Société Française d'Ophtalmologie

Monitoring of a patient with signs of posterior vitreous detachment

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Introduction

Posterior vitreous detachment

Posterior vitreous detachment (PVD) is a physiological change occurring in the vitreous, related to the normal aging of the gel structure that progressively liquefies with age: at a certain degree of liquefaction, the vitreous can no longer maintain its shape and collapses by separating first from the posterior pole and then from the retinal periphery up to the more or less extended anterior base of the ora serrata up to the equator. While the posterior vitreous cortex simply adheres to the internal limiting membrane of the posterior pole, the base is an area where the collagen fibers directly penetrating into the internal limiting membrane make the separation impossible.

This physical change occurs in more than 75% of the population over 65 years but may already be seen normally around 40 or 50 years. Although it may be accelerated in some particular cases, PVD is not a sign of illness or eye health problem.

In most cases, PVD is completely asymptomatic but may be a reason for urgent consultation in 15% of cases.

The most common complications to be investigated are retinal tear and intravitreal hemorrhage without tear that may occur acutely at the time of PVD. The other later complications are rather related to an abnormal incomplete vitreous detachment, including vitreoretinal traction syndromes and macular holes.

Review of the literature

We selected 5 publications presenting PVD pathophysiology and signs, its complications and consequences on patient monitoring.

1. Johnson MW. Posterior vitreous detachment: evolution and complications of its early stages. Am J Ophthalmol. 2010;149:371-382.

2. Hollands H[1], Johnson D, Brox AC et coll Acute-onset floaters and flashes: is this patient at risk for retinal detachment? JAMA. 2009; 302:2243-2249

3. Hikichi T. Time course of posterior vitreous detachment in the second eye. Curr Opin Ophthalmol. 2007;18:224-227.

4. Sebag J. Anomalous posterior vitreous detachment: a unifying concept in vitreo-retinal disease. Graefes Arch Clin Exp Ophthalmol. 2004;242:690-698.

5. Brasseur G. Décollement postérieur du vitré in Pathologie du vitré, Rapport de la Société Française d'ophtalmologie, Paris, Masson 2003

Causes of PVD:

Age is the main cause of PVD, through change in the macromolecular structure of the gel that liquefies and collapses (syneresis). This does not automatically result in a separation of the posterior viteous cortex (collapse) which may remain for a long time attached to the posterior pole and, in particular, to the optic disc as shown by the scarcity of the prepapillary ring when examining patients with all signs of PVD or even retinal tears.

The posterior vitreous cortex begins to separate temporally to the fovea and will extend circularly in the perifoveal area. The foveal adherence will then peel off while the posterior vitreous cortex is still attached to the optic disc, and the retinal separation continues in the temporal periphery before evolving to complete vitreous detachment, the remaining vitreous cortex being still attached to the base, somewhere between the retina equator and the ora serrata.

The detachment may be very fast when the vitreous fluid passes through a dehiscence of the posterior vitreous cortex (rhegmatogenous PVD). The separation may otherwise take weeks or months before to be complete.

PVD may be facilitated by aphakia, myopia, trauma, and uveitis but most often, there is a rapid liquefaction without necessarily detachment of the posterior vitreous cortex in these diseases as evidenced by the anatomoclinical confrontations in vitrectomy, since surgeons frequently find some vitreous adherent to the posterior retina.

Symptoms of PVD:

The majority of PVD is asymptomatic but when they occur, the signs are mostly the appearance or sudden worsening of floaters or a diffuse visual discomfort. The signs of retinal adherence or traction (photopsias, phosphenes) are rare.

The risks to find a PVD complication are indirectly correlated with symptoms: the floaters are suggestive of retinal tear, especially if they are associated with localized and quasi-permanent phosphenes.

The existence of a discrete intravitreal hemorrhage, although it should alert, is rather banal (almost 5% of cases) and does not necessarily reflect the existence of a tear. As the adherence of the posterior vitreous cortex is stronger on the retinal vessels, the traction due to PVD may cause a rarely abundant hemorrhage that may prevent any retinal examination. If the hemorrhage is occlusive, it should be considered, until proved otherwise, related to a retinal tear.

Complications of PVD

- Retinal tear is the most common complication, found in about 10% of symptomatic cases and in up to 18% when floaters are associated with phosphenes. In more than 90% of cases, they are located in the upper half of the retina, behind the vitreous base, as a horseshoe tear with a portion of the vitreous attached on the flap. The risk of progression to retinal detachment (RD) is of 30-40% if untreated. However, if no tear is found after 6 weeks of evolution, the risk of secondary retinal damage is very low.
- An intravitreal hemorrhage may also occur in 5-10% of cases, often but not systematically associated with a tear. Vascular tractions causing rarely very dense, often subhyaloid hemorrhages may also occur. They may be replaced by small retinal hemorrhages at the vascular adherence. If the hemorrhage is very dense and may not allow examination of the retinal periphery, vitrectomy should be rapidly scheduled if no resorption begins to be observed after a few days.

Discussion

Only symptomatic PVD requires monitoring until disappearance of initial signs.

The symptoms that lead most often to consult include:

- A sudden appearance of floaters or an increase in their size and number,
- Flashing lights and/or a change in their intensity,
- A blurred vision.

An ophthalmologic examination should then be performed after pupil dilation, to control the condition of the central and peripheral retina, seeking the only clinical sign of PVD which is the visualization of the prepapillary ring floating in the vitreous body, usually in front of the optic disc. The density and mobility of the vitreous should also be assessed.

The only complication of PVD requiring rapid treatment is retinal tear that may, without treatment with retinopexy, lead to retinal detachment in 40% of cases within days of occurrence: patients should therefore be examined very rapidly after the onset of symptoms, since vitreal signs (floaters) are much more common than retinal signs (flashing lights) but if they are associated, the risk is even higher.

If the initial examination shows no retinal anomalies, it should be repeated after one week, especially if flashing lights persist. Patients should be aware of the need for rapidly consult upon appearance of a black veil in all or part of their visual field. If at this 1-week examination, there is still no visible retinal anomaly after pupil dilation, the examination should be repeated after a few weeks and until disappearance of the symptoms. In the absence of retinal damage, the vitreous adherences, reflected by the flashing lights, eventually disappear after a few weeks.

The only additional examination that may be requested in case of suspicion of symptomatic PVD is ultrasound that is only useful in case of fluid disturbance, in particular intravitreal hemorrhage. The ultrasonographer should investigate the presence of a horseshoe tear visible, almost exclusively, in the upper half of the retina.

Patients should be aware that the symptoms, apart from complications, progressively improve over time, over several months, and that a consultation is needed if the initial signs worsen.

Asymptomatic PVD, which is the most common, requires no particular monitoring.

Conclusion:

Posterior vitreous detachment is a normal phenomenon, related to vitreous aging and therefore, its frequency increases with patient age.

It is asymptomatic in almost 85% of cases and requires no particular monitoring. When it is acute and symptomatic, the only fear is the risk of retinal tear and the rapid evolution to retinal detachment if untreated.

It is therefore necessary to rapidly receive patients with suggestive signs, repeat fundus examinations with pupil dilation over several weeks if the signs persist. In case of intravitreal hemorrhage, a rapid ultrasound made by a trained ultrasonographer is the only required additional examination.