Ophthalmologica

Ophthalmologica 2012;227:20-33 DOI: 10.1159/000330421

Received: June 30, 2011 Accepted: June 30, 2011 Published online: September 22, 2011

Retinal Complications after Damaging the Vitreolenticular Barrier

Carsten Framme Sebastian Wolf

University Eye Hospital Bern, Bern, Switzerland

Kev Words

Vitreous · Vitrectomy · Cataract surgery · Retinal detachment · Cystoid macular edema

Abstract

This article gives an overview of the vitreal anatomy and its changes in ageing, which have a significant impact on the two main retinal complications after damage of the vitreolenticular barrier, namely retinal detachment and cystoid macular edema. The possible reasons and pathomechanisms for this entity of retinal diseases in the context of anterior segment interaction are highlighted, and a summary of references is provided showing the epidemiology and consequences of such interventions.

Copyright © 2011 S. Karger AG, Basel

It is well known that damaging the vitreolenticular barrier in an eye frequently results in a bundle of complications such as mainly endophthalmitis in complicated cataract surgery or trauma, or rhegmatogenous retinal detachment (rhRD) and/or cystoid macular edema (CME). However, these adverse reactions appear only in a minority of patients undergoing such events. In most of the patients, these retinal complications are not noticed, neither in the acute situation nor after a longer period of

KARGER Fax +41 61 306 12 34 E-Mail karger@karger.ch

www.karger.com

© 2011 S. Karger AG, Basel 0030-3755/12/2271-0020\$38.00/0

Accessible online at: www.karger.com/oph time. Thus, the question remains which particular conditions of the retina and obviously predominantly of the vitreous lead to such adverse events and which do not. Moreover, it would be interesting to know whether it would be possible to predict retinal complications and to know how to avoid them if a vitreolenticular defect has appeared. In this review, we emphasize the well-known anatomical and pathological behavior of the anterior and posterior vitreous as research in this area took place decades ago and apply this knowledge to the specific condition of vitreolenticular damage. Any lesions of the vitreolenticular barrier in the anterior vitreous may have repercussions on the vitreoretinal zone in the posterior vitreous. The breakdown of the barrier may cause either a volume transfer (of formed vitreous substance) or a chemical transfer (of soluble liquids). In the following, we will describe the anatomy of the anterior vitreous, the diagnostic criteria of an intact and a broken vitreolenticular barrier, and the clinically relevant consequences of defects at the barrier.

This work was presented at the ESCRS/Euretina Clinical Symposium 'The role of the vitreolenticular interface' at the 10th Euretina Congress, Paris, September 2-5, 2010.

Prof. Carsten Framme, FEBO, MHM®, MBA University Eye Hospital Bern Freiburgstrasse 10 CH-3010 Bern (Switzerland) Tel. +41 31 632 9565, E-Mail carsten.framme@insel.ch



Fig. 1. The normal vitreous with the typical vitreous tracts and their corresponding circular zonular ligaments. Displayed are the ora serrata and the preretinal tract (1), the posterior epiciliary ligament and the corresponding posterior ciliary tract (2), the anterior epiciliary ligament and anterior ciliary tract (3), the retrolental ligament and retrolental tract (4) and the area hyaloidea (5). Between structures 1 and 2, there is the absolute and invariable vitreous base. In between points A and B, the posterior vitreous base is variable with its extension depending on age. In fact, it runs posterior towards B in the elderly and stays at A in the young.

Anatomy and Pathology of the Anterior Vitreous

It is undoubtedly true that the vitreous base – its posterior retinal part – plays the most important role in inducing clinical complications in the posterior segment when iatrogenically affected during surgery or trauma. This region of the eye is usually not visible without indentation. It is the most peripheral zone of the retina, the ora serrata, the posterior ciliary body and the vitreous base. The knowledge of the macroscopic and microscopic anatomy of this area has basically not changed since the work of Salzmann in 1912 [1] and Daicker in 1972 [2]. A detailed description of the vitreous anatomy and its examination in the living eye has been given by Eisner in 1973 [3].

The terminology for the vitreous structures varies depending on local traditions. In this paper, the terms chosen are strictly descriptive and the term 'hyaloid' is reserved only for phenomena related to the embryonic hyaloid channel and the hyaloid artery. The vitreolenticular interspace is a virtual space in the normal eye with the anterior vitreous face lying closely against the posterior lens capsule. The anterior vitreous face is formed by a condensation of the framework, the so-called anterior limiting lamina (ALL; synonyms: 'anterior hyaloid', 'anterior vitreous membrane'). In its retrolental section, this lamina is extremely thin and can easily be ruptured when not supported by the lens capsule. The ALL is characterized by the insertion of remnants of the embryonic hyaloid channel running slightly nasally from the posterior pole of the lens (hyaloid area).

The ALL is fixed to the surrounding tissue by radial vitreociliary zonular fibers which emerge from 3 circular zonular ligaments embedded in the anterior vitreous (fig. 1). These are the posterior epiciliary ligament, in the middle of the pars plana from where the ALL originates (synonyms: 'orbiculus ciliaris posterior', 'ligamentum medianum', 'white midline'), the anterior epiciliary ligament, overlying the posterior third of the ciliary processes (synonyms: 'orbiculus ciliaris anterior', 'ligamentum coronarium'), and the retrolental ligament, at the posterior border of the lens (synonyms: 'Wieger's ligament', 'Albrecht's ligament', 'ligamentum hyalocapsulare'). These 3 zonular ligaments are also the origin of the 3 main vitreous tracts, extending to the center of the vitreous: (1) the posterior ciliary tract ('tractus medianus'), (2) the anterior ciliary tract ('tractus coronarius') and (3) the retrolental tract (the widely used term 'hyaloid tract' is based on the erroneous assumption that it is the remnant of the wall of the embryonic hyaloid channel; Eisner's observations, however, have shown that the hyaloid system disappears in early childhood, with the exception of the tiny hyaloid area at the center of the lens, whereas the retrolental tract develops only later in life [3]). A fourth vitreous tract starts from the ora serrata called the preretinal tract. This delineates the preretinal dense vitreous cortex from the semiliquid central vitreous.

The *vitreous base* is the area in which the vitreous is firmly attached. It extends from the ora serrata anteriorly to the middle of the pars plana and posteriorly from the ora serrata towards the equator (fig. 1). The anterior, ciliary vitreous base is invariable, since here the vitreous never detaches and no limiting lamina exists ('absolute vitreous base'). The posterior, retinal vitreous base is variable in its extent and may be recognizable only after a posterior vitreous detachment (PVD). In a young person, the posterior vitreous may detach right to the ora serrata (fig. 1, point A). With increasing age, disseminated vitreoretinal adhesions develop posteriorly to a variable and individual degree [3]. Therefore, a detachment

Retinal Complications after Damaging the Vitreolenticular Barrier

Downloaded from http://karger.com/oph/article-pdf/227/1/20/3353813/000330421.pdf by Universitätsbibliothek Bern user on 21 September 2023

of the posterior limiting lamina (synonyms: 'posterior hyaloid' or 'posterior vitreous membrane') stops somewhere between the ora serrata and the equator (fig. 1, point B). Regarding vitreolental damage this is of particular interest since in a case of PVD the vitreoretinal separation in the young usually extends extremely anteriorly up to the ora serrata, whereas in the elderly, PVD would extend only to the area between the equator and the ora serrata, that is somewhere within the peripheral retina. This means that in the elderly, a PVD rarely reaches the ora serrata. Thus, the risks of complications differ in the young and the elderly.

The almost hidden ALL becomes visible in cases of anterior vitreous detachments (fig. 2), which may be caused by zonular weakness due to e.g. hereditary constitution, pseudoexfoliation or trauma [3]. Such anterior vitreous detachments may indicate the need for investigations of their cause but in themselves have no clinical consequences.

The biomicroscopic examination of the *lens capsule* is usually easy due to the strong reflection of the capsule. This produces intense specular reflexes in the intact lens and in case of extracapsular cataract extraction refraction phenomena in retroillumination at folds and breaks. Regarding the *circular zonular ligaments*, the epiciliary circular zonules become visible by slitlamp examination with scleral depression. The retrolental ligament remains invisible as long as it is attached to the lens. Only when stretched (in case of subluxation or in experiments with autopsy eyes [3]) can the vitreolental zonules be recognized. The circular ligaments appear more distinctly when impregnated by foreign material in case of hemorrhage, in pigment dispersion syndrome and exfoliation syndrome. The extremely thin retrolental ALL is more difficult to examine. Its diagnostic sign is the existence of the hyaloid area with a circular or semicircular remnant of the hyaloid channel and the residue of the hyaloid artery. This hyaloid area is the only reliable sign to distinguish the ALL from other, similarly looking vitreous laminae (pseudo-ALL). The hyaloid area is the sign of the presence of an otherwise invisible retrolental ALL behind a normal lens (fig. 1). The hyaloid area is also the sign proving that the ALL has remained intact in an eye where the posterior lens capsule has been ruptured or is totally absent (fig. 3a). If in such an eye a lamina is observed behind the pupil without a hyaloid area, the ALL has obviously been ruptured, and the lamina in question is a pseudo-ALL – either the pseudo-ALL is the retrolental tract (fig. 3b), proving a moderate shift of vitreous towards the anterior chamber, or it is the posterior limiting lamina (fig. 3c), indicating a major volume shift.



Fig. 2. Various forms of anterior vitreous detachments: 1 = combined anterior and posterior epiciliary detachments; 2 = retrolental detachment (the detached ALL is defined by the presence of the area hyaloidea); 3 = anterior ciliary detachment.

Clinically relevant consequences of a broken anterior vitreous barrier are a volume shift of the vitreous or chemical transfers. The volume shift may cause a posterior vitreous detachment exposing vitreoretinal adhesions in an eye with an attached vitreous. In an eye with a preexisting PVD, a further advancement of the posterior limiting lamina may exert traction more anteriorly leading to an exposure of previously hidden vitreoretinal adhesions. A chemical transfer may cause a transport of solubles from the anterior chamber to the vitreoretinal zone (e.g. inflammatogenic substances, potentially aggressing the macular area), or an invasion of vitreous material into the anterior chamber (e.g. hyaluronic acid, potentially affecting the intraocular pressure). Both mechanisms will be discussed in more detail below.

Influence of PVD

According to the nature of the volume transfer from the vitreous into the retrovitreal space, one can distinguish between a rhegmatogenous and nonrhegmatogenous vitreous detachment [4]. In a *rhegmatogenous posterior vitreous detachment (rhPVD)*, which is the typical

Color version available online





Fig. 3. ALL and pseudo-ALLs in case of rupture of the lens capsule. **a** The ALL has remained intact, the area hyaloidea is still in situ; 1 = margin of the ruptured lens capsule; 2 = area hyaloidea. **b** The ALL has been ruptured, and free vitreous substance has prolapsed into the anterior chamber (AC); 1 = the pseudo-ALL seen behind the pupil is the retrolental tract. **c** There is a major vitreous prolapse into the anterior chamber (AC); 1 = the pseudo-ALL behind the pupil is now the posterior limiting lamina; if in such a case free vitreous is observed behind the posterior limiting membrane, there is a prolapse of substance having passed through the prefoveal hole in combined PVD.

condition in the elderly, there is a hole in the posterior hyaloid membrane (nearly always a prefoveal hole) through which the destroyed, liquefied vitreous can pass abruptly into the newly formed retrovitreal space (fig. 4) and due to the rapidity of this process retinal hole formation at vitreoretinal adhesions may appear [3, 4]. Thus, rhPVD mostly promotes rhegmatogenous detachment due to horseshoe tears in the midperipheral retina. Since the transfer hole is always in the same position, the rhPVD configuration practically always looks similar on biomicroscopic examination [3, 4]. The main mass of vitreous rests in the inferior half of the eye and the course of the vitreal tracts is significantly altered since they run – as in all perforations of the vitreous body – straight towards the cortex hole [3, 4].

In a *non-rhPVD*, volume transfer is effected through an 'intact' posterior hyaloid membrane [3, 4]. Details of this phenomenon are still unknown. The process is slow and may lead to a small partial PVD or may also eventually develop a complete detachment [3, 4] (fig. 5). The vitreoretinal connections dissolve slowly, and where the vitreous remains attached, the retina is drawn into folds

Retinal Complications after Damaging the Vitreolenticular Barrier



Color version available online



Fig. 4. Typical aspect of an rhPVD. Fluid from the vitreous body enters the subvitreal space through the prefoveal hole. The main vitreous body rests on the lower half of the globe. The inferiorly settled posterior hyaloid membrane is covered by the prolapsed vitreal substance. The vitreal tracts run straight to the hole (image from Eisner [5]).

Fig. 5. Variable aspects of non-rhPVD. a Subtotal non-rhPVD. b Subtotal nonrhPVD with a hernia of intact vitreous at the prefoveal hole of the posterior limiting lamina. c Subcomplete non-rhPVD with a fold along the posterior margin of the vitreous base. d Partial non-rhPVD with a persistent vitreoretinal adhesion at the posterior pole. In all forms of the slow nonrhPVD process, vitreoretinal traction appears at various locations (image from Eisner [5]).

rather than torn [3, 4]. This might also lead to CME if traction appears at the macula itself. Since the vitreous body remains intact, the vitreal tracts maintain their typical shape, at least in those parts of the vitreous which are not affected by shrinkage [3, 4].

The PVD is a very important clinical condition, since the retina is no longer protected by the broad, stable vitreous cortex shielding it from the vigorous movements of the central vitreous [3, 4]. However, the main complications derive from the fact that there is now vitreous trac-



→ No complications!

Fig. 6. Theoretical aspects of an ALL rupture in cases of attached vitreous and no vitreoretinal adhesions. This condition might lead to a 'normal' rhPVD.

tion directly affecting the retina. It is only after PVD that a dense 'lamina' is formed, inserting at the retina itself, and this – as explained above – much more posteriorly in the elderly than in the young, where no retinal insertion may occur. Prior to the PVD, no laminae insert at the retina, since all normal vitreal tracts reach the inner wall of the globe anterior to the ora serrata in a region protected against tractional forces by adhesions between the ciliary epithelium and the pigment epithelium at the pars plana [3–5]. The traction affecting the retina and leading to the numerous and well-known complications such as mainly retinal detachment does not occur by the vitreous tract but by the posterior hyaloid membrane [6].

For the prognosis of retinal diseases due to PVD, it is important to determine precisely – if possible – whether vitreous detachment is complete, since no further complications are to be expected once the detachment process has come to a standstill. Then no further tearing is likely and if no retinal lesions have been produced by the PVD so far, the patient may be dismissed from observation [3]. However, if – manipulating the eye – due to a vitreolenticular opening the PVD process might be initialized again, especially in older patients when PVD normally does not reach the ora serrata, then new but more anterior break formation might be induced.

Lesions of the Vitreolenticular Barrier

Based on the previous explanations, it is now interesting to discuss how the vitreous in its variable appearance is theoretically acting in cases of damaging the vitreolenticular interface. For this it is important to consider that this opening means conclusively the rupture of the ALL and therefore definite affection of the vitreous body. One should note that opening the posterior lenticular capsule alone without affecting the ALL is surgically possible and for example performed in the 'optic buttonholing' technique for implanting the haptics of an intraocular lens (IOL) into the bag and shifting the IOL optics behind the posterior capsule and anteriorly to the 'intact' ALL, thus preventing lens epithelial cell migration [7]. Whether this attempt was successful becomes apparent by the presence of the hyaloid behind the IOL at its specific location. In case of rupture of the ALL, however, usually the vitreous immediately enters the area of the capsular bag and/or the anterior chamber of the eye. Thus, a vitreal shifting to the anterior part of the eye is obviously leading to tractional forces in the posterior segment of the eye. Herein, several conditions of vitreal-retinal interaction might be now imaginable.

In an eye with a completely attached vitreous body and no vitreoretinal adhesions in the midperipheral retina, the rupture might induce a 'simple' rhPVD up to the vitreous base and there especially up to the zone of 'denticulation', which is – as explained above – more posterior and with retinal contact in the elderly but predominantly without retinal contact up to the ora serrata in young people. For the latter ones, trauma would be a possible condition to induce such rhPVD. If in cases of complete rhPVD no vitreoretinal adhesions are present, no further retinal complications should be expected (fig. 6). In this context it might be noteworthy that also in uncomplicated cataract surgery the rate of PVD is signifi-





Fig. 7. Theoretical aspects of an ALL rupture in cases of a preexisting stabilized rh-PVD. In this condition, an anterior vitreal shift and an enhanced vitreous collapse might lead to an anterior movement of the insertion of the posterior lamina. New, more anterior adhesions may become exposed to traction and develop breaks in areas previously considered 'safe'.

Fig. 8. Theoretical aspects of an ALL rupture in cases of a preexisting non-rhPVD. In this condition, there are usually vitreoretinal adhesions in various locations. The anterior vitreal shift would increase traction in these areas and might induce and enlarge retinal folds.

cantly higher than in phakic eyes. Hilford et al. [8] observed a PVD rate of 50.8% in pseudophakic eyes versus 20.8% in phakic eyes. This can be explained by the increase in 'vitreal space' in the posterior segment after extracting the voluminous lens and exchanging it by a flat artificial lens. Due to this, enhanced vitreal movement might lead to PVD.

If the vitreolenticular opening appears in a situation of a preexisting stabilized rhPVD, enhanced vitreous collapse might appear and the insertion of the posterior vitreous lamina starts to move anteriorly (fig. 7). If there are no vitreoretinal adhesions in these anterior parts, no complications would appear; however, if adhesions exist – especially in this area of strong denticulation – new retinal breaks must be expected. These breaks would then be located more anteriorly than usually seen in 'classic' rhegmatogenous detachments; thus, in these cases it is consequently important to search for such breaks more anteriorly than 'usual'. This condition also explains why it can be important to carefully determine the exact appearance of the vitreous body preoperatively.

In cases of non-rhPVD vitreoretinal adhesions usually exist. An opening of the vitreolenticular interface leading to an anterior volume shift of the vitreous could consequently increase traction on adhesions, which leads to a progression of retinal folds (fig. 8). This progression

Color version available online



ture in cases of rhPVD with posterior prefoveal vitreal prolapse having remained attached to the macular area. Due to the anterior vitreal shift, traction may occur in the macular area with the risk of CME.

Fig. 9. Theoretical aspects of an ALL rup-



Fig. 10. Spectral-domain OCT image of vitreomacular traction clearly showing the adherent vitreal surface to the retina leading to intraretinal cysts, edema and macular thickening.

might induce a tractional RD; however, this regularly appears more slowly than an rhRD and is a rare condition.

Vitreoretinal adhesions might also exist in the macular region in cases of rhPVD or non-rhPVD. If a rupture of the ALL with a consequent vitreal volume shift towards the anterior eye segment appears in this condition, enhanced traction towards the macula may result leading to a vitreomacular traction syndrome and predominantly CME (fig. 9). Such vitreomacular tractions can be displayed nowadays in an easy noninvasive way using spectral-domain optical coherence tomography (OCT) clearly demonstrating the pinpoint attaching the vitreous to the retinal surface and the traction leading to the retinal elevations and CME (fig. 10).

Macular complications after opening the ALL might not only appear due to mechanical vitreous traction on the retina surface, but also by chemical problems (fig. 11). It is obvious that due to the anterior vitreal shift substances from the posterior segment might easily enter the anterior chamber of the eye, as e.g. hyaluronic acid. On the



Fig. 11. Theoretical aspects of an ALL rupture regarding biochemical alterations of vitreal conditions. Herein, hyaluronic acid and possibly other substances may reach the anterior chamber; anterior fluids may reach the posterior segment as well. Also topical drugs might penetrate better into the posterior segment.

other hand, also anterior fluids might easily enter the posterior segment of the eye, possibly inducing CME. In this context, also topically applied drugs might penetrate better into the posterior segment but the question arises whether for this there are differences between attached versus detached vitreous body. This must remain unclear. However, it seems to be undoubtedly true that a CME can be triggered by such biochemical processes (e.g. prostaglandins) as it might also be treated successfully by nonsteroidal anti-inflammatory drugs [9]. Thus, chemical pathways in inducing such macular pathology definitely exist. In fact, even after uneventful cataract surgery and intact capsular support - but without implanting an IOL - diffusion of tracer molecules from vitreous to aqueous was measured and even more enhanced by opening the posterior capsule; however, this was tested in a rabbit model and not in humans [10]. Similar observations in humans using fluorescein underline these findings showing enhanced diffusion of fluorescein into the vitreous in patients undergoing posterior capsulotomy [11].

In the context of CME induction especially Nd:YAG capsulotomy is an important and frequent procedure for opening the vitreolenticular interface. In fact, in daily praxis the laser surgeon surely aims at the posterior lens capsule; however, if in doubt it is usually tried not to achieve lens pits possibly leading to visual disturbances if applied in the center of the lens. Then laser focusing

might be more posterior than anterior in respect to the position of the posterior lens capsule. Presumably, today in any of the cases ALL is affected by Nd:YAG capsulotomy, then possibly leading to 'chemical interaction' between the anterior and posterior segments of the eye or even to vitreal shift if e.g. IOL luxation appears [12]. It might be suspected that the complication rate of inducing CME or rhRD after Nd:YAG capsulotomy is significantly reduced, if the ALL can be preserved during the intervention. However, as today's IOL designs predominantly have a biconvex shape, they usually reveal a stable attachment between the posterior lens capsule and the ALL with no possibility of preservation of the anterior vitreous in case of laser treatment. Interestingly, ALL preservation during capsulotomy was frequently possible decades ago using different IOL shapes such as planoconvex and concave-convex designs (fig. 12). In these two forms, the posterior lens capsule was normally constricted in a perpendicular direction towards the nonconvex posterior side of the IOL, thus dissolving from the ALL and leaving a larger retrolenticular space in front of the ALL. In this particular condition Nd:YAG capsulotomy would be possible without affecting the ALL, thus avoiding an opening of the vitreolenticular interface [the success of this procedure is visible - again - by the presence of the area hyaloidea behind the IOL (pers. commun. G. Eisner)].



Fig. 12. Theoretical aspects of a possible ALL preservation in Nd:YAG capsulotomy depending on different designs of IOLs implanted in the bag. **a** Biconvex IOL: no vitreolenticular interspace, the lens capsule as well as the ALL are closely apposed to the IOL. **b** Planoconvex IOL: the posterior capsule is slightly stretched and is apposed to the IOL; the ALL has retained its size and thus a small interspace exists now. **c** Concave-convex IOL: anteriorly as well as posteriorly from the stretched lens capsule, a fluid-containing interspace has developed, facilitating maneuvers at the lens capsule without damaging the ALL.

Rhegmatogenous Retinal Detachment

In the general population, the incidence of a phakic rhRD is estimated to be about 0.01% depending on axial length, lattice degeneration and history of RD in the fellow eye [13]; however, the risk increases up to 3.6% in uneventful extracapsular cataract surgery and implantation of a posterior chamber IOL [14, 15]. In an event of intraoperative capsular break and vitreous loss, the risk of suffering rhRD increases to 5–15% [13, 14, 16, 17].

Complicated cataract surgery is therefore one of the major risk factors for inducing rhRD; however, it is also reasonable in this context to consider that even in uncomplicated cataract surgeries the cumulative 'lifetime' risk for RD is about a factor of nearly 9 higher than in nonoperated eyes revealing a higher risk in younger ages of surgery [18]. This might be explained - as already discussed above - by the higher rate of PVDs in the pseudophakic eye [8] leading to altered vitreous geometry offering enhanced vitreal moving possibilities. This would also explain the higher risk of RD even years after surgery if a PVD occurred after the operation but not necessarily before. Approximately 10-15% of patients with symptomatic PVD suffer from retinal breaks [19]. Interestingly, a meta-analysis by Coffee et al. [20] showed an overall incidence of retinal breaks of 21.7% after PVD ranging from 8.2 to 47.6% evaluating 10 studies. As also underlined by Williams et al. [19] and others [21, 22],

there are well-known risk factors for the detection of retinal breaks following PVD as e.g. pigmented vitreous granules, vitreous hemorrhage, myopia, underlying retinal pathology, trauma and pseudophakia. However, the risk factors for delayed retinal breaks are less well published but also observed in the meta-analysis of Coffee et al. revealing vitreous hemorrhage, retinal hemorrhage or new symptoms [19, 20]. Such delayed retinal breaks were described in 1.5-2.1% of cases with uncomplicated PVD [20, 23], but this rate was considered to be much higher showing 3.95% up to 6 weeks after symptomatic PVD [19]. The latter results were obtained independently from prior cataract surgery, implicating possibly a higher risk for pseudophakic conditions. Therefore, it follows that after each status change of the vitreous condition, a careful follow-up examination at least up to 6 weeks after incidence should be undertaken looking for retinal breaks and to prevent RD by early intervention as laser or cryopexy. As described by Ripandelli et al. [24], RD was observed in 11 of 148 patients (7.4%) with a PVD onset after cataract surgery and in only 3 out of 265 patients (1.1%) with a PVD existing preoperatively. This underlines the hypothesis that, if a preexisting rhPVD is stabilized and no anterior vitreal shift as in complicated cataract surgery appears, no new breaks are to be expected. However, if the rhPVD is incomplete or anterior vitreal shift happens, new breaks might occur. Since the vitreous base inserts - as described above - more posteriorly





Fig. 13. Theoretical aspects of an anterior vitreal shift in cases of preexisting stabilized rhPVD. Due to the vitreal shift, the insertion line of the posterior limiting lamina moves anteriorly exhibiting new vitreoretinal adhesions. Thus, new breaks may develop more anteriorly in the area where previously the vitreous has been safely attached to the retina (image from Eisner [5]).

in the elderly showing a broader area of denticulation, new holes after preexisting PVD might appear much more anteriorly in the described condition. Usually, these breaks are smaller and due to the peripheral location often not easy to identify; thus, in up to one third of all cases suffering from pseudophakic RD, no definite break can be evaluated preoperatively [25]; nevertheless, careful examination is mandatory.

Regarding pseudophakic RD in complicated cataract surgery, intraoperative risk factors consist of capsular rupture, nucleus loss and associated vitrectomy [26]. Blomquist and Rugwani [27] reported a rate of complicated cataract surgeries including vitreous loss of 4.5% in 1,400 surgeries done by residents, and even for experienced surgeons the rate of intraoperative vitreous loss seems to be as high as up to 5.6% [28]. This underlines the still present impact of complicated cataract surgery even with the present-day 'less invasive' phacoemulsification and microincision techniques. As stated above, the risk of inducing rhRD after complicated cataract surgery is very inhomogeneous ranging from 5 to 15% [13, 14, 16, 17]. In the study by Blomquist and Rugwani [27], no RD was observed in 63 complicated cases in spite of the fact that the mean follow-up was nearly 1 year, which was considered to be adequately long. Thus, the observation time seems

to play a major role making it unfeasible to compare different study results with their heterogeneous baseline and follow-up criteria. However, what becomes clear reviewing the literature is that the status of the vitreous is nearly never documented before cataract surgery. Even in large population-based cohorts, it is explained that PVDs were not documented although the preoperative status of the vitreous is considered a highly important risk factor for pseudophakic RD [29]. Due to these circumstances it appears difficult to differentiate the relative importance of the vitreal status as the possibly main risk factor regarding the other known risk factors such as myopia, lattice degeneration or fellow eye pathology in clinical settings. However, it is discussed that the presence of a PVD in older patients ('preexisting stabilized rhPVD' - see above) before cataract extraction may protect against subsequent RD by limiting the transmission of forces to the retina that are produced during surgery [29]. Furthermore, it was observed that pseudophakic eyes have alterations in the vitreous humor structure that are not seen in phakic eyes [29, 30]. Alterations such as proteome differences, viscosity and the size distribution of macromolecules may also contribute to the development of retinal complications following regular cataract surgery [30].

In this context, the incidence of RD following Nd:YAG capsulotomy is controversially discussed. Some studies did not find a higher risk [29], others found a significantly increased risk of about a factor of 4 [31]. About 80% of such studies report a positive correlation; however, a comparison is difficult because of different baseline criteria and also enhanced operation techniques during the years. Ranta et al. [32] observed rhRD in 8 out of 350 cases (2.3%) within 5 years after Nd:YAG capsulotomy; however, 6 out of these 8 eyes revealed additional risk factors for RD as e.g. long axial length, history of RD, posterior capsule rupture and vitreous loss during cataract surgery. Interestingly, they also found consistent data for the hypothesis that roughly one half of pseudophakic RDs after Nd:YAG posterior capsulotomy result from horseshoe tears and the other half from potentially antecedent small atrophic holes [32, 33]. For the latter fact, again an anterior vitreal shift in the area of the 'denticulated' vitreal base might be the explanation for RD due to atrophic holes (fig. 13); however, also in these studies the status of the vitreous condition was not documented. On the other hand, a considerably 'low' rate of about 2% in 5 years might also be a hint for the firm attachment between the vitreous and underlying tissue within the vitreous base. A most recent prospective study of Burg and Taqui [34] in an Asian population with enhanced myopic refraction with respect to the Western world revealed an RD rate of 1.9% (n = 104) within 12 months after Nd:YAG capsulotomy, which seems to be in the range of other studies; however, regarding the previously discussed study by Ranta et al. [32], the observation time was considerably short. The importance of this follow-up time in terms of the 'normal' frequency of suffering RD must remain unclear. Thus, even today, the 'problem' of Nd:YAG capsulotomy towards RD seems to be controversial.

Cystoid Macular Edema

In the study by Burg and Taqui [34], the incidence of CME after Nd:YAG capsulotomy was 9.6%. After uneventful extracapsular cataract surgery, CME occurs in 0.1–2.35% of all cases (Irvine-Gass syndrome) [35]. However, after opening the vitreolenticular interface, this incidence significantly rises up to 21% in cases of complicated cataract surgery with vitreous loss [36, 37]. These studies were published in the 1990s, and since then the surgical technique has improved; thus, it is believed that the incidence of CME might be reduced nowadays. On the other hand, imaging techniques have also been enhanced, and CME can be detected in a very sensitive manner using today's OCT technique [38]. Interestingly, using OCT, a thickening of the macular volume of about 10% was detected in 20% of eyes following uneventful cataract surgery [39]. This indicates that even without opening the ALL significant alterations within the vitreal environment must exist as already shown by Neal et al. [30] and that detecting a CME is also a kind of definition. It is a difference whether this pathological condition is seen clinically, fluorescein angiographically or by OCT and more enhanced using the spectral-domain OCT technique [38].

Today a CME occurs in about 10% of all cases undergoing cataract extraction with vitreous loss, which seems to be the same rate as after Nd:YAG capsulotomy [27, 34]. A publication by Ang and Whyte [40] in 2006 described the outcomes of posterior capsular rupture in a district general hospital setting showing a CME rate of only 2.2%. This shows the general heterogeneity of appearance and/ or diagnosis of CME in clinical settings. In fact, the authors discussed their unexpected low CME rate as an eventual failure of diagnosing all cases of CME [40]. This might also be explained by the CME expression representing mild to severe forms itself, and also by the time points of follow-up examination after surgery; thus, 'missing' of proper diagnoses seems possible.

In spite of the fact that a lot of studies deal with the problem of CME after complicated cataract surgery, the status of vitreous appearance is not reproducibly documented. Therefore, vitreomacular traction as described above has not been described in a larger patient group as a main reason for CME. This might change in future using the spectral-domain OCT technique as a noninvasive measurement in even miotic eyes to determine possible macular traction preoperatively. In cases of vitreal strands after complicated cataract surgery within the anterior chamber coexisting with CME, a revision and removal of these strands by e.g. anterior vitrectomy is usually indicated and CME also usually disappears after the intervention. However, also in these patients not only the tractional forces on the macula might be the reason for CME but also biochemical alterations. In fact, it is believed that CME after capsular rupture is mostly attributed to chemical reactions in the vitreous as already displayed in figure 11. The exact pathomechanisms are still unclear; however, inflammatory conditions are discussed. Since the incidence of CME is reported to be higher also in patients receiving chronic topical glaucoma medication [41], in diabetic patients [42] and after any operative procedures including laser capsulotomy [43], the pathophys-

iological mechanism is believed to be anterior segment inflammation with release of endogenous inflammatory mediators including prostaglandins [44]. Findings of increased levels of prostaglandins after cataract surgery [45] support this theory. Nonsteroidal anti-inflammatory drugs, which inhibit the production of prostaglandins by blocking the cyclooxygenase cycle, are effective in treating CME after cataract surgery [44, 46, 47]. Since therefore also prostaglandin therapy of chronic glaucoma was accused of inducing CME, more careful evaluations in such studies mainly revealed other cofactors such as aphakia, pseudophakia with ruptured lens capsule, history of uveitis or retinal inflammatory or vascular disease to be predominantly responsible for CME [44]. Thus, the most important factor for this condition still seems to be the opening of the vitreolenticular interface - as an intraoperative complication or by Nd:YAG capsulotomy - but mostly based on the described biochemical rather than a tractional mechanism.

Conclusion

Opening the vitreolenticular interface might lead to significant changes in the vitreal structure and in the biochemical conditions of the eye. The latter one seems to be the main risk factor for inducing CME. Regarding the vitreal structure it seems to be reasonable to differentiate attached vitreous conditions versus rhPVDs and nonrhPVDs, which might lead to different reactions in cases of interface ruptures and vitreous loss. For the typical rhPVD, an anterior vitreal shift would result in a concomitant anterior movement of the vitreal base being more posterior in the elderly. Possible existing vitreoretinal adhesions might then be newly exposed leading to the creation of new breaks and/or vitreomacular traction. Retinal detachment is one of the severest complications in this entity of cases.

Therefore, even nowadays – having fine and safe surgical instruments and procedures and expected low complication rates – careful indication for cataract surgery and Nd:YAG capsulotomy is recommended. A careful documentation of the vitreal status before and after cataract extraction should be mandatory to be able to define a certain risk for retinal breaks and/or RD during followup. A status change of the vitreous in the time context of the surgery is an alarm signal, and a closer follow-up should be advised. In these cases a careful search for 'new' and more anterior breaks is necessary to prevent retinal detachment.

Acknowledgement

The authors thank Prof. Dr. Georg Eisner for his outstanding contributions to this review, including particularly the parts of the vitreal anatomy and the schematic drawings. This paper could not have been written without his encouraging support.

Disclosure Statement

No financial interests.

References

- Salzmann M: Anatomie und Histologie des menschlichen Augapfels. Leipzig, Deuticke, 1912.
- 2 Daicker B: Anatomie und Pathologie der menschlichen retinociliaren Fundusperipherie. Basel, Karger, 1972.
- 3 Eisner G: Biomicroscopy of the peripheral fundus. Berlin, Springer, 1973.
- 4 Eisner G. Autoptische Spaltlampenuntersuchung des Glaskörpers I–III. Graefes Arch Klin Exp Ophthalmol 1971;182:1–40.
- 5 Eisner G: Dr. Sherlock's vitreous: an introduction to biomicroscopy in the clinical examination of the vitreous. DVD supplement to the Goldmann Lecture 2008. Heerbrugg, Schweizerische Ophthalmologische Gesellschaft, 2008.
- 6 Tasman W: Posterior vitreal detachment and peripheral retinal breaks. Trans Am Acad Ophthalmol Otolaryngol 1968;72:217–224.
- 7 Menapace R: Posterior capsulorhexis combined with optic buttonholing: an alternative to standard in-the-bag implantation of sharp-edged intraocular lenses? A critical analysis of 1,000 consecutive cases. Graefes Arch Clin Exp Ophthalmol 2008;246:787– 801.
- 8 Hilford D, Hilford M, Mathew A, Polkinghorne PJ: Posterior vitreous detachment following cataract surgery. Eye 2009;23:1388– 1392.
- 9 Lee MS, Lass JH: Rapid response of cystoid macular edema related to Nd:YAG laser capsulotomy to 0.5% ketorolac. Ophthalmic Surg Lasers Imaging 2004;35:162–164.

- 10 Thompson JT, Glaser BM: Role of lensectomy and posterior capsule in movement of tracers from vitreous to aqueous. Arch Ophthalmol 1985;103:420-421.
- 11 Ohrloff C, Schalnus R, Rothe R, Spitznas M: Role of the posterior capsule in the aqueousvitreous barrier in aphakic and pseudophakic eyes. J Cataract Refract Surg 1990;16: 198–201.
- 12 Framme C, Hoerauf H, Roider J, Laqua H: Delayed intraocular lens dislocation after neodymium: YAG capsulotomy. J Cat Refr Surg 1998;24:1541–1543.
- 13 Wilkinson CP, Rice TA: Michel's Retinal Detachment, ed 2. St Louis, Mosby, 1997.

- 14 Coonan P, Fung WE, Webster RJ Jr, et al: The incidence of retinal of retinal detachment following extracapsular cataract extraction; a ten-year study. Ophthalmology 1985;92: 1096–1101.
- 15 Olsen G, Olson RJ: Update on a long-term, prospective study of capsulotomy and retinal detachment rates after cataract surgery. J Cataract Refract Surg 2000;26:1017–1021 (erratum published in J Cataract Refract Surg 2001;27:180).
- 16 Javitt JC, Tielsch JM, Canner JK, et al: National outcomes of cataract extraction – increased risk of retinal complications associated with Nd:YAG laser capsulotomy: the Cataract Patient Outcomes Research Team. Ophthalmology 1992;99:1487–1497.
- 17 Norregaard JC, Thoning H, Anderson TF, et al: Risk of retinal detachment following cataract extraction: results from the International Cataract Surgery Outcomes Study. Br J Ophthalmol 1996;80:689–693.
- 18 Boberg-Ans G, Henning V, Villumsen J, la Cour M: Longterm incidence of rhegmatogenous retinal detachment and survival in a defined population undergoing standardized phacoemulsification surgery. Acta Ophthalmol Scand 2006;84:613–618.
- 19 Williams KM, Watt L, Williamson TH: Acute symptomatic posterior vitreous detachment and delayed retinal breaks. Acta Ophthalmol 2011;89:e100-e101.
- 20 Coffee RE, Westfall AC, Davis GH, et al: Symptomatic posterior vitreous detachment and incidence of delayed retinal breaks: case series and meta-analysis. Am J Ophthalmol 2007;44:409–413.
- 21 Sharma S, Walker R, Brown GC, et al: The importance of qualitative vitreous examination in patients with acute posterior vitreous detachment. Arch Ophthalmol 1999;117: 343–346.
- 22 Grand MG: The risk of a new retinal break or detachment following cataract surgery in eyes that had undergone repair of phakic break or detachment: a hypothesis of a causal relationship to cataract surgery. Trans Am Ophthalmol Soc 2003;101:329–364.
- 23 Richardson PS, Benson MT, Kirby GR: The posterior vitreous detachment clinic: do new retinal breaks develop in six weeks following an isolated symptomatic PVD? Eye 1999;13: 237–240.

- 24 Ripandelli G, Coppe AM, Parisi V, Olzi D, Chiaravalloti A, Stirpe M: Posterior vitreous detachment and retinal detachment after cataract surgery. Ophthalmology 2007;114: 692–697.
- 25 Feltgen N, Weiss C, Wolf S, et al: Scleral buckling versus primary vitrectomy in rhegmatogenous retinal detachment study (SPR study): recruitment list evaluation. Study report No 2. Graefes Arch Clin Exp Ophthalmol 2007;245:803–809.
- 26 Lois N, Wong D: Pseudophakic retinal detachment. Surv Ophthalmol 48:467–487.
- 27 Blomquist PH, Rugwani RM: Visual outcomes after vitreous loss during cataract surgery performed by residents. J Cataract Refract Surg 2002;28:847–852.
- 28 Ng DT, Rowe NA, Francis IC, et al: Intraoperative complications of 1,000 phacoemulsification procedures: a prospective study. J Cataract Refract Surg 1998;24:1390–1395.
- 29 Erie JC, Raecker ME, Baratz KH, Schleck CD, Robertson DM: Risk of retinal detachment after cataract extraction, 1980–2004: a population based study. Trans Am Ophthalmol Soc 2006;104:167–175.
- 30 Neal RE, Bettelheim FA, Lin C, Winn KC, Garland DL, Zigler JS Jr: Alterations in human vitreous humour following cataract extraction. Exp Eye Res 2005;80:337–347.
- 31 Tielsch JM, Legro MW, Cassard SD, Schein OD, Javitt JC, Singer AE, Bass EB, Steinberg EP: Risk factors for retinal detachment after cataract surgery: a population-based casecontrol study. Ophthalmology 1996;103: 1537–1545.
- 32 Ranta P, Tommila P, Kivelä T: Retinal breaks and detachment after neodymium: YAG laser posterior capsulotomy: five-year incidence in a prospective cohort. J Cataract Refract Surg 2004;30:58–66.
- 33 Ranta P, Kivelä T: Retinal detachment in pseuophakic eyes with and without Nd:YAG laser posterior capsulotomy. Ophthalmology 1998;105:2127–2133.
- 34 Burg MA, Taqui AM: Frequency of retinal detachment and other complications after neodymium: YAG laser capsulotomy. J Pak Med Assoc 2008;58:550–552.
- 35 Loewenstein A, Zur D: Postsurgical cystoid macular edema. Dev Ophthalmol 2010;47: 148–159.
- 36 Frost NA, Sparrow JM, Strong NP, Rosenthal AR: Vitreous loss in planned extracapsular cataract extraction does lead to a poorer visual outcome. Eye 1995;9:524–427.

- 37 Balent A, Civerchia LL, Mohamadi P: Visual outcome of cataract extraction and lens implantation complicated by vitreous loss. J Cataract Refract Surg 1988;14:158–160.
- 38 Wolf S, Wolf-Schnurrbusch U: Spectral-domain optical coherence tomography use in macular diseases: a review. Ophthalmologica 2010;224; 333–340.
- 39 Degenring RF, Vey S, Kamppeter B, et al: Effect of uncomplicated phacoemulsification on the central retina in diabetic and non-diabetic subjects. Graefes Arch Clin Exp Ophthalmol 2007;245:18–23.
- 40 Ang GS, Whyte IF: Effect and outcomes of posterior capsule rupture in a district general hospital setting. J Cataract Refract Surg 2006;32:623–627.
- 41 Handa J, Henry JC, Krupin T, et al: Extracapsular cataract extraction with posterior chamber lens implantation in patients with glaucoma. Arch Ophthalmol 1987;105:765– 769.
- 42 Pollack A, Leiba H, Bukelman A, et al: Cystoid macular oedema following cataract extraction in patients with diabetes. Br J Ophthalmol 1992;76:221–224.
- 43 Keates RH, Steinert RF, Puliafito CA, et al: Long-term follow-up of Nd:YAG laser posterior capsulotomy. J Am Intraocul Implant Soc 1984;10:164–168.
- 44 Alm A, Grierson I, Shields MB: Side effects associated with prostaglandin analog therapy. Surv Ophthalmol 2008;53(suppl 1):93– 105.
- 45 Miyake K, Sugiyama S, Norimatsu I, et al: Prevention of cystoid macular edema after lens extraction by topical indomethacin (III) radioimmunoassay measurement of prostaglandins in the aqueous during and after lens extraction procedures. Graefes Arch Klin Exp Ophthalmol 1978;209:83–88.
- 46 Flach AJ, Jampol LM, Weinberg D, et al: Improvement in visual acuity in chronic aphakic and pseudophakic cystoid macular edema after treatment with topical 0.5% ketorolac tromethamine. Am J Ophthalmol 1991; 112:514–519.
- 47 Weisz JM, Bressler NM, Bressler SB, et al: Ketorolac treatment of pseudophakic cystoid macular edema identified more than 24 months after cataract extraction. Ophthalmology 1999;106:1656–1659.