–60 %) and the suspected increased risk of PVR in the presence of haemorrhage, it is safer to proceed to PPV allowing examination of the inferior retina (also see Chap. 5).

4.1.2.4 Ophthalmoscopy

Note: The patient requires 360° examination of the retina with indirect ophthalmoscopy and indentation of the far periphery.

This aids identification of breaks both by introducing peripheral retina into the view but also allowing a dynamic observation of a break. The break can be opened up and more clearly seen by movement of the retina. Moving the choroid under the break changes the colour of the choroid seen through the orifice of the break discriminating a break from retinal haemorrhage or pigmentation which remains the same colour despite indentation. If a patient presents early with PVD, it is worth re-examining the retina at 6 weeks after symptoms occur because 1.8–3.4 % will have tears seen at the second examination that were not seen at the first (Coffee et al. 2007). If patients have vitreous haemorrhage, retinal haemorrhage or develop new symptoms, they may be more likely to have breaks seen at the second examination.

Note: If a patient presents early with PVD symptoms of a few days, another examination at 1–2 weeks is useful.

	Odds ratio for detection of a retinal break (Hollands et al. 2009)	95 % confidence interval
Subjective vision reduction	5.0	3.1–8.1
Vitreous haemorrhage	10	5.1–20
Absence of vitreous pigmentation	0.23	0.12–0.43

Other Signs

PVD may induce optic disc haemorrhages (causing subtle visual field loss) (Katz and Hoyt 1995; Roberts and

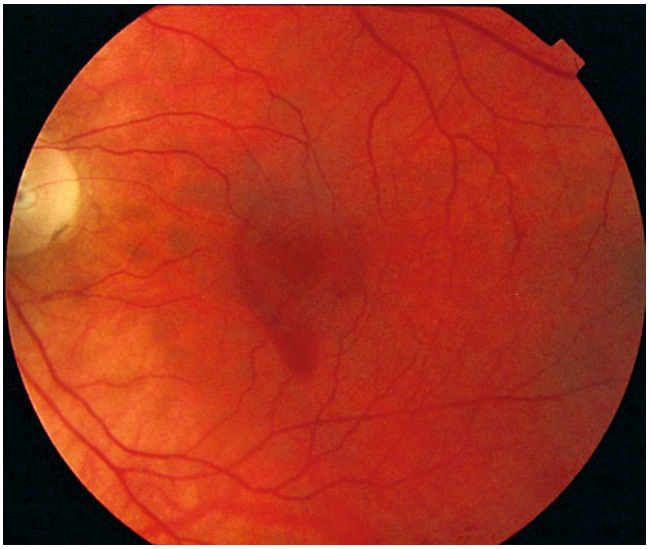

Fig. 4.13 Posterior vitreous detachment can cause a macular haemorrhage from traction on the retina as it detaches, rupturing small blood vessels on the surface

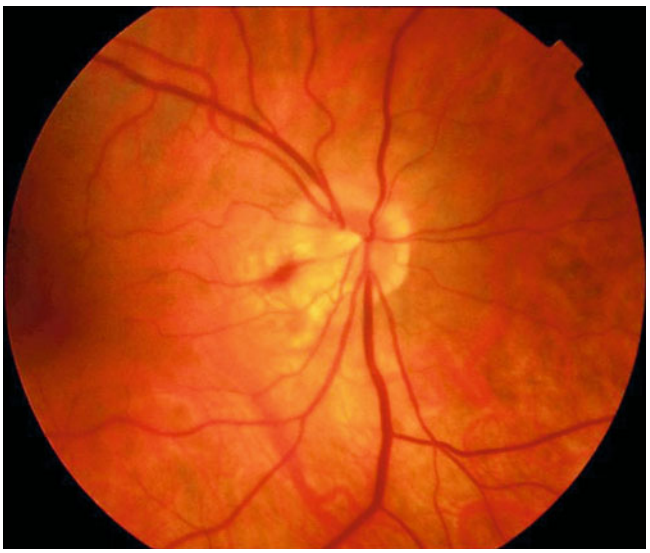

Fig. 4.14 A haemorrhage has occurred at the optic disc in a patient with posterior vitreous detachment

Gregory-Roberts 1991) or retinal haemorrhages in the periphery or in the macula (Cibis et al. 1975; Schachat and Sommer 1986).

4.1.3 Retinal Tears

4.1.3.1 U Tears

All U tears (or tears caused by PVD) require treatment by retinopexy, either by laser or cryotherapy. These present with the base of their flaps anteriorly in the direction of the traction of the vitreous. Any retinopexy must surround the

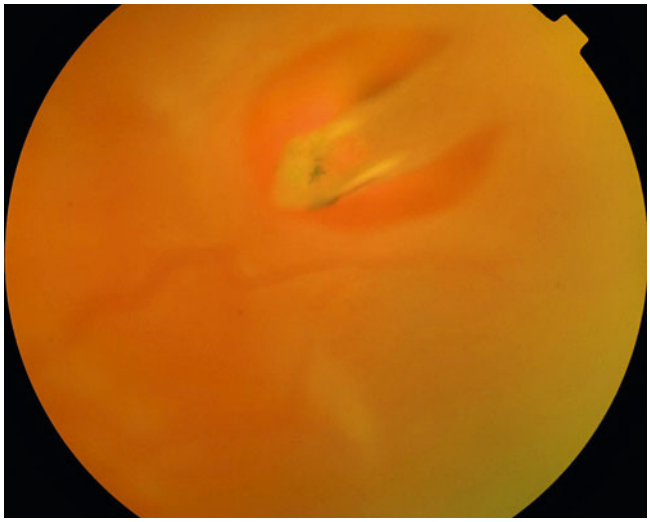

Fig. 4.15 A U tear with a prominent flap

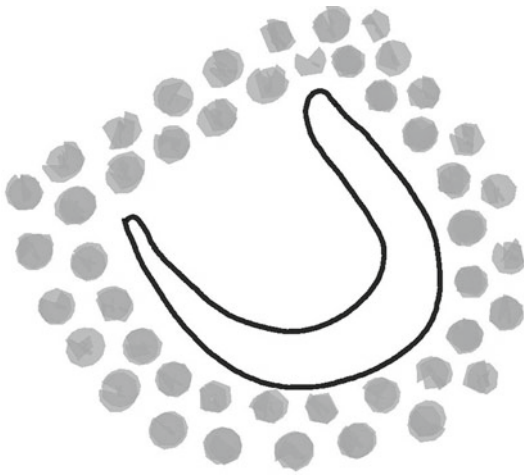

Fig. 4.16 Laser should be placed in two rows around a retinal tear as shown; laser the flat retina close to the edge of the tear

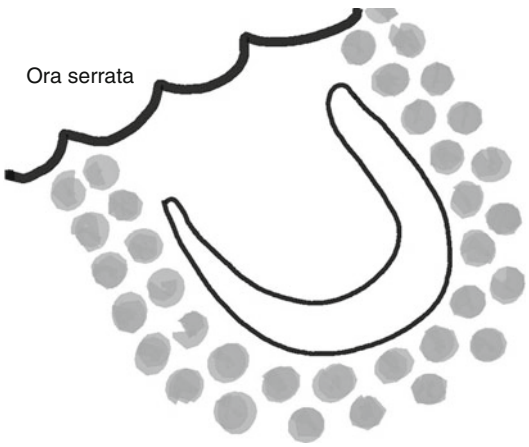

Fig. 4.17 If the retinal tear is close to the ora serrata, laser up to the ora serrata if you are unable to laser around the anterior edge of the tear

whole tear. Tears close to the ora serrata can be treated by retinopexy around the tear and up to the ora serrata. Retinopexy should be performed soon after the diagnosis has been made, for example, the same day, and subsequent reviews are merely to determine that the retinopexy is adequately encompassing the defect. Retinopexy should be secure after 2 weeks.

Posteriorly placed holes can be treated easily with laser therapy employing a contact lens or a super field lens. More anterior tears require indirect laser ophthalmoscopy and indentation. Alternatively, cryotherapy retinopexy can be applied with a subconjunctival or localised peribulbar local anaesthetic injection in the region of the eye to be treated.

Note: Retinal tears are often multiple (50–60 % of eyes), and, therefore, the surgeon must not be distracted by finding one tear from examining the rest of the retina for any more tears.

4.1.3.2 Atrophic Round Holes

Round holes are often seen in asymptomatic eyes associated with snail track or lattice degeneration. These are not associated with posterior vitreous detachment. The work of Norman Byers suggests that these holes do not need to be treated because any retinal detachment associated with these in an asymptomatic eye is unlikely to be progressive with an approximate risk of 1:200 (Byer 2001, 1982). However, retinal detachment surgeons will have seen many patients who have presented asymptomatically with macular off retinal detachments from such holes (Murakami-Nagasako and Ohba 1983). Therefore, there is a subset of patients that will progress to cause symptomatic retinal detachment. The patient should be made aware of the small risk and symptomatology of retinal detachment.

4.1.3.3 Other Breaks

Paravascular tears can occur often associated with paravascular lattice degeneration (seen in Stickler's syndrome). These tears will produce retinal detachment and should be treated immediately. Other breaks such as dialysis and giant retinal tears usually present with retinal detachment and therefore are not amenable to prophylaxis and will be discussed later.

4.1.3.4 Progression to Retinal Detachment

Any subretinal fluid around the hole indicates that there is a retinal detachment present. A surgical retinal detachment procedure is usually required. If the SRF is very minimal, cryotherapy can sometimes be successful on its own, probably because the tissue swelling induced by the 'burn' causes the hole to close. This however should only be tried very rarely and the retina closely monitored for failure of the procedure.

4.1.4 Peripheral Retinal Degenerations

A number of peripheral degenerations will be seen in the retina.

Associated with retinal break formation:

1. Lattice degeneration is usually an equatorial circumferential hyalinisation of the retinal blood vessels with associated pigmentation giving a crossed pattern and is often associated with round retinal holes and can be associated with U-shaped tears with PVD. It is present in 5 % of eyes but more frequently in moderate myopia (Semes et al. 2001; Celorio and Pruett 1991). Long-term studies suggest that the chance of tractional tears is 2.9 % in 10 years but with very few cases of clinically significant retinal detachment (Byer 1989). Routine treatment of

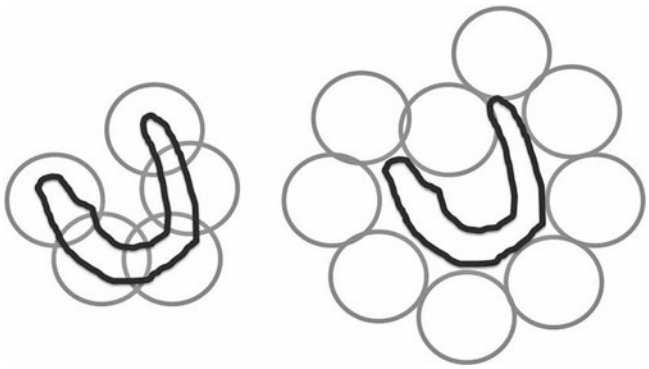

Fig. 4.18 Apply cryotherapy to a large tear as shown on the *left*, although this applies cryotherapy to the bare RPE in the tear; this is preferable to the increased number of burns required if the retina around the tear is treated as shown on the *right*

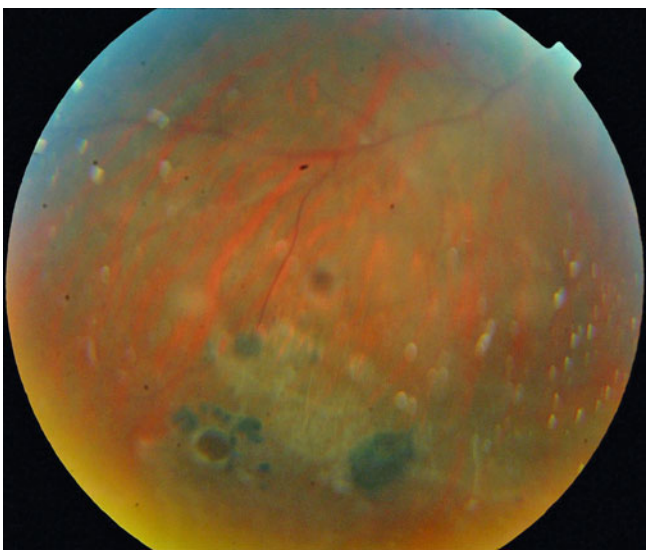

Fig. 4.19 A round hole can be seen in lattice degeneration

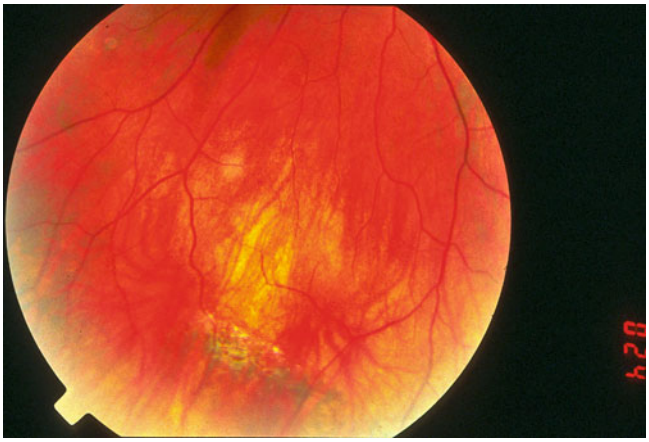

Fig. 4.20 Lattice degeneration is associated with posterior vitreous detachment, U tears and also with round hole retinal detachments with vitreous attached. During vitrectomy, should you encounter large areas of lattice, examine the lattice for any holes within it. It is, however, often safe to leave lattice untreated if you are confident that there are no breaks hidden within it. Partial thickness breaks are commonly seen and require no treatment

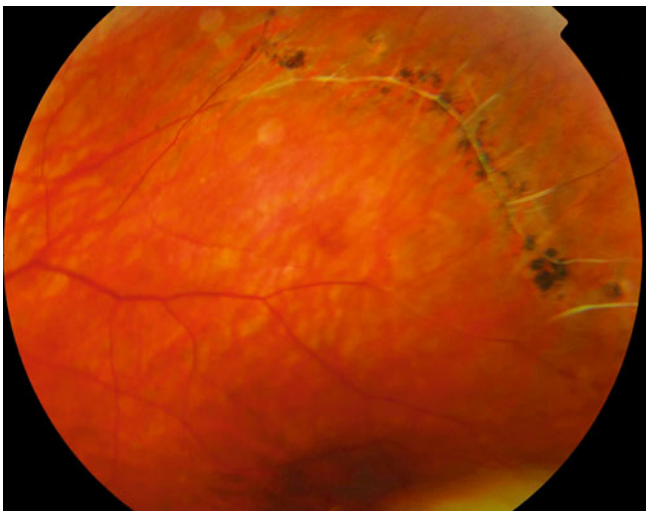

Fig. 4.21 Lattice degeneration

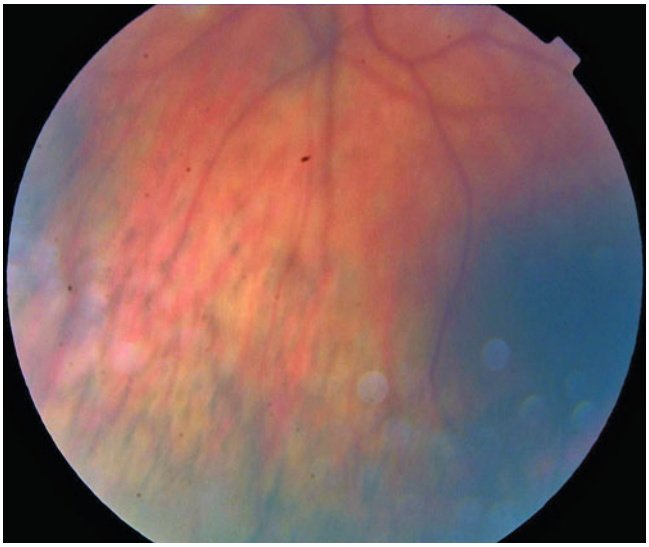

Fig. 4.22 Peripheral reticular degeneration may be associated with age-related macular degeneration

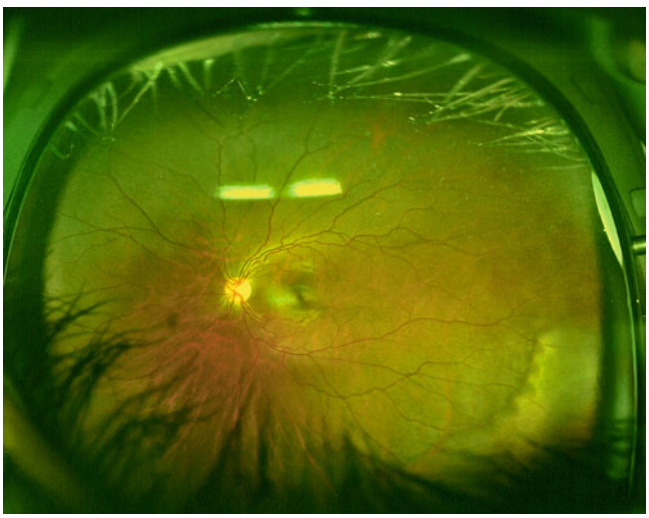

Fig. 4.23 White without pressure is visible in the inferotemporal peripheral retina

lattice is inappropriate because many holes that occur with retinal detachment appear outwith the areas of lattice (Benson et al. 1977). Radial lattice in a paravascular orientation may indicate risk of slitlike paravascular tears. In this case, Stickler's syndrome should be excluded.

2. Snail track degeneration has no pigmentation and is associated with round tears and retinal detachment with attached vitreous gel. This may be more often seen in the black population and is probably a variant of lattice degeneration (Shukla and Ahuja 1981).
   Associated with other retinal conditions:
3. Reticular degeneration is a honeycombed pigmentary change occurring in the aged population and is insignificant apart from an association with age-related macular degeneration (Lewis et al. 1985; Humphrey et al. 1984).
4. Cystoid changes may be seen as yellow flecks, and this can be associated with retinoschisis.
   Others:
5. Cobblestone degeneration is characterised by punched out atrophic areas of depigmentation of the choroid and retina. It is significant in retinal detachment because it may hold back any subretinal fluid, changing the configuration of the retinal detachment.
6. White without pressure shows a crenated edge and pallor of the retina but is not significant of retinal pathology and is often more obvious in highly pigmented fundi. There is a vague association with giant retinal tear formation.

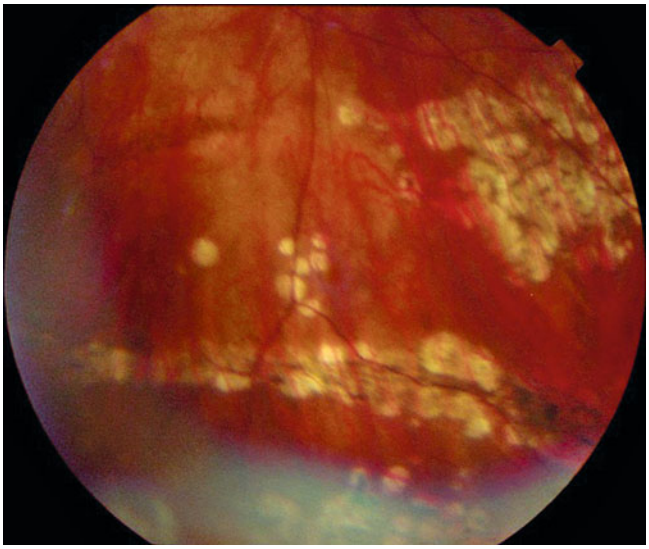

Fig. 4.24 Cobblestone degeneration is commonly encountered by a vitrectomy surgeon and is generally insignificant. Occasionally, the degeneration may cause an adhesion of the retina to the choroid, thereby altering the direction of travel of SRF in retinal detachment. On other occasions, however, it can be split by the retinal detachment, leaving atrophic areas of retina within the retinal detachment itself, which should not be mistaken for retinal holes

7. Ora serrata changes such as oral cysts, bays and meridional complexes may also be seen

Unfortunately, studies on prophylaxis in retinal detachment do not meet the standards of statistical scrutiny (Shukla and Ahuja 1981), and, therefore, there is uncertainty about the correct management of many retinal degenerations. However, in most circumstances, retinopexy of these lesions is not required.

4.2 Summary

Posterior vitreous detachment is a common accompaniment to ageing of the eye. The process is implicated in the causation of a large proportion of vitreoretinal disorders.

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