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**Clinical Guideline for Finnish Optometrists – Assessment of Posterior
Vitreous Detachment**

Research Development

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Master's Thesis
Fall term 2023
Master of Healthcare,
Clinical Optometry
Oulu University of Applied Sciences

ABSTRACT

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Master of Healthcare, Clinical Optometry

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Title of the thesis: Clinical Guideline for Finnish Optometrists – Assessment of Posterior Vitreous Detachment

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Term and year of thesis completion: Fall term 2023

Pages: 82

Purpose: The purpose of this research development was to produce evidence-based material about posterior vitreous detachment and its complications. The thesis aimed to create a clinical guideline for Finnish optometrists on assessing posterior vitreous detachment. The research development was commissioned by the Finnish Ethical Board of Optometry (OEN).

Methods: This research development was conducted as a literature review analysis-based work for the Finnish Ethical Board of Optometry between spring 2022 and fall 2023. The research development consisted of three phases. The first phase included a comprehensive theoretical background on the vitreous, posterior vitreous detachment, and complications of posterior vitreous detachment. The second phase included a narrative literature review of complications of posterior vitreous detachment and their prognosis. The third phase consisted of searching and selecting existing international guidelines and defining the content and structure for the Finnish posterior vitreous detachment assessment guideline. The content of the Finnish guideline was based on the selected international guidelines and the results of the narrative literature review.

Results: As a first result of this research development work, nine studies were chosen for the narrative literature review. As a second result, four international guidelines about posterior vitreous detachment-related disorders were identified for further evaluation. As a third result, the posterior vitreous detachment assessment guideline for Finnish optometrists was created. The guideline includes three parts: patient history/anamnesis, ocular examination, and patient education. Anterior vitreous and peripheral retina should have particular emphasis in ocular examination during the acute posterior vitreous detachment. Early-stage posterior vitreous detachment may cause complications in the macula area. Patient education is an essential part of the care process. Retinal breaks may be missed in the initial examination or have not been developed yet. Therefore, all patients should be educated about the risks and signs of retinal detachment.

Conclusions: Finnish optometrists have all the required elements to perform a thorough adult eye examination that is needed in the evaluation of posterior vitreous detachment. In the future, there will be a growing demand for adequately harnessing the clinical skills of Finnish optometrists. By using this guideline, Finnish optometrists will have a uniform way of assessing posterior vitreous detachment.

Keywords: clinical guideline, macular hole, optometrist, posterior vitreous detachment, retinal breaks, retinal detachment, vitreomacular traction

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1 INTRODUCTION

Flashes and floaters are frequently encountered visual symptoms in optometric practice. These symptoms are classical signs of acute posterior vitreous detachment (PVD). Examiners cannot separate harmless PVD from anomalous PVD, which can cause retinal tears and lead to retinal detachment just by the symptoms. Therefore, a comprehensive adult eye examination is necessary. PVD may also cause vitreomacular traction (VMT), macular hole (MH), and epiretinal membrane (ERM), with a variety of visual symptoms, such as metamorphopsia and reduced visual acuity. Although PVD is often an innocuous age-related phenomenon, it can lead to severe consequences unless managed and treated correctly.

In PVD, the posterior vitreous cortex separates from the retina's internal limiting membrane (ILM). Aging of the vitreous goes through substantial liquefaction and concurrent aggregation of collagen fibrils. These physical changes may end up in harmless PVD or cause structural damage to sites where the adherence between the retina and the vitreous is strongest, especially at the macula and peripheral retina. PVD is relatively rare in patients under 50 years old, but the prevalence increases rapidly after 60 years. With patients over 80 years old, almost nine out of ten patients have undergone the separation of the posterior vitreous cortex from the neurosensory retina. (Bond-Taylor et al., 2017.)

Symptomatic acute PVD causes retinal tears in approximately 15 % of the cases (Bond-Taylor et al., 2017). If left untreated, up to 50 % of symptomatic retinal tears will develop into rhegmatogenous retinal detachment (RRD) (Steel, 2014). Patients with high myopia, previous ocular surgery, or trauma are particularly at risk of developing tears during acute PVD. According to Mityr et al. (2010), the incidence of RRD is highest during the seventh decade, attributable to the onset of PVD. The causative reason for retinal detachment is most often a PVD-related horseshoe tear. (Mityr et al. 2010.) Early-stage PVD may harm the macula area by causing VMT and MH. Severity ranges from mild reduction of visual acuity to cases with significant visual deficiency. If VMT progresses to MH, rapid intervention may be necessary. (Flaxel et al., 2020b.)

At the time of this Thesis, many working sectors in Finland struggle with labour resources. The social and healthcare sector is no exception, and the future does not seem to bring any change. In addition, large population groups are getting older, increasing the prevalence of age-related eye illnesses. In 2019, more than 1.2 million Finns were 65 years or older. The aging population has posed a significant challenge to the Finnish social and healthcare system. (Sosiaali- ja terveystieteiden ministeriö, 2022.) The decreasing number of ophthalmologists, especially in public healthcare, and the reformation of Finland's health and social services will inevitably impact providing adequate services regardless of the patient's location. So far, customers with signs of PVD have often been referred to ophthalmologists in Finnish optician stores. Depending on the

local resources, the appointment queue may vary from days to months. To decrease the workload of ophthalmologists in public healthcare, the resources of optometrists have been utilized in selected tasks. (Hautala, 2020) Similar work distribution is a topical subject also in the private sector.

This research development was ordered by the Finnish Ethical Board of Optometry (OEN). OEN wants to create uniform eye examination procedures across the Finnish optometric field, and producing clinical guidelines on particular topics is a starting point for this endeavour. OEN has the right to modify and apply the results of this research development.

The purpose of this research development was to produce evidence-based material about posterior vitreous detachment and its complications. The thesis aimed to create a clinical guideline for Finnish optometrists on assessing posterior vitreous detachment.

2 THEORETICAL BACKGROUND

The theoretical background of this research development work consists of four parts: the vitreous, posterior vitreous detachment, complications of acute posterior vitreous detachment, and complications of early-stage posterior vitreous detachment. Initial literature search for the theoretical background was done during the autumn of 2022 from Pubmed, Google Scholar, and Medic. A more thorough literature search and writing of theoretical background took place between January 2023 and March 2023. PubMed was the primary database for the theoretical background. A wide range of search terms was used, for example, “posterior vitreous detachment” OR “PVD”, “rhegmatogenous retinal detachment” OR “RRD”, “macular hole” OR “idiopathic macular hole”, “epiretinal membrane” OR “idiopathic epiretinal membrane”, “vitreomacular traction” OR “VMT”, “peripheral retinal degeneration” AND “posterior vitreous detachment”. In addition, several ophthalmologic books were also used during the process.

2.1 The Vitreous

The vitreous is a gel-like and transparent extracellular matrix between the crystalline lens and retina. In an emmetropic adult eye, the vitreous is 16.5 mm long and approximately 4 ml in volume. It is the largest eye structure and constitutes 80 % of the ocular volume. Optically, the vitreous is a clear, colourless media, and its refractive index is 1.33. (Forrester et al., 2016, p.37; Yanoff & Duker 2018, p.432.)

Anteriorly vitreous is bound to the crystalline lens, ciliary body, and posterior part of the lens zonules. Vitreous is attached to the posterior lens surface by the hyaloid capsular ligament of Weiger in an annular 1-2 mm width region. Weiger's ligament is 8-9 mm in diameter, and in the centre of the ligament is Berger's space. From Berger's space arises a canal of Cloquet that extends through the central vitreous into the funnel-shaped region of Martegiani close to the optic disc. Cloquet's canal is the former site of hyaloid artery that, during embryonic development, nourishes the lens and primary vitreous. (Forrester et al. 2016, 37; Sebag 1989, 35.)

Remnants of the hyaloid artery are sometimes seen in a clinical examination. These remnants may be seen in isolation or in combination with other remnants of persistent vasculature. If the eye is fully developed, there is none or only a little effect on vision. **Mittendorf's dot** appears as a focal opacity on the inferonasal posterior lens capsule and represents a termination point of the hyaloid artery. **Bergmeister's papilla** is seen as a “tuft” at the optic disc. It represents a remnant of the fibrous sheet that covers the fetal hyaloid artery. Initially, the hyaloid artery loses its perfusion around the seventh month of gestation and then

regresses. If regression is incomplete, a part of the **persistent hyaloid artery** may be seen attached to the posterior lens capsule or optic nerve. (Goldberg, 1997, p. 14-15.)

Traditionally, the vitreous is divided into a cortical zone, where collagen fibrils are more densely organized and a more liquid central vitreous. Anatomically, the vitreous can be subdivided into three major zones: preretinal, intermediate, and retrolental. The vitreous cortex has the most robust adhesion to the retina in the periphery, where the vitreous base straddles ora serrata and pars plana via a 3-4 mm wide band. The cortical vitreous also has relatively strong adhesion to optic disc margins, and it is loosely attached to the macula area and over the retinal blood vessels. Vitreous may exhibit abnormally strong adhesion to vitreoretinal disorders, mainly at the peripheral retina, which can give rise to retinal tear formation following posterior vitreous detachment (PVD). (Kanski & Bowling, 2015, p.682-683; Forrester et al., 2016, p.36-37; Yanoff & Duker, 2018, p.435.)

The vitreous cortex is a thin layer of tightly packed collagen fibrils. Internally, it surrounds the more liquid central vitreous, and posteriorly it adheres to the internal limiting membrane of the retina (ILM), specifically to the end plates of retinal Muller cells. Vitreomacular attachment occurs in an approximately 4 mm diameter region around the fovea, but the optic disc has no attachment with the vitreous cortex. In PVD, peripapillary glial tissue might be torn away around the papilla. If it stays attached to vitreous cortex, ring-like pattern (Weiss ring) may be seen in clinical examination. (Sebag, 1989, p.42.)

Despite the extensive knowledge of vitreous structures and composition, the exact physiological function of the vitreous is not entirely understood. One of the main functions of vitreous is to provide a transparent media to enable maximal visual acuity. Vitreous also has a hassle in regulating the shape and growth of the eye development. Haltfer et al. (2006) propose that the vitreous cortex with ILM provides mechanical strength against pressure when the sclera is still developing. An improper extracellular matrix predisposes the eye to expand too large. Furthermore, the vitreous offers structural support to the retina during eye movements and physical activity and acts as a shock absorber—similar to synovial fluid between joints. The vitreous is also essential in being a metabolic repository (glucose, antioxidants, oxygen) for the lens and retina. (Forrester et al., 2016, p.241-242; Haltfer et al., 2006, p. 3593; Sebag, 1989, p. 60-64.)

2.1.1 Composition of the Vitreous

The vitreous is a connective tissue mainly composed of water (98-99%), hyaluronan, collagen, and cells. Collagen fibrils from 10 to 25 nm are thinner than half of the wavelength of the light and similarly transmit light as the cornea. The space between the individual collagen fibrils is filled with hyaluronan

(glycosaminoglycan) that interacts with collagen to provide minimal diffraction along the light pathway to the retina. Collagen in the vitreous gel is predominantly type II and IX, but small amounts of type V, VI, and XI collagens are also present. Type II collagen is fibril-forming collagen accounting for up to two-thirds of vitreous collagen capacity. Type IX collagen accounts for approximately 20 % of collagen concentration, and it is the second largest collagen type in the vitreous. Type IX is non-fibrillar collagen containing multiple non-collagenous domains that act as proteoglycan bridges. (Forrester et al., 2016, p.240; le Goff & Bishop, 2008; Bishop *et al.*, 1994.) In Stickler syndrome, there is a congenital abnormality in vitreous development due to mutation in genes that encode fibrillar type collagens, most commonly type II and XI. Stickler syndrome is a common cause of inherited rhegmatogenous retinal detachment (RRD). (Snead *et al.*, 2011, p. 1389.)

Hyaluronan, formerly hyaluronic acid, is the vitreous centre's primary glycosaminoglycan (GAG). Hyaluronan occurs in a stiff disaccharide chain with a 100 to 400 µg/ml concentration. If the concentration exceeds 300 µg/ml, hyaluronan becomes entangled, supporting the gel-like structure of the vitreous. The highest hyaluronan concentration is found in the cortex. (Forrester et al., 2016, p.240.)

The vitreous is mainly acellular, containing just a few hyalocytes that line the peripheral cortex, optic disc, and retinal blood vessels. Hyalocytes are bone marrow-derived mononuclear phagocytes. Their functional importance is not entirely understood. It is suggested that the highest concentration of hyalocytes in the area with the highest hyaluronan concentration might mean that hyalocytes are responsible for hyaluronan synthesis. Sakamoto et al. (2011) demonstrated that hyalocytes might have an essential role in the pathophysiology of proliferative vitreoretinopathy, including macular pucker formation. (Sakamoto & Ishibashi, 2011; Yanoff & Duker, 2018, p.434.)

2.1.2 Aging of the Vitreous

The vitreous goes through substantial alterations almost during the entire adulthood. The thick vitreous gel starts to degenerate to a thinner liquid-like substance. Liquefaction (**synchysis**) starts much earlier than visible changes in the clinical examination are detectable. Already at the age of 18 years, approximately 20 percent of vitreous is liquefied. After 45 years, the vitreous undergoes a steady decrease in gel volume and a concomitant increase in liquefaction. At 80-90 years, more than 60 % of vitreous is liquefied. (le Goff & Bishop, 2008.)

Liquefaction and concurrent aggregation of collagen fibrils (**syneresis**) into collagen clusters occur due to the breakdown of regular hyaluronan-collagen interaction. The process is not uniform within the entire vitreous. This might lead to the formation of pockets or lacunae of liquid, especially in the posterior vitreous. Although the mechanism of liquefaction is poorly understood, it is hypothesized that the vitreous liquefaction

might be exacerbated by daily UV-light exposure. UV light predisposes to free radical formation, which eventually alters the hyaluronan-collagen complex. (Yanoff & Duker, 2018, p. 435.) In diabetic patients, collagen might exhibit abnormal cross-linking between individual fibrils due to the nonenzymatic glycation of collagen. This may lead to advanced liquefaction and posterior vitreous detachment (PVD) earlier in life. (Sebag, 1996.)

The exogenous administration of enzymatic agents can also induce liquefaction. One of these agents is ocriplasmin. Ocriplasmin is a proteolytic enzyme that enhances liquefaction and separates vitreoretinal adhesions. In addition, it breaks down the bonds of laminin and fibronectin, which maintain adhesion between the retina and the vitreous. Ocriplasmin is used as an intravitreal injection in small macular holes (<400 µm) and vitreomacular traction cases. (Neffendorf *et al.*, 2017.)

Wang *et al.* (2003) demonstrated in their study that the posterior border of the vitreous base migrates toward the equator during life — more on the nasal than the temporal side. (Wang *et al.*, 2003) Posterior migration, along with the changes in collagen fibril synthesis and how collagen fibrils penetrate the ILM, may increase the risk of vitreoretinal traction in the peripheral retina. Thus, contributing to retinal tear formation and, eventually, rhegmatogenous retinal detachment. (Yanoff & Duker, 2018, p. 436.)

2.2 Posterior Vitreous Detachment

Posterior vitreous detachment is one of the most common age-related phenomena of the eye. In clinical work, PVD is the most frequently encountered event related to the aging vitreous. PVD is defined as a “separation between the posterior vitreous cortex and the ILM of the retina.” It can be partial, localized, or complete, depending on the extension of the residual vitreoretinal adhesion. PVD is complete when the vitreous is detached up to the posterior border of the vitreous base. (Yanoff & Duker, 2018, p. 436.)

PVD results from several changes in the gel-like vitreous body and at the interface of the retina and vitreous. Aging causes vitreous to become liquefied, collagen fibrils aggregate into bundles, and vitreoretinal adhesion weakens. Breaks in the adhesion between the posterior vitreous cortex and ILM let the liquid vitreous enter the retrocortical space. This volume movement to preretinal space causes the vitreous to collapse. True PVD is likely to be contributed by rotational eye movements, which allows the liquid vitreous to dissect the vitreous cortex from the retinal ILM. (Yanoff & Duker, 2018, p. 436.)

According to autopsy studies, PVD will be present in 51 % of eyes by the seventh decade, and prevalence will increase to 63 % by the eighth decade. Only fewer than 10 % of people younger than 50 years have PVD. PVD is very rare before the fourth decade in emmetropic eyes. (Foos & Wheeler, 1982.) Similar

findings have been reported in clinical studies. The prevalence of PVD is approximately 24% at the age of 50–59; among 80–89 years, the prevalence can be as high as 87%. (Rahman *et al.*, 2002; Hollands *et al.*, 2009.) Typically, the mean onset age of PVD is between 60 and 65 years (Yonemoto *et al.*, 1994; Rahman *et al.*, 2002). The prevalence of PVD increases significantly when more than 60 % of the vitreous has liquefied (Foos & Wheeler, 1982).

2.2.1 Classification of Posterior Vitreous Detachment

According to Johnson (2010), age-related PVD starts insidiously and progresses into complete PVD asymptotically and slowly during the following months or even years. Johnson (2010) has proposed that PVD occurs in five stages (Figure 1):

- Stage 0: Vitreous cortex is completely attached to the retina.
- Stage 1: Vitreous cortex is detached from the perifoveal area. Firm adhesion of 500- μ m diameter still occurs around the fovea.
- Stage 2: Complete separation between the vitreous cortex and macula.
- Stage 3: Vitreous cortex is detached from the entire retina, except the papillary zone.
- Stage 4: Separation of vitreopapillary adhesion, total PVD.

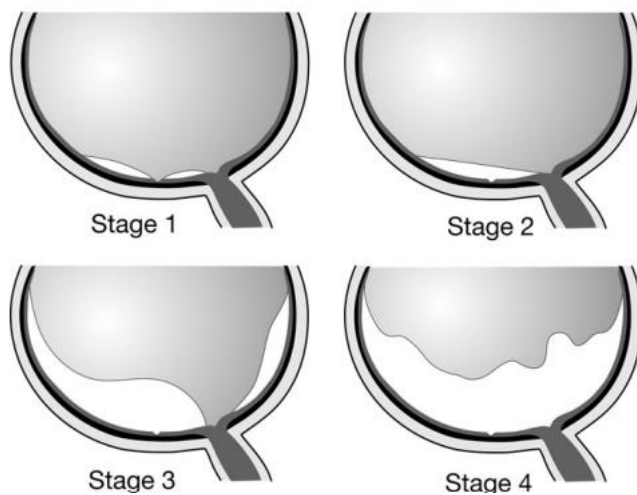


Figure 1. Stages of posterior vitreous detachment. (Johnson 2005)

Early stages of PVD are often asymptomatic and develop smoothly in most people. However, residual solid vitreous adhesion along the posterior pole might lead to anteroposterior traction and a large spectrum of retinal pathologies. Therefore, symptoms often occur during the last stages of age-related PVD when various pathological changes might occur in the peripheral retina. (Johnson, 2010.)

Takehashi et al. (2013) have made an illustrative classification of PVD (Figure 2). According to Takehashi et al. (2013), PVD can be classified as complete PVD (C-PVD) or partial PVD (P-PVD). C-PVD is subdivided depending on the presence of collapse or without collapse. P-PVD is classified depending on the shrinkage of the posterior hyaloid membrane. C-PVD, without collapse, has a shallow detached hyaloid membrane that can be visualized just before the retina. This kind of PVD is a typical finding in eyes with diabetic retinopathy or in young patients with central retinal vein occlusion (CRVO) or uveitis. In contrast to C-PVD without collapse, C-PVD with collapse is a typical finding in otherwise healthy eyes. P-PVD without shrinkage is thought to be most often an interphase that will evolve to C-PVD with collapse. P-PVD with shrinkage is a typical finding in proliferative diabetic retinopathy, where the hyaloid membrane and neovascular tissue might cause significant traction at the posterior pole. (Takehashi, Takezawa & Akiba, 2013.)

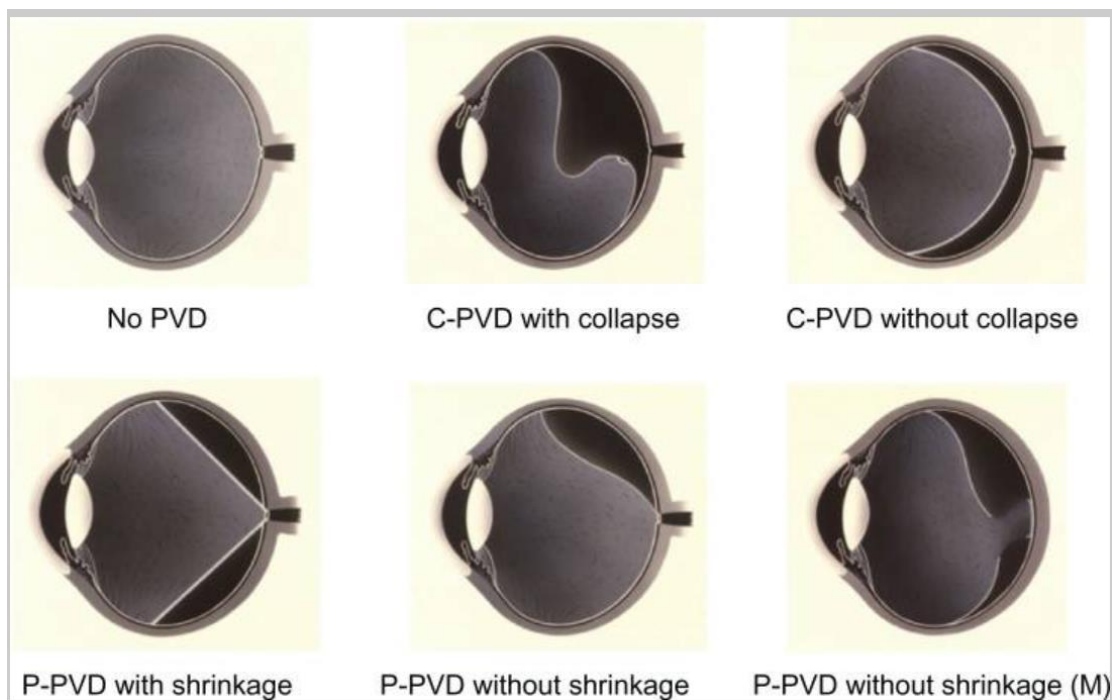


Figure 2. Classification of PVD (Takehashi, Takezawa & Akiba, 2013)

2.2.2 Risk Factors

The prevalence of PVD increases with **age**, but several other risk factors can impact the earlier onset of PVD. To optometrists, the most familiar risk factor is **myopia**. In myopic eyes, vitreous volume is much higher compared to emmetropic eyes. One hypothesis for early myopic PVD is that the vitreous gel formation occurs during the embryonic phase, and the elongation occurs during postnatal development. This leads to increased liquid content and low-protein content in the vitreous. Therefore, myopic vitreous resembles premature synchysis. (Yanoff & Duker, 2018, p. 118.) According to Yonemoto et al. (1994), myopia can speed up the onset of PVD by 0.91 years per every diopter of myopic refractive error (Yonemoto *et al.*, 1994).

Thus, in highly myopic eyes (>-6 to -10 dpt), PVD can occur up to ten years earlier compared to emmetropic eyes, and nearly everybody more than 70 years old has had PVD (Akiba, 1993).

Along with myopia, several other risk factors have been identified. In **collagen metabolism disorders**, such as Stickler, Marfan, and Ehlers Danlos the vitreous undergoes premature vitreous liquefaction and syneresis, leading to earlier onset of the PVD. Additionally, liquefaction and vitreoretinal dehiscence in collagen disorders do not occur at a similar rate. Therefore, this group of disorders is at higher risk for non-innocuous PVD. Maybe the most interesting risk factor for PVD is the **female gender**. The hypothesis behind the female gender is related to postmenopausal loss of estrogen, which leads to a decrease in hyaluronan synthesis and increased liquefaction. (Tozer, Johnson & Sebac, 2014, p. 141.) According to Chuo et al. (2006), menopause and high dietary intake of vitamin B6 are significant risk factors for PVD. The hormonal processes behind these factors are not well understood. (Chuo *et al.*, 2006.)

Additionally, various **eye surgeries** are risk factors for PVD. PVD can occur after panretinal laser photocoagulation, retinal cryopexy, or even after refractive laser surgery, but most commonly after cataract surgery. (Ahmed & Tripathy, 2022.) Cataract surgery interferes with the normal physiological aging process by altering the vitreous humour composition and structure (Neal *et al.*, 2005). Aggressive surgical techniques and intraoperative or postoperative posterior capsule breaks induce vitreous destabilization. Destabilized vitreous can lead to anomalous PVD, tear formation, and subsequent RRD. (Coppé & Lapucci, 2008.) It has been reported that in emmetropic eyes, PVD occurs in approximately 78% of the eyes during the first two and a half years after the cataract surgery. In the eyes where lattice degeneration is present, the incidence might be even higher (87%). (Ripandelli *et al.*, 2007.)

Other factors that may accelerate vitreous liquefaction and advance the onset of PVD include retinal vascular diseases, trauma, aphakia, inflammation, and vitreous hemorrhage (Johnson, 2010). Traumatic PVD has been studied in monkey eyes. **Trauma** may alter the structure of the vitreous and retina, thus causing PVD and leading to retinal detachment. In the research of Hsu et al. (1986), PVD was a predisposing factor to retinal detachment, but detachment was not rhegmatogenous. Instead, PVD caused breaks in the ILM, and the subsequent epiretinal membrane was the causative factor for tractional retinal detachment. (Hsu & Ryan, 1986.) Blunt trauma or penetrating ocular injury also increases the risk of having RRD. Vitreoretinal changes may occur shortly after the accident or take years to appear. (Flaxel et al., 2020c.)

While many factors may advance the onset of PVD, in **diabetes**, complete PVD occurs later compared to normal eyes. In the eyes of a diabetic patient, vitreous collagen exhibits abnormal crosslinking and nonenzymatic glycation, leading to premature liquefaction of the central vitreous. However, abnormally strong adhesion between the vitreous and retina hinders the PVD process. This often leads to incomplete and anomalous PVD, especially in proliferative diabetic retinopathy. Diabetic retinopathy is usually worse in the

area of firm vitreoretinal adhesion, and complications are exacerbated, such as diabetic macular edema. (Gale, Aiello & Sebag, 2014, p. 57, 65.) In contrast, complete posterior vitreous detachment may hinder the progression of diabetic retinopathy and reduce the need for treatment (Anderson *et al.*, 2019).

2.2.3 Symptoms of Posterior Vitreous Detachment

For optometrists and other clinicians, it is essential to understand where symptoms start to occur in the process of PVD and what they can be in each stage. The most common findings related to PVD are **floaters** (myodesopsia) and **flashes** (photopsia). Floaters appear when the light scatters from dense bundles of collagen fibers and casts a shadow onto the retina. Floaters are often described as spots, cobwebs, or strands by patients. Floaters are common complaints of older patients, even without PVD, but they tend to increase in size and amount when the complete PVD occurs. PVD is the most common cause of floaters, but floaters can also be symptoms of Asteroid hyalosis or vitreous debris from various reasons, such as infection or inflammation. Floaters may also appear from vitreous haemorrhage due to a retinal tear or break in the retinal blood vessel. A presence of the **Weiss ring** may be seen as ring-shaped floater or a larger solitary lesion in the visual field. A Weiss ring is frequently kept as a sign of complete PVD. Dense floaters may reduce contrast sensitivity and visual acuity but usually do not cause significant harm and tend to become less noticeable over time. However, vitrectomy or YAG laser operation is sometimes performed on very symptomatic patients. (Huang *et al.*, 2014, p. 772-780; Kanski & Bowling, 2015, p. 694-695.)

Photopsia is a perception of flashing lights in the visual field without any external stimulus. Flashes in PVD are thought to occur when the vitreous exerts traction to the optic disc or another possible site of vitreoretinal adhesion. They are more often visualized on the temporal side and might be exacerbated by eye or head movements. It has been reported that PVD with symptoms of flashing lights has a 5.3% association with retinal tears. If flashes are seen in tandem with floaters, association with retinal tears has been reported to be as high as 20 %. (Gishti *et al.*, 2019; Kanski & Bowling, 2015, p.694-695.) Although monocular flashing lights may be seen in several ocular conditions, they are most often related to PVD, retinal breaks, and retinal detachment. Bilateral photopsia is commonly associated with visual auras of migraine headaches but can occasionally reflect other underlying neurologic conditions, such as occipital stroke or transient ischemic attack (Sharma *et al.*, 2015). In contrast to PVD, where photopsia are frequently rapid, short-lived, and monocular, in migraine, photopsia or other visual disturbances, such as distortions and scintillating scotomas, last longer (up to one hour) and may be bilateral (Kanski & Bowling, 2015, p. 694-695, 846).

A third common complaint related to PVD is blurred or hazy vision, with variably **reduced visual acuity**. Haziness may be due to posterior hyaloid membrane or dense floaters, but visual acuity in these cases is generally not much affected. Significant haze with concurrently reduced VA is often caused by a retinal tear

or torn retinal blood vessel, leading to dispersed haemorrhage within the vitreous gel. (Kanski & Bowling, 2015, p.694-695.) Visual acuity may also be affected in the early or late stages of PVD by vitreomacular traction (VMT), epiretinal membrane (ERM), or macular hole (MH). Medium or large full-thickness macular hole (FTHM) reduces VA significantly, and without treatment, the vision will often end up in the 20/200 to 20/400 range. (Flaxel et al., 2020b.)

In the early stages of PVD, abnormally strong adhesion between the vitreous and macula may alter the contour of the fovea and lead to significantly troublesome distortions, **metamorphopsia**. Metamorphopsia may be accompanied by variably reduced visual acuity. (Johnson, 2010.) Epiretinal membranes are common findings in the later stages of PVD. ERM may cause metamorphopsia and reduce VA but is also associated with **aniseikonia** and **diplopia**. The remarkable difference between eyes in the perceived size of images characterizes Aniseikonia. Aniseikonia is thought to develop when retinal photoreceptor cells are abnormally distributed. Furthermore, ERM causes image distortion that can prevent fusion. This may lead to suppression or double vision, which may be challenging to treat with conventional prism correction. (Okamoto *et al.*, 2014; Chung *et al.*, 2015; Benegas, 1999.)

2.2.4 Anomalous Posterior Vitreous Detachment

Age-related PVD is most often an innocuous event that does not cause significant visual hindrance. In innocuous PVD, vitreous liquefaction occurs concurrently with the weakening of the vitreoretinal interface, resulting in a harmless and clean separation of the vitreous from the retina. In contrast to innocuous PVD, anomalous posterior vitreous detachment (APVD) occurs when the vitreous gel liquefaction and dehiscence at the vitreoretinal interface do not happen concurrently. (Tozer, Johnson & Sebac, 2014, p. 142.)

In APVD, a disconnection between liquefaction and gel dehiscence leads to tractional forces upon the retina. APVD may lead to various retinal disorders depending on the site where the vitreous is most adherent to the retina. In the peripheral retina, APVD causes tears, which may lead to subsequent RRD. In addition, APVD causes vitreomacular traction syndrome at the macula area, promoting vitreoschisis, macular pucker, and macular hole formation. APVD is also associated with diabetic macular edema and proliferative diabetic vitreoretinopathy. Intense and persistent adhesion to retinal blood vessels may lead to retinal haemorrhage. In addition, vitreopapillary traction may contribute to neovascularization of the optic disc. APVD can be facilitated by many underlying conditions, such as Marfan or myopia, but may also occur without any risk factors. (Tozer, Johnson & Sebac, 2014, p. 142; Sebag, 2004.)

According to Johnson (2010), complications of age-related PVD can be divided into two main categories depending on their time course. Complications are listed in Table 1.

Table 1. Complications of age-related PVD. (Adapted from Johnson, 2010)

Early-stage PVD	Late-stage PVD (complete)
Epiretinal membrane Macular microhole Foveal red spot Idiopathic macular hole <ul style="list-style-type: none"> • Pseudo-operculum • Inner lamellar macular hole Vitreofoveolar traction (cystoid macular edema) Vitreomacular traction syndrome Traction diabetic macular edema Myopic traction maculopathy Neovascular age-related macular degeneration Vitreopapillary traction syndrome	Retinal or optic disc hemorrhage Vitreous hemorrhage Retinal tear Rhegmatogenous retinal detachment

Complications in the posterior pole are highly related to the anatomical site and size of the persistent adhesion (Table 2). Johnson (2010) promoted the concept of smaller ($\leq 500 \mu\text{m}$) and larger ($\geq 1500 \mu\text{m}$) sizes of adhesion, which were revised by The International Vitreomacular Traction Study (IVTS) Group to be focal ($\leq 1500 \mu\text{m}$) and broad ($> 1500 \mu\text{m}$). Traction is strong and localized in smaller adhesion sizes, causing significant stress over the underlying area. Thus, total thickness macular hole (FTMH), lamellar macular hole (LMH), and vitreo-foveolar traction syndrome are common findings with the adhesion of smaller diameters. In addition, the smallest zones of adhesion (50-150 μm) are likely responsible for macular microhole and foveal red spot syndrome. When the zone of adhesion exceeds 1500 μm , tractional forces are more dispersed over the larger area, and full thickness defects are less likely to occur. Conversely, larger zones of adhesion are more likely to contribute to vitreomacular traction syndrome, diabetic macular edema, myopic traction maculopathy, and neovascular age-related macular degeneration. (Johnson, 2010.)

Table 2. Macular complications of early-stage PVD. (Adapted from Johnson, 2010)

Macular complications of early-stage PVD
<p>Variable vitreomacular adhesion</p> <ul style="list-style-type: none"> • Epiretinal membrane <p>Adhesion size $\leq 500 \mu\text{m}$</p> <ul style="list-style-type: none"> • Macular microhole • Foveal red spot • Idiopathic macular hole <ul style="list-style-type: none"> - Pseudo operculum - Lamellar macular hole • Vitreofoveolar traction (cystoid macular edema) <p>Adhesion size $\pm 1500 \mu\text{m}$</p> <ul style="list-style-type: none"> • Vitreomacular traction syndrome • Traction diabetic macular edema • Myopic traction maculopathy • Neovascular age-related macular degeneration

After the posterior vitreous cortex has fully separated from the macula area, it may cause vitreoretinal traction in the peripheral retina. This is the moment when vitreous traction can result in tear formation, which, in the worst cases, leads to RRD when vitreous fluid accumulates under the retina. (Yanoff & Duker, 2018, p. 436.) It has been reported that symptomatic PVD (flashes or floaters) develop retinal breaks in 6-18 % of the cases, and retinal breaks progress into RRD in 35-47% of the cases. Although asymptomatic PVD is less likely to develop breaks and progress into RRD, subclinical RRDs are found in up to 13.8 % (0-13.8%) of the cases. (Blindbaek & Grauslund, 2015.)

2.2.5 Management of Posterior Vitreous Detachment

According to current knowledge, there is no effective way to prevent PVD. It is part of the normal aging process of the eye. The main goal of managing PVD is to detect complications that may lead to vision loss and functional impairment.

Symptomatic PVD (e.g., floaters, photopsia) develop retinal breaks in up to 20 % of the cases (Gishti et al., 2019). Thus, symptomatic patients should be examined within the first days to the first weeks, depending on the risk factors and severity of the symptoms. Evidence suggests that patients with single small floater without flashes are not at high risk of developing retinal tears, and urgent assessment is unnecessary. In

contrast, patients with risk factors, such as high myopia (>6 dpt), previous family history of retinal detachment, pseudophakia, and collagen disorders (e.g., Stickler or Marfan) increase the urgency to have examination within the first few days. Furthermore, substantial acute symptoms of very prominent floaters significantly reduced visual acuity, and visual field defect increase the risk of retinal tears and detachment, and examination should be done as soon as possible (24-28 h). (Kanski & Bowling, 2015, p. 695-697; Flaxel et al., 2020c.)

In the case of symptomatic PVD, a comprehensive adult eye examination is recommended. The examination should include the best corrected visual acuity, confrontation visual field testing, pupillary assessment for possible relative afferent pupillary defect (RAPD) and slit-lamp biomicroscopy. In slit-lamp biomicroscopy, clinical signs of PVD should be assessed to rule out other possible causes of symptoms, such as infection, inflammation, and neoplasia. The presence of the Weiss ring is often kept as a sign of complete PVD (Figure 3). It is important to remember that the Weiss ring may also be destroyed in the PVD process. When the vitreous collapses, the detached posterior hyaloid membrane may be seen as a crumpled translucent membrane in the vitreous cavity. (Kanski & Bowling, 2015, p. 695-697; Flaxel et al., 2020c.) Shallow complete PVD is often difficult to observe by slit-lamp examination. In the case of shallow PVD, OCT is a valuable tool to confirm the PVD. OCT also shows the first stages of PVD with significant accuracy. (Kakehashi et al., 2013.)

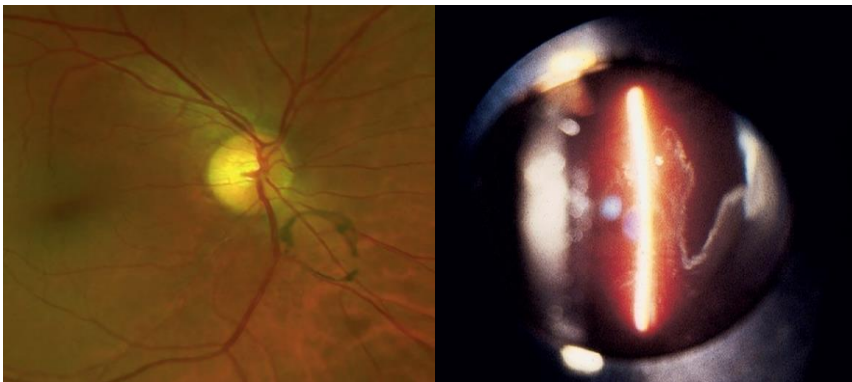


Figure 3. (Left) Weiss ring, the optic disc in the background; (Right) Detached and collapsed vitreous, opaque membrane, and behind the membrane is a dark area. Reprinted with permission (Kanski & Bowling, 2015, p. 697)

The anterior vitreous should be carefully examined by slit lamp for the presence of blood and/or pigment. Pigment granules (**Shafer sign or Tobacco dust**) are more prominent, darker, and less reflective than red blood cells (Figure 4). Pigment granules arise from retinal pigment epithelium (RPE) and end up in the vitreous through the retinal break. The presence of pigment cells in the anterior vitreous is often kept pathognomonic for a retinal break, as the sensitivity is as high as 95%. Red blood cells arise either from retinal break or torn retinal blood vessel. Red blood cells may appear in the anterior vitreous or as small intragel

clusters or preretinally. Sometimes, blood may be trapped between the retina and posterior hyaloid membrane, forming a crescent-shaped configuration. (Kanski & Bowling, 2015, p. 695.) It has been reported that approximately two-thirds of patients with vitreous hemorrhage have at least one retinal break at the initial examination. Only a few percent develop breaks after the first assessment. About 80 % of these cases have either red blood cells or pigment in the vitreous in the initial examination or develop new symptoms before the follow-up evaluation. Most breaks tend to be on the superior side of the retina. (Coffee et al., 2007; Sarrafizadeh, 2001.)

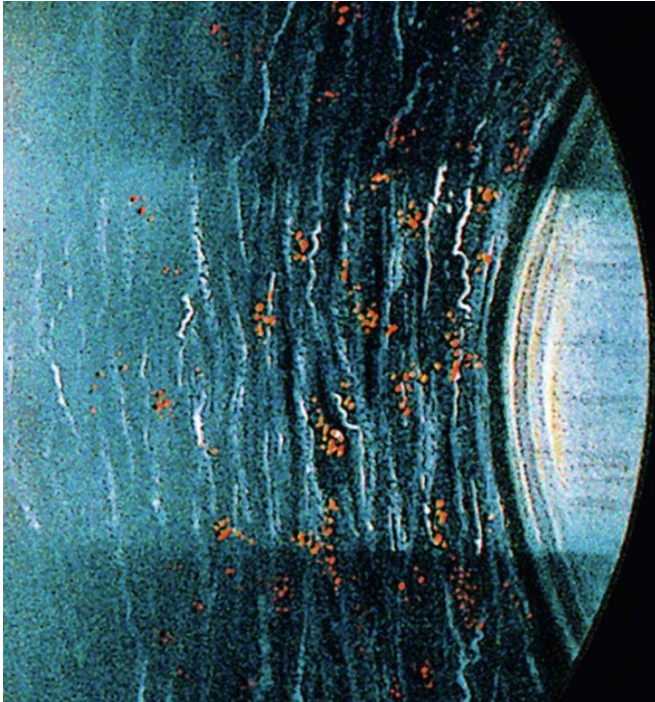


Figure 4. "Tobacco dust" in the anterior vitreous. Reprinted with permission. (Kanski & Bowling, 2015, p. 702)

Most breaks appear at the peripheral retina (from the equator to ora serrata), so a careful 360-degree examination of the peripheral retina should be performed. An indirect ophthalmoscope combined with scleral indentation is the gold standard examination method for the peripheral retina. Alternative method for fully dilated eyes includes slit-lamp biomicroscopy with mirrored contact or condensing lens. Sometimes wide-field photography is performed to detect retinal breaks, but according to current consensus, it is not superior to careful ophthalmoscopy or 3-mirror contact lens examination. In the case of dense haemorrhage that obscures visibility to the retina, B-scan ultrasonography should be utilized to detect retinal breaks and retinal detachment or to look for other possible causes of haemorrhage. (Kanski & Bowling, 2015, p. 695-697; Flaxel et al., 2020c.)

Subsequent follow-up after the initial examination should be defined according to the findings and pre-existing risk factors. The need for a follow-up is questionable if no retinal breaks, haemorrhages, or pre-existing

risk factors are identified. Current literature shows only little evidence for the follow-up demand in these cases. Although, it is good practice to have a follow-up in 4 weeks to ensure that new retinal breaks have not been developed. Patients with multiple prominent floaters and photopsia should be followed up every two weeks until the symptoms have resolved. In the case of mild vitreous haemorrhage or small punctate retinal haemorrhage without an identified retinal break, follow-ups should be performed within one week, two weeks, and four weeks. Some specialists recommend additional review in 6-12 months. In the presence of significant vitreous haemorrhage or pigment in the anterior vitreous without an identified retinal break, a retinal specialist should perform a re-examination the following day. (Bergstrom & Czyz, 2023; Kanski & Bowling, 2015, p. 695-697; Optometric Clinical Practise Guideline: Care of the Patient with Retinal Detachment And Related Peripheral Vitreoretinal Disease, 2004, p. 32.)

The acute symptomatic PVD management plan should include explicit instruction for all discharged patients. Patients should be educated about retinal detachment and possible symptoms when prompt re-evaluation is necessary. Instruction should consist of at least the following notes:

- increase in floaters
- increase in flashing lights
- reduced visual acuity
- curtain or shadow in the visual field
- sudden onset of tiny black specks

Floaters may affect the quality of a patient's life and cause concern initially. Therefore, it is wise to inform patients about the nature of floaters, as they tend to resolve and become less bothersome over time. (Kanski & Bowling, 2015, p. 695-697; Optometric Clinical Practise Guideline: Care of the Patient with Retinal Detachment And Related Peripheral Vitreoretinal Disease, 2004, p. 32.)

2.3 Complications of Acute Posterior Vitreous Detachment

2.3.1 Retinal Breaks

Retinal breaks, either **tears**, **holes** or **dialyses**, are full-thickness defects in the sensory retina (figure 5). The full-thickness defect is where retrohyaloid fluid can access the subretinal space and cause RRD. Retinal breaks can be found anywhere in the retina, but they mainly occur at the peripheral retina from the equator to the ora serrata. Most breaks appear at the superotemporal retinal quadrant (60%), followed by superonasal (15%) and inferotemporal (15%), and the least breaks occur at the inferonasal area (10%). The spread of subretinal fluid (SRF) is governed by gravitation; therefore, superotemporal breaks are the most

detrimental to vision. In these cases, SRF tends to proceed faster, and the macula area is affected earlier than with breaks of other locations. Breaks at the peripheral retina do not cause significant visual hindrance unless they lead to vitreous haemorrhage or RRD in the worst case. (Yanoff & Duker, 2018, p.642; Kanski & Bowling, 2015, p. 698-701.) According to autopsy studies, the prevalence of retinal breaks in some form is between 6% and 11% of people aged over 20 years. Asymptomatic retinal breaks have only 0.5 % association to RRD, if the other eye has not been affected before. Therefore, most breaks do not lead to RRD, and prophylactic treatment for every break may not be necessary. (Steel, 2014.)

Retinal tears usually occur during the onset of symptomatic PVD or soon after. Only a minority (<5%) of the breaks occur several weeks afterward. Most tears are located at the vitreous base, where vitreoretinal adhesion is strongest. Tears can be classified according to shape, size, and possible vitreous traction. U-tears, or horseshoe tears, occur secondary to vitreoretinal traction. The apex of the U-tear flap is pulled anteriorly by the vitreous, while the base of the flap remains attached to the retina. A giant tear is a variation of U-tear, extending at least three clock hours (90 degrees) of the retinal circumference. Dialysis is also a circumferential retinal defect along the ora serrata. In contrast to U-tear, in dialysis, the vitreous is attached to the anterior margin, and the flap is pulled posteriorly. Dialyses commonly occur after blunt trauma in the absence of PVD. (Yanoff & Duker, 2018, p.642; Kanski & Bowling, 2015, p. 698-701.)

Round or oval retinal holes are smaller than tears and may occur at the site of vitreoretinal traction or without traction. Retinal tear with a firm vitreoretinal adhesion may lead to avulsion of the tear base, resulting a round defect (hole) in the neurosensory retina. A small part of retinal tissue, an **operculum**, is often visible in the vitreous cavity, close to the retinal defect, when the vitreoretinal adhesion is completely torn off (Figure 5). Round atrophic holes occur due to the thinning of the retina, and vitreoretinal traction is not part of the mechanism. Atrophic holes are often present within lattice degeneration but may also occur in isolation. (Yanoff & Duker, 2018, p.642; Kanski & Bowling, 2015 p. 698-701.)

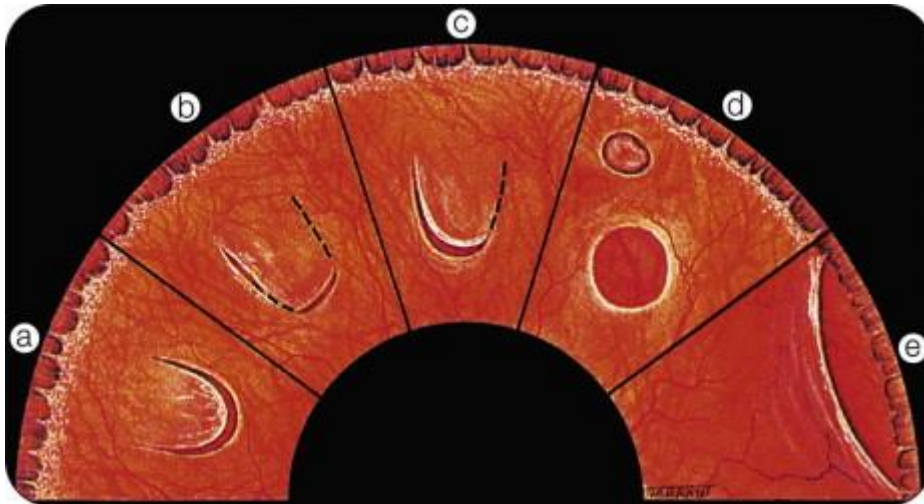


Figure 5. a, b, and c: variations of horseshoe tear, d: operculated tear, e: dialysis. Reprinted with permission. (Kanski & Bowling, 2005, p. 720)

In clinical examination, fresh retinal breaks have a reddish appearance, or sometimes whitish, in the case of high myopia. Subretinal fluid may be seen around the break, and an operculum can be visualized in the vitreous cavity. Chronic breaks are often encircled by pigmented demarcation line. The most significant risk factors for retinal breaks are like risk factors in RRD: high myopia, previous cataract surgery, age, some collagen-vascular disorders, and family history of RRD. (Kanski & Bowling, 2015 p. 698-701.)

According to Mitry et al. (2011), more than 85 % of RRD cases are associated with PVD and subsequent tractional tears. Retinal horseshoe tears (single or multiple) are responsible for up to 98.5 % of all RRD cases. (Mitry et al., 2011.) As mentioned previously, symptomatic PVD is thought to cause retinal breaks in 6-18 % of the cases (see Chapter 2.24). However, in a recent retrospective cohort study by Seider et al. (2022), the association between retinal breaks and RRD after PVD was much lower. During the symptomatic PVD, retinal breaks were diagnosed with 5.4 % and RRD in 4.0 % of the cases. High-risk variables for retinal breaks or RRD were vitreous pigment or haemorrhage, lattice degeneration, and visual acuity lower than 20/40. (Seider et al., 2022.) Asymptomatic breaks carry a very low risk of developing RRD (0.5 %) if the fellow eye has not a history of RRD (Steel, 2014).

2.3.2 Rhegmatogenous Retinal Detachment

In Rhegmatogenous Retinal Detachment (RRD), liquefied vitreous accumulates through retinal break into the subretinal space between the neurosensory retina and RPE and separates these layers. RRD is the most common type of retinal detachment; other types are exudative and tractional retinal detachment. The pathogenesis of RRD arises from vitreous syneresis and synchysis, which causes PVD and leads to retinal break formation. Thus, in most cases (80-90 %), PVD is the causative factor for RRD. RRD may occur either

after complete or partial PVD, but especially patients who have partial PVD and symptomatic retinal tear with persistent vitreoretinal traction are at high risk of developing RRD. If symptomatic RRD is left untreated, it will most likely progress to total retinal detachment and blindness. (Yanoff & Duker, 2018, p. 646-647.)

RRD is relatively uncommon in the general population. The incidence of RRD has been reported to vary a lot with populations, but the overall incidence is approximately 10 cases per 100,000 people each year. The most significant annual peak is among 60–70 years, and the secondary peak is within young myopes. Lattice degeneration and high myopia (>6 dpt) are the most critical risk factors, being part of RRD in up to 66 % of the cases. (Mity et al., 2010.) Other significant risk factors include hereditary collagen–vascular disorders, such as Stickler and Marfan, trauma, and previous cataract or lens extraction surgery. Cataract surgery is associated with RRD in 40 % of cases, although the actual risk of having RRD after cataract surgery is less than one percent. Young male patients with intraoperative posterior capsule rupture are especially at high risk of RRD after the cataract operation. Lens extraction is thought to increase the induction of early PVD and thus raise the possibility of RRD. Vitreoretinal changes are often bilateral; therefore, RRD in one eye significantly increases the risk of RRD in the other eye. (Yanoff & Duker, 2018, p.647-648.) According to Mity et al. (2012), approximately 7 % of the cases of RRD also develop in the fellow eye. Myopic patients with RRD in one eye should be followed carefully during the next years after the RRD. (Mity et al., 2012.)

More than half of the patients with spontaneous RRD experience flashes and floaters, similar to PVD. A curtain-like visual field defect usually follows initial symptoms after a variable period. A lower visual field defect tends to progress more quickly than an upper defect due to gravitational forces that shift fluid faster on the upper part of the retina. Patients may also complain about a sudden increase in floaters or black spots when blood or pigment runs through the retinal break to the vitreous cavity. Visual acuity may be slightly affected due to obscuring floaters. A significant reduction of VA occurs when the macula is affected by the detached retina. (Kanski & Bowling, 2015, p. 701-703; Yanoff & Duker, 2018, p. 648-649.)

The detached retina appears as a convex, bullous, and corrugated configuration in clinical examination due to intraretinal edema. Retinal blood vessels are darker and more prominent because the underlying chorioidal pattern is compromised. Chronic retinal detachment develops pigmented demarcation lines at the junction of the detached and normal retina. The proliferation of RPE cells increases the adhesion at the junction, but it does not necessarily prevent the spread of detachment. In subclinical RRD, the dissection of subretinal fluid is limited within two-disc diameters from the break. These patients may also be asymptomatic at the time of diagnosis. Young myopic patients may develop RRD without concurrent PVD. In these cases, RRD starts when vitreous fluid migrates through atrophic holes under the retina. Lattice degeneration is often present. Other clinical features occasionally accompanying RRD are **relative afferent pupillary defect (RAPD)**, **lowered IOP**, **iritis**, and **tobacco dust**. RAPD may be present when there is extensive retinal detachment. Intraocular pressure tends to be about 5 mmHg lower in the eye with retinal detachment. Iritis,

if present, is usually mild but may cause synechiae and disregard the underlying retinal detachment. The presence of pigment in the anterior vitreous is highly specific to retinal breaks and should always raise a concern about possible RRD. Additionally, extensive vitreous haemorrhage or accumulation of inflammatory cells into the vitreous is always alarming. (Kanski & Bowling, 2015, p. 701-705; Yanoff & Duker, 2018, p. 648-649.)

Sometimes, the difference between RRD and exudative or tractional detachment may be hard to accomplish. Also, retinoschisis or choroidal lesions that elevate the retina may confuse the diagnoses of RRD. In tractional retinal detachment, the retinal surface is usually concave compared to the convex appearance in RRD. Tractional detachment is common in the eyes with proliferative diabetic retinopathy. Various reasons, such as tumours or AMD that leak fluid under the retina, may cause exudative retinal detachment. The underlying cause of exudative retinal detachment is often visible within or beneath the retina. Retinoschisis has a smoother appearance than RRD, and the elevation of the retina is noncorrugated. (Yanoff & Duker, 2018, p. 650.)

Peripheral retinal lesions

Peripheral retinal lesions are a group of anatomical variations and degenerative changes. Vitreous might have strong adhesion to these lesions; thus, during PVD, the risk of having tears and subsequent RRD is higher than in normal eye. Only the main peripheral lesions associated with RRD during PVD will be discussed in this chapter.

Lattice degeneration is clinically the most essential peripheral retinal lesion. Lattice is part of RRD in approximately 20-40 % of the cases (Mitry et al., 2010). The prevalence of lattice in the general population is 6-8 % (Flaxel et al., 2020c). In the lattice, the involved peripheral retina thins and becomes fibrotic. Above the lesion, the vitreous might form a lacuna (pocket of liquefaction), and in many cases, the affected retina becomes hyperpigmented. Vitreous is often firmly attached to the margins of the lattice. Lattice occurs commonly in moderate or highly myopic patients. It is usually bilateral and located at the temporal and/or superior retinal periphery from the equator to the vitreous base. Blood vessels within the lattice might become sclerotic and form a network of white lines. Small holes are common findings within the lattice. (Kanski & Bowling, 2015, p. 688-689.)

Snailtrack degeneration has similar features as a lattice. Peripheral retina thins, and "snailtrack" gets a glistening white, frost-like appearance. Some authors keep it a precursor of lattice or a variant of the same disease process. Snailtrack appears to be an elongated and isolated lesion in the peripheral retina from the equator to the ora serrata. In contrast to the lattice, the vitreous rarely exhibits strong adhesion to snailtrack, and thus, the risk of having retinal tears is lower than with the lattice. (Kanski & Bowling, 2015, p. 688-689.)

Degenerative retinoschisis (RS) is an idiopathic, degenerative process where retinal microcystic changes are thought to coalesce and split the neurosensory retina into inner and outer layers. Splitting leads to irreversible loss of retinal function in the affected area. RS is a relatively common finding in adult eyes (<40 years), as it is present in up to 7% of the population, especially in short (hypermetropic) eyes. The incidence of tractional retinal tears in retinoschisis is very low but may occur secondary to PVD. RS is thought to be responsible for less than 2.5 % of RRD cases. (Lewis, 2003.) Clinically, RS may be difficult to distinguish from RRD as both have a convex and bullous appearance. In RS, the elevation is smooth and immobile; in RRD, the elevation is corrugated, and fluid may shift underneath the retina. Visual field defect in RS is often absolute and in RRD relative. (Kanski & Bowling, 2015, p. 690-691.)

White with or without pressure degenerations appear as a whitish, translucent area in the peripheral retina. White with pressure is only apparent if scleral indentation is used. White with pressure may have abnormally strong adhesion to the vitreous, but the risk of retinal break formation is generally very low. White without pressure, in turn can be visualized without indentation and is thought to have stronger adhesion and carries more risk of RRD than white with pressure. White without pressure may need prophylactic treatment in the case of RRD in the fellow eye. (Kanski & Bowling, 2015, p.693-694.)

Chorioretinal atrophy is a common finding in highly myopic eyes and usually occurs in the posterior pole or occasionally in the equatorial area. It appears as patchy or diffuse choroidal depigmentation. In diffuse form, atrophic areas occur as yellowish-white lesions and patchy forms in greyish-white lesions. Commonly, the overlying retina is thinned or absent. (Ohno-Matsui et al., 2016.) Sometimes, retinal holes occur at the site of the atrophic retina and may cause retinal detachment (Kanski & Bowling, 2015, p. 693-694).

Retinal tufts are congenital, gliotic degenerations of peripheral retina, often with abnormally strong adhesion to the vitreous. Cystic retinal tufts are present in approximately 5% of the population and are associated with RRD in up to 10 % of the cases. Clinically, cystic tufts appear round or oval, small, and slightly elevated whitish lesions. The base may have pigmentary changes. Generally, tufts are localized from the equator to the ora serrata, and occasionally (20%), tufts can be found in both eyes. Tractional zonular tufts are thickened lens zonules projected posteriorly towards the anterior retina. Zonular tufts are not commonly associated with RRD. (Lewis, 2003.)

2.3.3 Management of Retinal Breaks and Rhegmatogenous Retinal Detachment

Currently, there is no clear international consensus on which kind of retinal breaks should be treated and which should be followed up. The benefits of treatment should be considered comparing to possible risks and costs of the treatment. Therefore, treatment plans are often made on a case-by-case basis. In each

case, considerations of at least the following factors should be weighted: age and systemic health of the patient; refractive error of the eye; location, age, type, and size of the break; status of the fellow eye; and whether the patient has aphakia or pseudo-phakia. (Yanoff & Duker, 2018, p. 642-643.) In this chapter, recommendations by the American Academy of Ophthalmology and current literature are utilized to gather a better understanding of the topic (Table 3).

According to Blindbaek et al. (2015), there is a big difference in whether retinal breaks are symptomatic or asymptomatic. Asymptomatic retinal breaks rarely (0-13.8%) lead to RRD, and therefore, prophylactic treatment may not be necessary. In contrast to asymptomatic breaks, symptomatic retinal breaks lead to RRD in 35-47 % of the cases. Despite the prophylactic treatment, RRD occurs in 2.1-8.8% of the cases. Prophylactic treatment is indicated with symptomatic retinal breaks because it reduces the possibility of subsequent RRD. (Blindbaek & Grauslund, 2015.) Acute symptomatic retinal horseshoe tears account for up to 98.5 % of all RRD cases related to PVD thus, treatment is indicated and should be performed promptly (Mityr et al., 2011).

Table 3. Treatment and follow-up plan of retinal breaks. (Adapted from Flaxel et al., 2020c)

Type of Lesion	Treatment and follow-up plan
Acute symptomatic horseshoe tears	Treat promptly.
Acute symptomatic operculated holes	Treatment may not be necessary. Follow-up interval: 2-4 weeks, 1-3 months, 3-6 months, then annually.
Acute symptomatic dialysis	Treat promptly.
Traumatic retinal breaks	Usually treated.
Asymptomatic horseshoe tears without SRF	Consider treatment unless there are signs of chronicity. Follow-up interval: 1-4 weeks, 2-4 months, 3-6 months, then annually.
Asymptomatic operculated tears	Treatment is rarely recommended. Follow-up interval: 1-4 weeks, 2-4 months, 6-12 months, then annually.
Asymptomatic operculated holes	Treatment is rarely recommended. Follow-up interval: 1-4 months, 6-12 months, then annually.
Asymptomatic atrophic round holes	Treatment is rarely recommended. Follow-up interval: 1-2 years.
Asymptomatic dialysis	Insufficient evidence to guide management. Follow-up interval if untreated: 1-4 weeks, 3 months, 6 months, then every 6 months.

Prophylactic treatment for retinal breaks is usually performed using cryopexy or laser photocoagulation (retinopexy). The idea of treatment is to generate solid chorioretinal adhesion. Adhesion is created immediately

adjacent to retinal breaks and surrounding the defect. Treatment blocks vitreous fluid from entering through the break further into the subretinal space. In cryotherapy, choriocapillaris, retinal pigment epithelium, and outer retina are destroyed by using a cryoprobe placed on the conjunctiva that overlies the break. The adhesion after treatment is not immediate but takes 1-3 weeks to develop fully. In laser photocoagulation, the retinal break is surrounded by three or four rows of laser burns. In contrast to cryotherapy, instant adhesion is accomplished in laser photocoagulation, and maximal adhesion does not take more than a week or so to be established. (Yanoff & Duker, 2018, p. 644.)

RRD is usually treated with pneumatic retinopexy, scleral buckle (SB), pars plana vitrectomy (PPV), or by a combination of these techniques. In pneumatic retinopexy, the detached retina is re-attached using an intravitreal gas bubble and cryotherapy or laser. The procedure is quick and minimally invasive, but success rates are inferior to SB or PPV. In SB, soft or hard silicon explant is sutured onto the sclera, creating an inward indentation that seals the retinal breaks by apposing retinal pigment epithelium to the sensory retina. A buckle should surround the detached area from the vitreous base to the tear to prevent possible re-detachment. (Kanski & Bowling, 2015, p. 708-716.)

In PPV, small instruments are used to cut the vitreous gel into tiny pieces and simultaneous suction for removing the vitreous. Tamponating agents are combined with subretinal fluid drainage to flatten the retina and help the re-attachment. With expanding gases such as sulfur hexafluoride, perfluorethane, or perfluoropopane, prolonged tamponade is achieved. (Kanski & Bowling, 2015, p. 708-716.) Patients are often recommended to keep specific postoperative positioning to maintain tension around the detached area, although patients' compliance with postoperative recommendations varies a lot. Male patients who are recommended to keep face-down positioning after RRD and PPV may be challenging to motivate due to inconvenience factors. (Suzuki et al., 2018.)

According to Dhoot et al. (2022), success rates of the final re-attachment between SB and PPV are very similar (96.7% vs. 97.7%). Slightly better corrected visual acuity is achieved with SB (20/48 vs. 20/43), but the difference may not influence the quality of the patient's life. SB is associated with fewer cataracts and iatrogenic retinal breaks, but PPV has reduced the risk of choroidal detachment and subretinal haemorrhage. (Dhoot et al., 2022.) A few percent of the patients also develop diplopia after SB operation, mainly due to the mechanical restriction of one extraocular muscle. Conventional prism correction is a sufficient treatment in most cases. (Goezinne et al., 2012.)

In every RRD operation, intra, or post-operative complications are possible. Visual acuity in the macula-on retinal detachment is in 90 % of the cases 6/12 or better, but in macula-off cases, only about 50% will have visual acuity 6/15 or better if the macula has been detached for a week or more. (Steel, 2014.) Epiretinal and subretinal membranes (scar tissues) are common findings after the operation, and 5-10 % of the patients

develop proliferative vitreoretinopathy (PVR). PVR may cause disappointing visual outcomes due to contracting scar tissue that leads to macular pucker, retinal breaks, and recurrent retinal detachment. Management of PVR is a significant challenge to vitreoretinal surgeons. (Pastor et al., 2016.)

2.4 Complications of Early-stage Posterior Vitreous Detachment

Anomalous posterior vitreous detachment may lead to complications in the peripheral retina or lead to complications in the macular area. Complications in the macular area are usually part of the same pathogenesis, abnormally strong vitreomacular adhesion. Macular complications have many overlapping features but might also appear in isolation. According to Johnson (2010), tractional effects during the early stages of PVD cause or exacerbate various macular pathologic features. Although many of the complications in the macula area may happen at any stage of the PVD process, they mainly occur during the early stages. (Johnson, 2010.) This chapter covers the most significant macular complications of anomalous posterior vitreous detachment.

2.4.1 Vitreomacular Traction

Vitreomacular adhesion (VMA) refers to the residual adhesion between the vitreous cortex and retinal ILM around the macular area within a 3 mm zone. It may be classified as focal ($\leq 1500 \mu\text{m}$) or broad ($> 1500 \mu\text{m}$) depending on the diameter of the adhesion. VMA is a normal finding during the natural course of PVD and is not associated with distortion of macular architecture and visual symptoms. When the PVD progresses, and the weakening of VMA does not occur concurrently with liquefaction or gel contraction, excessive traction may result in the macula area. (Duker et al., 2013.)

In contrast to VMA, vitreomacular traction (VMT) refers to the condition where PVD has taken anomalous features and the anatomical contour of the fovea and macula is altered. Just like VMA, VMT can also be classified into focal or broad. As mentioned in Chapter 2.2.4, both VMT types, focal and broad, are related to specific disease entities. Focal adhesion with traction tends to cause foveal distortion in several forms, and broader adhesion may exacerbate concurrent diseases and lead to macular schisis and vitreomacular traction syndrome. In contrast to the macular hole, where there is full-thickness disruption of all retinal layers, in VMT, there might occur distortion of the foveal surface, intraretinal structural changes, and elevation of the fovea above the RPE, but there is no full-thickness disruption of all layers. (Duker et al., 2013; Johnson, 2010.)

VMT is frequently associated with symptoms of metamorphopsia, micropsia, reduced visual acuity, and occasionally photopsia. The most important risk factors for VMT are age and female gender. (Shao et al.,

2021.) Another term that is often used in relation to anomalous early-stage PVD is symptomatic vitreomacular adhesion (SVMA). SVMA refers to visual loss secondary to foveal damage caused by abnormal VMT. SVMA covers isolated VMT, impending macular hole, and macular hole with persisting vitreous adhesion. (Jackson et al., 2013b.)

According to Shao et al. (2021), the prevalence of VMT in the Chinese population is approximately 2.3 %, and the mean age is 64.6 ± 9.8 years (Shao et al., 2021). Another study by Menzler et al. (2019) found the prevalence of VMT to be slightly lower, approximately 1.4% (Menzler et al., 2019). Studies that have been made about symptomatic VMA and VMT are not entirely transformable for the idea of classic symptomatic VMT. Thus, Shao et al. (2021) proposed that the actual prevalence for the classic symptomatic VMT is probably around one percent. These patients are at high risk of developing associated diseases, such as macular hole, epiretinal membrane, diabetic maculopathy, and AMD. (Shao et al., 2021.)

According to Flaxel et al. (2020), surgical intervention for VMT is relevant when the patient cannot cope with daily activities. In the cases with VMT smaller than $1500 \mu\text{m}$, the VA is generally stable, and up to 50% of the patients undergo spontaneous release of traction. In contrast, patients with a broad VMT area ($>1500 \mu\text{m}$), pathological detachment of macula, or poor visual acuity are more likely to need surgical intervention. (Flaxel et al., 2020a.) According to the author's knowledge, pars plana vitrectomy is the mainstream treatment for VMT in Finland. Enzymatic vitreolysis is not generally used in Finnish healthcare settings.

Traction Diabetic Macular Edema refers to the condition where diabetic macular edema (DME) is exacerbated by vitreomacular traction, typically during stage 1 PVD. The exact mechanism of DME and the role of the vitreous in its development is not well understood. The posterior hyaloid is thickened and vitreomacular adhesion strong, which leads to longstanding traction and DME formation. Vitrectomy with peeling of the posterior hyaloid and possible ERM often promotes the resolution of DME. (Johnson, 2010.)

Myopic Traction Maculopathy (MTM) refers to anomalous PVD with associated vitreomacular traction in highly myopic (>-6 dpt) eyes. It's a relatively rare entity in which VMT is often significantly different from emmetropic eyes. MTM, or by another name, foveoschisis, is a broad spectrum of pathologies including at least inner schisis, outer schisis, lamellar macular hole, full thickness macular hole, intact fovea, and macular detachment. It is considered multifactorial, and vitreomacular traction is only one part. Usually, the inner retina is affected first, and schisis progresses to encompass the outer retina. MTM leads to full-thickness macular hole formation and macular detachment in advanced stages. Patients usually complain of gradual, often progressive, painless deterioration of vision. Pars plana vitrectomy and internal limiting membrane (ILM) peeling seem to be the best treatment option in MTM. (Raizada & Sahu, 2023.)

Neovascular Age-Related Macular Degeneration has been associated with vitreomacular traction in the early stages of PVD. Partial PVD with persistent VMT has been reported to increase the incidence of wet AMD. In contrast, total PVD is a protective element against AMD. Persistent VMT exposes the retina to at least low-grade inflammation, free radicals, cytokines, and lower macular oxygen levels, which may promote neovascular disease. (Johnson, 2010.) According to Jackson et al. (2013), the prevalence of vitreomacular adhesion in the eyes with wet AMD is roughly two times higher than those without wet AMD. PVD is less likely to occur in the eyes with wet AMD. The release of VMT will most likely improve the course of neovascular AMD and lead to anatomical and functional improvement. (Jackson et al., 2013a.)

Vitreopapillary Traction Syndrome is usually seen in patients with existing vascular disease, such as diabetic retinopathy, but may also occur in otherwise healthy eyes. In clinical examination, the optic nerve appears “full” and elevated. Sometimes, subtle whitening might occur in the peripapillary nerve fiber layer, which can mimic nontractional disc edema. Also, intra- or peripapillary haemorrhages may occur. Patients are mainly asymptomatic but can complain of transient photopsia. (Johnson, 2010.)

Vitreomacular traction syndrome (classic form) refers to the condition where the vitreous is detached throughout the peripheral fundus but remains adherent to the posterior pole. The result is broad anteroposterior traction that encompasses the macula area and papilla. The adhesive area may be multifocal and typically more extensive than in vitreofoveolar traction, generally from 1.5 to 3 mm in diameter. The development of a full-thickness macular hole is rare, but associated findings include macular pucker, cystoid macular edema (CMO), and tractional macular detachment. If ERM is present concurrently, it increases the adhesion between the macula and vitreous, thus resulting in longstanding vitreomacular traction. (Johnson, 2010.)

2.4.2 Idiopathic Macular Hole

A macular hole is a full-thickness defect in the neurosensory retina in the centre of the macula. It is a relatively common cause of visual deterioration in the aging population of 55 years or older. Females are affected more often than males, although macular holes can occur regardless of sex or race. FTHM commonly occurs due to high myopia, blunt ocular trauma, or advanced VMT. (Flaxel et al., 2020b.) In the case of VMT-related macular hole, the clinical term idiopathic macular hole (IMH) is often used. The approximate incidence of full-thickness IMH has been reported to vary from 4 to 8 per 100,000 per annum. The highest incidence has been found in women aged 60 to 70, approximately 30 per 100,000. Bilaterally, IMH occurs in 10 % of the cases. If the macular hole is left untreated, it will most likely lead to significant visual reduction with best corrected VA 20/200. (Murphy et al., 2023.)

Common complaints related to macular holes are metamorphopsia and reduced visual acuity. The central scotoma will appear in advanced stages when the macular hole enlarges. Sometimes, FTMH may be confused with a **lamellar macular hole** or **macular pseudohole**, which may have similar clinical features (Figure 6). (Flaxel et al., 2020b.) In the lamellar macular hole, vitreofoveal separation leads to defects in the inner retinal layers, and the outer photoreceptor layers stay intact. Vision usually is relatively good, but development of FTMH may occasionally occur. Macular pseudohole develops concurrently with ERM and may mimic the clinical appearance of FTMH. In macular pseudohole, the perifoveal retina has heaped edges, but the neural retina stays intact, and the foveal thickness is near-normal. (Kanski & Bowling, 2015, p.622-623.)

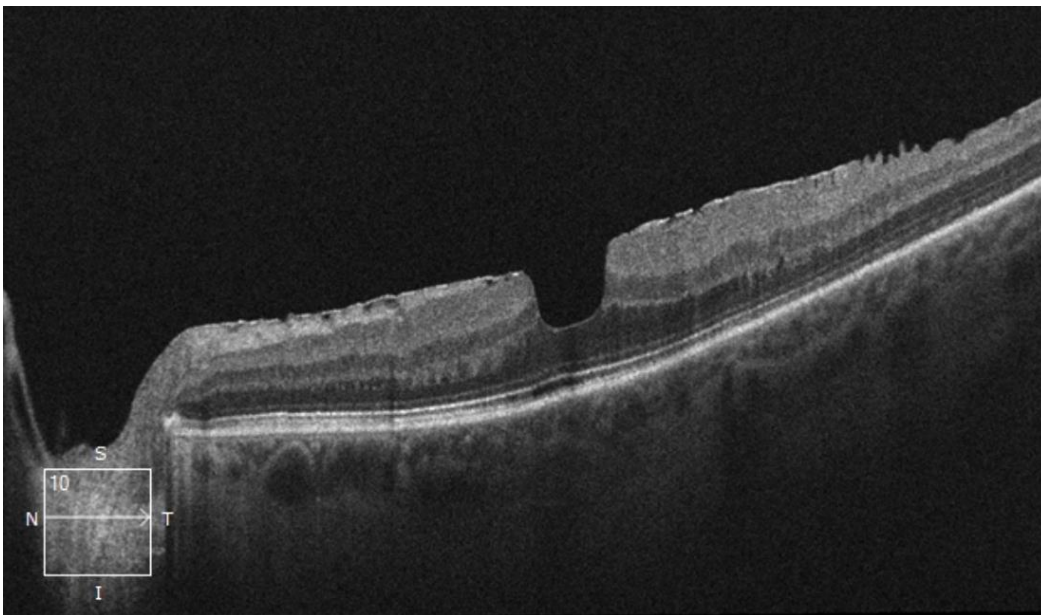


Figure 6. OCT image of macular pseudohole. The neurosensory retina at the base of the fovea is intact. ERM is visible on the retina as a thin hyperreflective line. BCVA 20/20. OCT image: Anssi Hakala

Depending on the clinical features and OCT findings, a macular hole has been classified into four stages. Table 4 lists all stages, clinical findings, and management recommendations. Some findings, such as VMT and pseudocyst formation, are easiest seen with OCT. (Duker et al., 2013.) A yellow spot and reddish appearance of FTHM can be visualized, for example, by using a biomicroscope and fundus contact lens (Figure 7) (Kanski & Bowling, 2015, p. 620). **OCT** imaging has developed the understanding of vitreomacular interface diseases and has become a significant part of eyecare (Figure 8). OCT is a primary diagnostic tool in vitreomacular diseases. (Stalmans et al., 2013.)

If OCT is not available, alternative methods in the clinical examination include the **Amsler** grid and the **Watzke-Allen** slit lamp beam test. Amsler grid is frequently used to detect eye diseases that cause metamorphopsia or central scotoma, such as vitreoretinal interface diseases. However, the sensitivity of the

Amsler grid is poor, and thus, it does not replace the need for a thorough clinical eye examination (Tripathy & Salini, 2023). In the Watzke-Allen test, a narrow slit-lamp beam using a fundus contact lens is focused over the macula, first vertically and then horizontally. In the case of a macular hole, the patient will report that the beam appears broken or thinned. In other pathological conditions, the beam appears bent or distorted but is usually uniform in thickness. (Kanski & Bowling, 2015, 621.)

Table 4. Appearance and symptoms of VMT and FTMH, and their recommended follow-up and management plan. (Adapted from Flaxel et al., 2020b; Duker et al., 2013)

Stage	Clinical appearance and symptoms	Follow-up and management
1a	<ul style="list-style-type: none"> - Loss of foveal depression due to persistent VMT - Pseudocyst formation (OCT) - Yellowish foveal spot, 100-200 μm - VA: 20/25 to 20/80 	<ul style="list-style-type: none"> - Observation - Follow-up in 2-4 months intervals - Amsler grid home testing
1b	<ul style="list-style-type: none"> - Persistent VMT - Yellow ring 200-350 μm - Pseudocyst with disruption of the outer retinal layers (OCT) - VA: 20/25 to 20/80 	<ul style="list-style-type: none"> - Observation - Follow-up in 2-4 months intervals - Amsler grid home testing
2	<ul style="list-style-type: none"> - Small or medium FTMH (<400 μm), often eccentric - VMT is present - Metamorphopsia - VA: 20/25 to 20/80 	<ul style="list-style-type: none"> - Urgent referral to an ophthalmologist within one week (local protocols) - Enzymatic vitreolysis (Ocriplasmin) - Vitreoretinal surgery
3	<ul style="list-style-type: none"> - Medium or large FTMH (>250 μm) - Posterior hyaloid is detached from the macula - Visible operculum - Drusen like deposit may be seen at the base of the macular hole - ERMs may be present - Cuff of subretinal fluid and/or intraretinal oedema and cysts may be seen - VA: 20/100 to 20/400 	<ul style="list-style-type: none"> - Urgent referral to an ophthalmologist within one week (local protocols) - Vitreoretinal surgery
4	<ul style="list-style-type: none"> - Large FTMH (>400 μm) - Total PVD with Weiss ring - ERMs are frequently present - Cuff of subretinal fluid, intraretinal oedema, and cysts are usually present - Drusen-like deposits may be seen at the base of the macular hole - VA: 20/100 to 20/400 - Central scotoma 	<ul style="list-style-type: none"> - Urgent referral to an ophthalmologist within one week (local protocols) - Vitreoretinal surgery

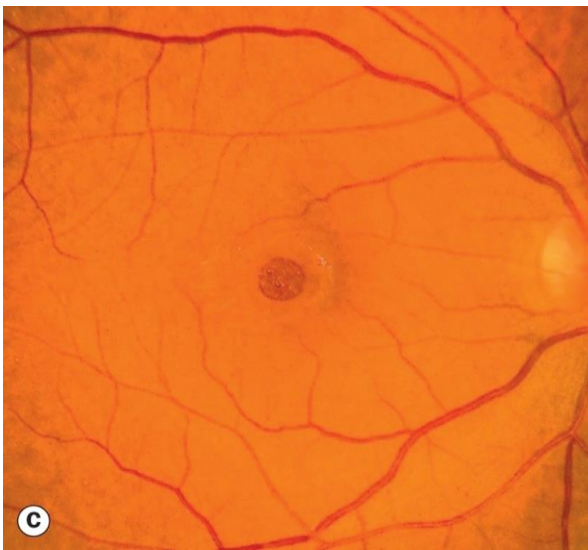
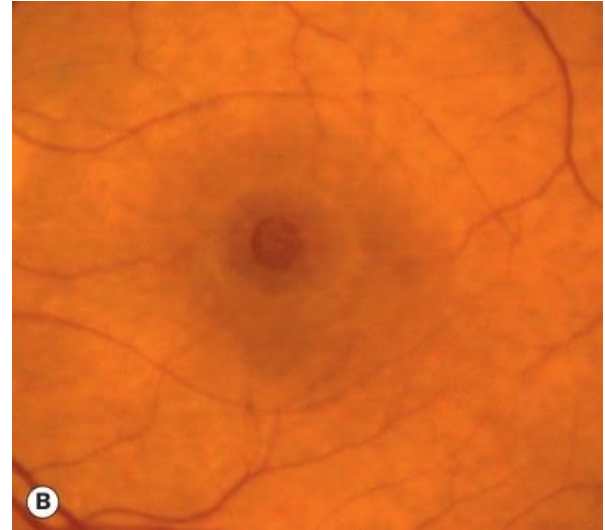
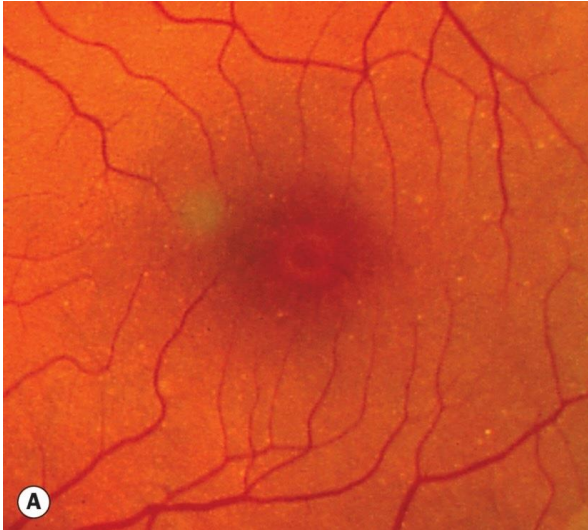


Figure 7.

A: **Stage 1b** – Impending macular hole, yellow ring visible.

B: **Stage 2** – Small full-thickness macular hole

C: **Stage 3** – Large full-thickness macular hole

Reprinted with permission. (Kanski & Bowling, 2015, p. 620)

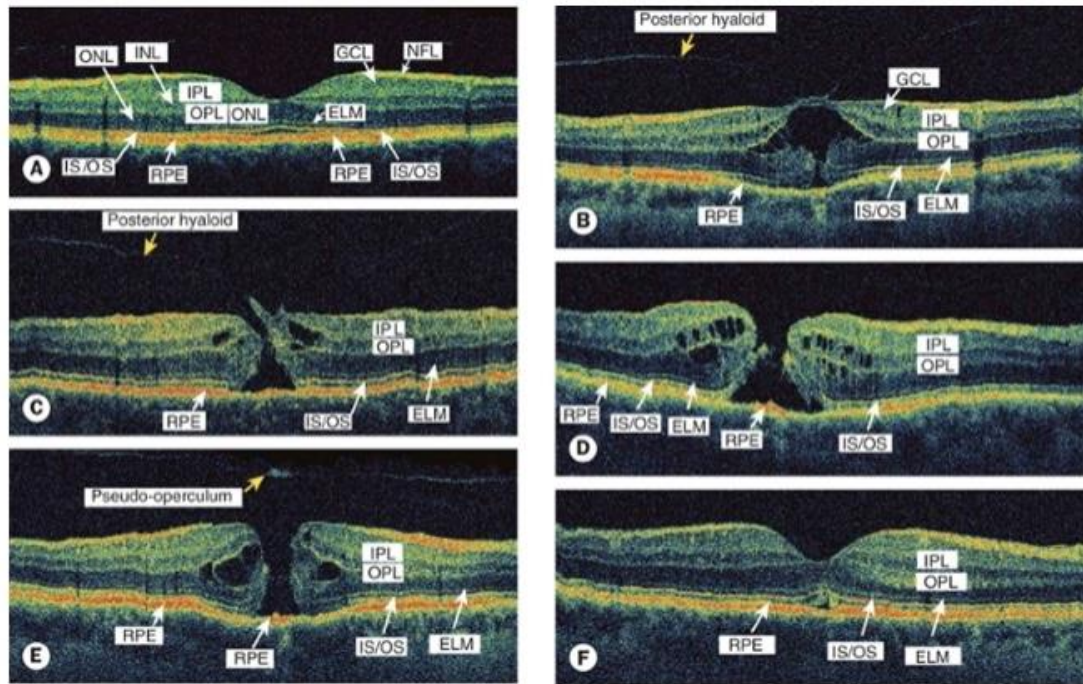


Figure 8. OCT findings of macular hole. (A): **Normal OCT**, (B): **Stage 1b**. Focal VMT and separation of a small portion of the sensory retina from RPE, small intraretinal cystic changes. (C): **Stage 2**. Eccentric small FTMH, VMT still present, cystic changes. (D): **Stage 3**. Medium/Large FTMH with intraretinal cystic changes, VMT still present. (E): **Stage 4**. Large FTMH without VMT, operculum present. (F): Surgical closure after stage 4 FTMH. The outer retinal disturbance is visible. (ELM= external limiting membrane; GCL: ganglion cell layer; INL= inner nuclear layer; IPL= inner plexiform; IO/OS= photoreceptor inner-segment/outer-segment junction; RPE= retinal pigment epithelium). Reprinted with permission. (Kanski & Bowling, 2015, p. 621)

Macular holes are treated with vitrectomy. Intravitreal injection of different agents is used to seal the hole and re-achieve the normal anatomic architecture of the fovea and macula. Surgical outcomes vary greatly depending on the hole's size and the symptoms' duration. Closure rates of FTMH have been reported to be from 91% to 98%. The best corrected VA is dependent on the VA before the operation. The median best corrected VA is approximately 20/40 after the operation, which is significantly better than the VA without closure operation. (Flaxel et al., 2020b.)

2.4.3 Idiopathic Epiretinal Membrane

Epiretinal membrane (ERM) is “an avascular, fibrocellular membrane that proliferates on the inner surface of the retina to produce various degrees of macular dysfunction.” ERM may be caused idiopathically or secondary to various conditions, such as retinal breaks and rhegmatogenous retinal detachment, retinal vascular diseases, intraocular inflammation, and blunt trauma. ERM is a transparent, opaque, or pigmented

membrane that causes tangential (sideways) traction onto the retina. The severity of visual symptoms depends on the opacity of the ERM and the distortion that contracting tissue causes to the retinal surface. (Yanoff & Duker, 2018, p. 616.) Patients with ERM typically complain of metamorphopsia, reduced VA, micropsia or macropsia, binocular vision problems, and even diplopia. Significant metamorphopsia often affects a patient's quality of life, leading to problems with reading, vision, and daily activities. (Kanukollu & Agarwal, 2023.)

Although the exact mechanism behind idiopathic ERM is poorly understood, partial or complete PVD has been found in 80 to 95 % of the cases. Thus, the PVD has been suggested to be the causative reason in most of the idiopathic ERMs. The pathogenesis of idiopathic ERM is considered multifactorial and most likely differs from the pathogenesis of ERM after the retinal breaks and RRD. Partial or complete PVD causes ILM ruptures, allowing glial cells to migrate and proliferate onto the retinal surface. Also, remnants of the cortical vitreous that contain hyalocytes may proliferate and cause ERM after the PVD. Some idiopathic ERMs (10-25%) may occur without clinical signs of PVD when pre-existing defects in the retinal ILM lead to cellular migration and subsequent ERM formation. (Johnson, 2010.)

The primary diagnoses are often made according to clinical findings on fundus contact lens (e.g., Volk 90) biomicroscopy. The additional examination includes fundus photography with red-free light and OCT. (Yanoff & Duker, 2018, p. 618.) OCT is a fast and non-invasive tool that has become the procedure of choice for diagnosing and assessing ERMs and possible alterations in retinal morphology (Kanukollu & Agarwal, 2023). OCT is also helpful in differentiating macular pseudoholes, lamellar, and full-thickness macular holes, which may be hard to distinguish according to biomicroscope findings. As mentioned previously (see chapter 2.4.2), Watzke-Allen may be used to differentiate macular holes from other macular conditions, such as a lamellar macular hole or macular pseudohole, thus, it is also helpful in the case of ERM. (Yanoff & Duker, 2018, p. 618-619.)

The mildest form of ERM is called **cellophane maculopathy**. In cellophane maculopathy, the membrane on the retinal surface is thin and transparent. In clinical examination, abnormal glistening of the retina is often present, but there is no visible vessel tortuosity or striae formation. Patients are mainly asymptomatic. If the thin membranes undergo contraction, it leads to irregular striations or wrinkles of the inner retinal surface and ILM. In clinical examination, striae are often most apparent when they radiate out from the margins of the membrane (Figure 9). Fine capillaries may be tortuous, even if the larger blood vessels have a normal appearance. (Yanoff & Duker, 2018, p. 616-617.)

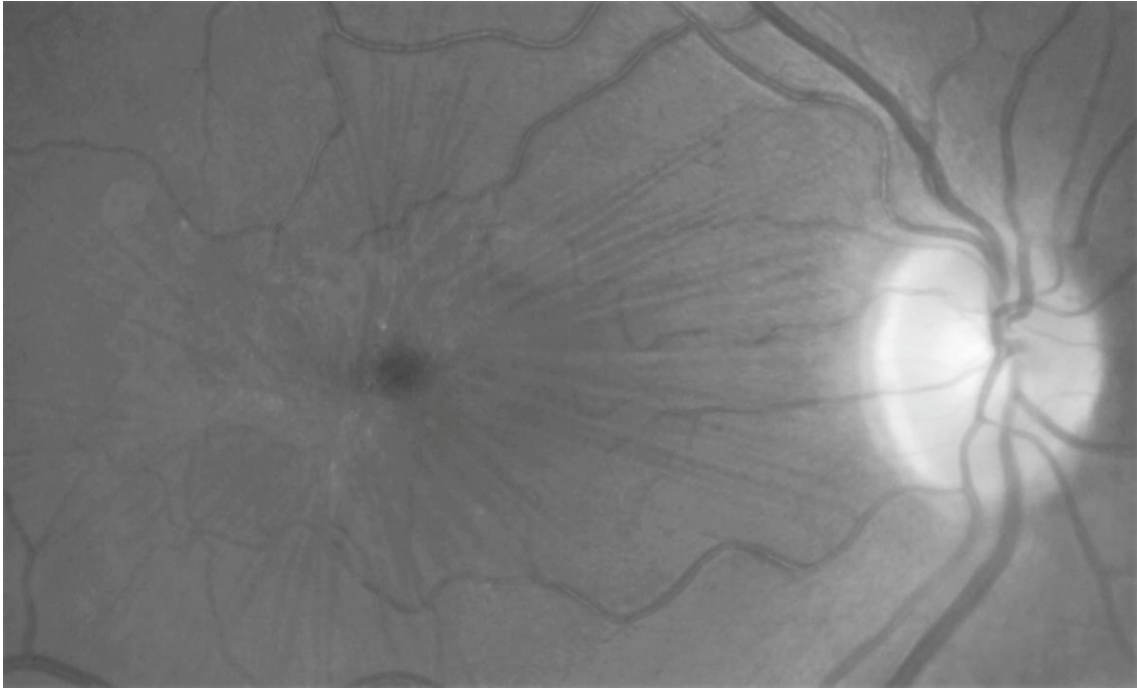


Figure 9. Red-free photography of mild ERM around the fovea. Picture: Anssi Hakala

Thicker ERMs, also called **macular pucker** or **preretinal macular fibrosis**, with significant contraction, cause full-thickness tangential traction to the retina and may lead to severe macular dysfunction (Figure 10). Thicker ERM may appear as a grey-white translucent membrane or stay relatively invisible in clinical examination. Blood vessels are often significantly tortuous or straightened up. Common complaints with thicker contracted membranes include reduced VA, metamorphopsia, and occasionally central photopia. Patients may even complain of diplopia if the contracted membrane displaces the fovea. A contracting membrane may lead to macular edema, preretinal or intraretinal haemorrhage, or traction macular detachment. Furthermore, ERM is a common cause of macular pseudohole with or without inner lamellar macular defect, but FTHM due to tangential traction is relatively uncommon. In the cases of longstanding ERM or vascular leakage, RPE hypertrophic or atrophic changes are possible. (Yanoff & Duker, 2018, p. 617-618.)

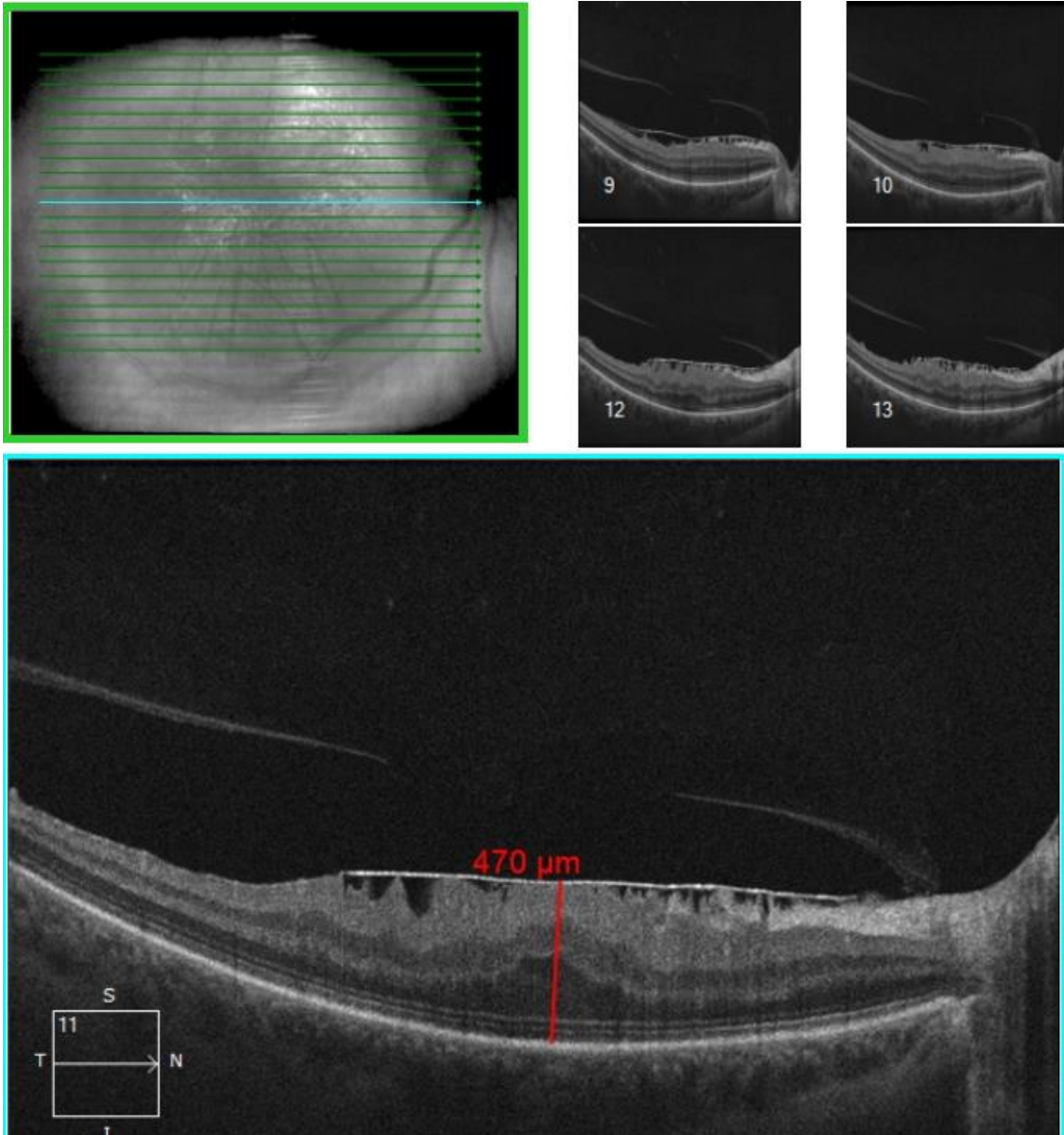


Figure 10. ERM and retinal thickening after partial PVD. The hyaloid membrane is still attached to the optic nerve and visible above the retinal layers. BCVA 20/40. OCT image: Anssi Hakala

According to Xiao et al. (2017), ERM in some form is present in approximately 9 % of the general population. Cellophane maculopathy is more common (6.5 %) than the more advanced, thicker, fibrotic ERM (2.5 %). Female gender and old age are the most significant risk factors of ERM, but race and ethnicity are thought to have only a limited role in ERM prevalence. (Xiao et al., 2017.) Fraser-Bell et al. (2003) studied the progression of different forms of ERM over five years in the Australian population 49 years of age or older (n = 2335). ERM was classified as either preretinal macular fibrosis or less severe cellophane maculopathy. The progression from cellophane maculopathy to preretinal macular fibrosis was observed in 9.3 % of the cases. Either form of ERMs progressed, regressed, or remained stable in 28.6%, 25.7%, and 38.8% of the cases. 13.5 % of the patients who had ERM in one eye also developed ERM in the other during the 5-year interval. (Fraser-Bell et al., 2003.)

Many patients with ERM stay relatively asymptomatic, whereas others may experience a significant reduction in their quality of life. As mentioned, roughly one-third of the ERM cases will remain stable over time, one-third will regress, and only one-third will worsen. It's essential to educate patients about the nature of ERM and give information about alternative option of the surgical procedure. The mildest form of ERM, cellophane maculopathy, progresses to more advanced ERM only in one out of ten cases; therefore, observation alone is sufficient for practise. ERM will progress over time in little less than one-third of the cases. Thus, regular home testing with **Amsler grid** is recommended. Patients should be advised to do the test monocularly with their reading correction. If ERM progresses and patients experience increasing metamorphopsia, reduced visual acuity, or central scotoma, they should be advised to seek urgent clinical eye examinations. (Flaxel et al., 2020a.)

Surgery is often the treatment of choice if patients experience severe symptoms and ERM negatively impacts their daily activities. Prophylactic surgical intervention is unnecessary in mild cases because ERM often shows little or no progression. The surgical treatment option for ERM is **vitrectomy** with peeling of the epiretinal tissue from the macular surface and release of the possible VMT. Sometimes, ILM is peeled concurrently with ERM. It is thought to reduce the recurrence rate, but the benefit of this technique remains controversial. (Yanoff & Duker, 2018, p. 620.) In the research by Kim et al. (2012), 77 % of the patients achieved VA improvement of more than two Snellen lines within 6 to 12 months after the operation. This result is in line with previous studies, in which VA has reported an increase of $2\geq$ Snellen lines in 70-80 % of the cases. (Kim et al., 2012.) The rest of the patients mainly have unchanged VA, and only a minority experience a reduction in VA. Vitrectomy is frequently associated with several complications, such as nuclear sclerotic cataract, endophthalmitis, retinal breaks, and RRD. Therefore, the pros and cons should be calculated before the decision of vitrectomy. (Yanoff & Duker, 2018, p. 620.)

3 THE PURPOSE AND OBJECTIVES OF THE RESEARCH DEVELOPMENT WORK

3.1 Purpose and Objectives of the Research Development

Purpose: The purpose of this research development is to produce evidence-based material about posterior vitreous detachment and its complications. The thesis aims to create a clinical guideline for Finnish optometrists on assessing posterior vitreous detachment.

Objectives: This research development work has two study objectives. The **first** study objective is to conduct a narrative literature review about the most common complications of posterior vitreous detachment and their prognosis. The **second** study objective is to produce a clinical guideline for Finnish optometrists: Assessment of the Posterior Vitreous Detachment.

3.2 Statement of the Research Question

The research question for this narrative literature review was based on a Population, Concept, and Context (PCC). PCC consists of the following pieces:

P (Population) = Patients with posterior vitreous detachment (either early-stage or acute)

C (Concept) = Complications related to posterior vitreous detachment

C (Context) = Healthcare setting

3.2.1 Research Question of Research Phase

1. What are the most common complications of posterior vitreous detachment?
2. What is the prognosis for posterior vitreous detachment's most common complications?

3.2.2 Research Question of Development Phase

What are the main clinical elements of the posterior vitreous detachment assessment in an optometrist's eye examination?

4 IMPLEMENTATION OF THE RESEARCH DEVELOPMENT WORK

4.1 Narrative Literature Review as a Research Method

Literature reviews are traditionally divided into three main categories: systematic review, narrative review, and meta-analysis (Stolt et al. 2016). Systematic literature reviews have been considered a backbone of the clinical practice guidelines, but as a master program thesis, a systematic review would have been too extensive to conduct. Thus, the decision to conduct a narrative literature review was made. A narrative literature review aims to “summarize, explain and interpret evidence on a particular topic/question drawing on qualitative and/or quantitative evidence” (Mays et al., 2005).

The fundamental idea of the literature review is to create an understandable general view of previous research knowledge. In literature reviews, some typical components are universal, regardless of the different main categories and subtypes that each literature review type consists of. These components are literature **Search**, critical **Appraisal**, **Synthesis**, and **Analysis** (SALSA). In each literature review type, the implementation of these components has distinctive features. When the literature review is executed systematically, and each stage is described thoroughly, the reliability and overall conduction of the review is easier to evaluate. (Grant & Booth, 2009.)

A narrative literature review conducts a thorough and critical overview of published material, providing an inspection of previous or current literature. Generally, the material in narrative literature reviews cover a wide range of subject matter with variable degree of comprehensiveness. The included material possesses some degree of permanence and may have undergone a peer-review process. (Grant & Booth, 2009.) The research question is typically broad and may include subcategories. A narrative literature review is not the most systematic and rigorous, and therefore, it has been criticized for being prone to weaknesses and bias. For instance, the critical evaluation process of previous studies does not include as systematic approach compared to a systematic review. (Stolt et al. 2016, p. 9, 10) However, narrative review is useful to demonstrate what has been accomplished previously. It identifies the literature gaps and summarizes the evidence on a particular topic. (Grant & Booth, 2009.)

4.2 Selection of the Studies

Literature search and selection is a critical part of the review process. Also, in a narrative literature review, a search strategy should have a systematic approach to increase the reliability of the review. If the search is done without any strategy, this may lead to bias. The literature search aims to find all the essential material

to answer the research questions. (Stolt et al. 2016, p. 24-25.) Traditionally, the search strategy and collection of data in narrative literature reviews have been criticized for lacking an explicit intent to maximize the scope of data (Grant & Booth, 2009). However, nothing restrains the author from being as explicit and systematic as possible in conducting a narrative literature review (Vilkkä, H. 2023).

Searching from different databases may give an enormous number of results if the keywords and search terms are not correctly designed. Information specialists are valuable sources to ensure and determine the optimal key search terms. Furthermore, using the key search terms often gives a wide range of studies unsuitable for the review. Inclusion and exclusion criteria clarify the selection process, which starts from the title, proceeds to the abstract, and finally to the whole text. A literature search is often the most time-consuming part of the review, but it should always be done with such accuracy that the reader may repeat the same search process if necessary. The search process is never perfect, and the implementation depends on the available resources. Thus, the conclusion section should include the evaluation of the pros and cons, which are inevitable in every research process. (Stolt et al. 2016, p. 27-28.)

Inclusion and exclusion criteria were guided by the Finnish laws and regulations related to the optometrist's work (Table 4 and Table 5). Optometrists and opticians are licensed healthcare professionals in Finland. Optometrists have the right to use diagnostic pharmaceuticals; thus, their working description is broader and more focused on examining the healthiness of customers' eyes. The most important regulations are as follows:

Regulation on health care professionals (564/1994, 16 §): Optometrists and opticians are not allowed to prescribe glasses independently:

- a child under the age of eight
- a person who has previously had eye surgery on the eyeball
- a person who appears to have an eye disease
- a person whose visual acuity will not become normal with eyeglasses

(Asetus terveydenhuollon ammattihenkilöistä 564/1994 – Ajantasainen lainsäädäntö – FINLEX ® s.a.)

Regulation on health care professionals (785/1992, 5 §): Optometrists have an obligation to inform the patient about their findings and conclusions (Laki potilaan asemasta ja oikeuksista 785/1992 - Ajantasainen lainsäädäntö - FINLEX ® s.a.)

Table 4. Inclusion criteria for the narrative literature review

Studies that investigate PVD or at least one of the main complications

Studies that are made between 2010 and 2022

The study is available in full text, and it is free

Only studies in Finnish or English are eligible

Only studies that are related to at least one of the Research Questions are eligible

Only studies that included adult patients

Only studies with more than 900 participants are eligible ($N \geq 900$)

Table 5. Exclusion criteria for the narrative literature review

Studies that are not directly related to PVD or its main complications

Studies that do not answer either research questions

Studies included pediatric patients

Studies that are not free and the full text is not available

Studies that are not published in Finnish or English

Studies that are published before 2010

Studies that do not have enough participants ($N \leq 900$)

The initial search process started in the spring and fall of 2022 from different databases, primarily from PubMed and Google Scholar. The purpose of the initial search was to identify and evaluate the key subjects for the thesis and scope the number of studies that are likely to be found. A more thorough search process started in January 2023. As a topic, posterior vitreous detachment is broad, and a literature review is needed to serve the purpose of making guidelines for Finnish optometrists. Furthermore, two separate research questions were considered to suit best for the guideline development stage. A meeting with an Oulu University of Applied Sciences information specialist was arranged to determine the essential search terms. Also, the relevant databases were selected with information specialists. Two databases, Pubmed and Medic, were selected. PubMed has the broadest range of studies in the optometric field; therefore, it was an obvious choice for the main database.

All databases have their way of processing information. Therefore, the key search terms need to be adjusted individually for each database to yield the most appropriate findings. (Stolt et al. 2016, p.42-43.) The main literature search was conducted on 15 May 2023 from PubMed and Medic. The key search terms for PubMed: "posterior vitreous detachment" AND ("complication*" OR "retinal detachment" OR "epiretinal membrane" OR "vitreomacular traction" OR "vitreomacular adhesion" OR "macular hole" OR "retinal break"). Time limitation was set up to cover studies between 2010 and 2022. Only studies published in English were selected. At least an abstract from each study needed to be available, but no other limitations were used. The search yielded **518** results. The key search terms for Medic were ("lasiaisirtauma" OR "verkkokalvon irtauma"). Time limitations were set up to be the same as in PubMed 2010-2022; no other limitations were used. A search from Medic yielded **9** results. In Medic, also wide range of individual Finnish and English searching terms related to posterior vitreous detachment were used, but no additional results were found.

Results were assessed according to the inclusion and exclusion criteria, starting from the title and proceeding to the abstract. If the title included either eye surgeries or significant ocular disease, it was excluded automatically. Also, studies regarding pediatric patients were excluded in the title screening stage. Finnish optometrists are not allowed to prescribe glasses to customers with eye diseases or previous eye surgeries, and therefore, most patients who seek the service of optometrists have healthy eyes. Overall, **527** titles were screened, and **369** records were excluded in the title screening stage. The second stage included the screening of the abstracts. **158** abstracts were screened to find the studies for the final stage, in which the whole text was read through. After reading full versions of **15** studies, **5** were excluded because they did not meet the inclusion criteria, and **1** was excluded due to reliability issues (Figure 11). **5** of the selected studies had direct open access, and the remaining **4** were obtained through an information specialist with the access rights of the University of Oulu.

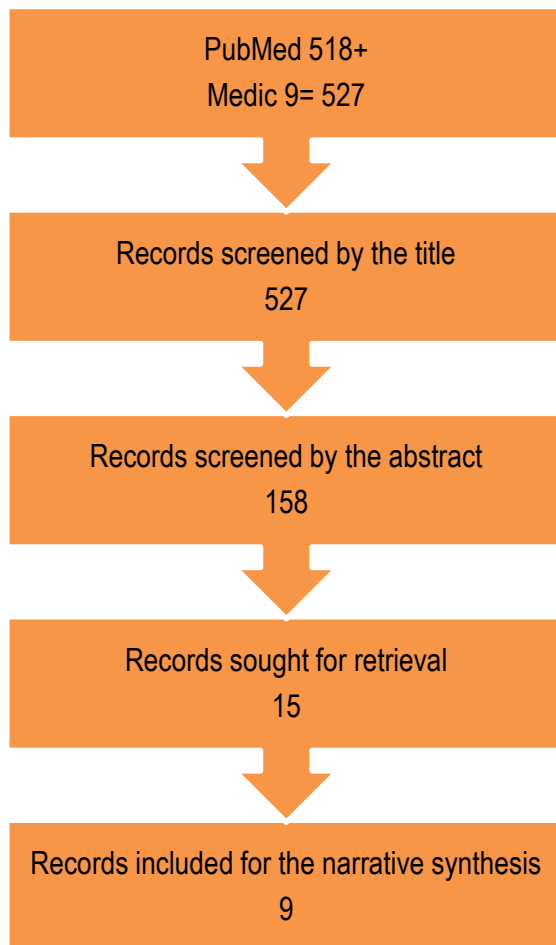


Figure 11. The data search process

4.3 Evaluation of the Data

The third stage of the literature review is to evaluate selected studies. In the evaluation phase, representativeness and coverage of the selected studies and the knowledge is assessed, as well as their relevance to the research question. The evaluation process starts with familiarizing oneself with the selected studies. Initially, chosen studies may be divided according to study design; for instance, are they qualitative, quantitative, or both? Studies can be evaluated by general criteria or narrower and more specific criteria. General criteria assess the study's strengths and weaknesses and the generalizability of the results. If the selected studies include a specific study design, the evaluation process can be made according to criteria that are typical to this particular approach with the help of pre-existing evaluation tools. These evaluation tools increase the quality of the review but may be challenging for the beginner. The use of evaluation tools should be considered according to available resources. (Stolt et al. 2016, p. 28-30.)

Selected studies may also be evaluated and scrutinized according to author, publisher, publishing year and country, and research method. Selected studies should be assessed by the inclusion and exclusion criteria.

(Stolt et al 2016, p. 29-30.) Due to time and labour resources, evaluation tools were not used during the narrative literature review process. Evaluation tools may be complicated for beginners, and thus, the decision not to use them was made. Studies were first evaluated according to the publishing year, place, and study method. During the abstract screening stage, sample sizes were evaluated. Study samples needed to be more than **900** so that the study was eligible for the review. As this narrative literature review was designed to serve the guideline process, sample sizes needed to be comprehensive enough that some kind of generalization of the topic could be concluded.

4.4 Analysis of the Data

In the literature review, the objective of the analysis is to form a thorough and unbiased synthesis of evidence in relation to the research question and problem (Stolt et al. 2016, 30-31). Qualitative synthesis may be conducted using a wide range of methods, such as Meta-ethnography, Grounded theory, and Thematic analyses (Barnett-Page & Thomas, 2009). Thematic analysis was chosen for this narrative literature review because it is flexible and very useful to summarize the key features of an extensive information set. Thematic analysis is a suitable method for identifying, analyzing, organizing, describing, and reporting themes that are found within data. When Thematic analyses is conducted rigorously, it provides trustworthy and insightful results. (Nowell et al., 2017.)

Data from primary sources are ordered, categorized, and summarized during the analysis stage into a unified and integrated conclusion. Extracted data is compared item by item, and similar data is organized and grouped. In the final stage of the data analysis, the author synthesizes essential elements and conclusions of each subgroup. This way, the topic will be summarized into a new integrated understanding of the phenomenon. (Stolt et al. 2016, 30-31.) Absolute honesty is vital during the process, and the researcher must be aware of one's pre-understandings, i.e., personal assumptions and knowledge, that may have an impact on results (Erlingsson & Brysiewicz, 2017).

According to Nowell et al. (2017), thematic analysis may be divided into six phases. Researchers familiarize themselves with the content during the first phase and search for meanings and patterns. (Nowell et al., 2017.) The First phase may also include the organization of the records according to essential information, for example, authors, publishing year and country, sample size, research method, and the aim of the study. Tabulated records are often informative ways for the reader to increase their understanding of the topic and results. (Stolt et al. 2016, p. 30) In this narrative literature review, selected studies were first read many times to create an understanding of the most significant findings. After the reading stage, the essential basic information of the selected studies was organized together (Table 5). Table 5 consisted of four parts: 1.

Study (title, author, publication place, and year); 2. Aim of the study; 3. Research method and sample size; 4. Key findings. The fourth part, Key findings, was developed during the analysis's last stage.

The second analysis stage aims to find similarities and differences between selected studies and then interpret findings. The results and conclusion are critical parts of the original studies. The initial coding process starts during the second phase. (Stolt et al 2016, p. 30-31.) Qualitative coding allows the researcher to simplify the information and focus on specific data characteristics. Significant sections of the text are labeled. Coding should ease organizing and interpreting the data; therefore, codes must have explicit boundaries. Proper coding eases the comparison process. (Nowell et al., 2017.) In practice, this stage is often iterative, meaning that small parts of the process are repeated several times to find the conclusion (Stolt et al. 2016, p. 30-31).

During the second stage of this narrative literature review, the focus turned more on the results and conclusions. After reading these sections several times, initial codes were made. During the whole coding process and analysis, the primary purpose of this research development was kept in mind: What could help the making of guidelines most? This had an impact on every step during the analysis process. Key findings were coded with Microsoft Edge pdf editor. Coding included five categories: **complications, risk factors, symptoms and visual function, and prognosis**. The fifth code was designed to include **other significant findings** that did not include to any other code.

The third and fourth phase of thematic analysis starts when all the selected data has been coded and organized. During the third phase, relevant codes are extracted into themes, and during the fourth phase, themes will be refined to the final format. Some of the initial codes begin to form main themes, some turn into subthemes, and some seem to belong nowhere. Themes link essential data elements together and should capture something relevant in relation to the research question. Refined themes are specific enough to be discrete but broad enough to capture ideas in several text segments. In the inductive approach of thematic analysis, the process of coding and theme-making is data-driven. This means that the researcher is not trying to fit data into a pre-existing coding frame, and the researcher's preconceptions do not play a significant role in the process. Describing the whole process of data collection, coding, organizing, and analysing is necessary so that the reader may evaluate the outcome in relation to the original data. (Nowell et al., 2017.)

This narrative literature review's third and fourth phases included theme extraction and refinement. After coding, three main themes were created: **complications of PVD, risk factors for anomalous PVD, and prognosis of anomalous PVD**. During further refinement, complications of PVD was further divided into **early-stage complications** and **complications of acute PVD**. Risk factors for anomalous PVD included both non-ocular and ocular risk factors. The prognosis of anomalous PVD included symptomatic VMA,

retinal breaks, and epiretinal membrane. Risk factors for PVD do not automatically answer either of the research questions, but knowing the risk factors is a valuable tool while assessing patients with PVD. Thus, this theme was decided to be included in this narrative literature review.

During the fifth phase, each theme is revised for the last time. The researcher should clearly define the themes and also what they are not. Investing enough time to develop the themes will increase the odds of generating meaningful findings. The sixth and final phase of the analysis begins when themes are revised and thoroughly established. The sixth phase includes the final analysis and write-up of the results. The write-up should provide concise and engaging data reportage within and across the themes. How all the findings were developed should be clearly communicated to increase the validity and reliability of the review. All the relevant results should be presented to increase the credibility. This also includes unexpected findings that do not correspond to the pre-existing assumption of the studied phenomenon. The researcher should try to confirm or reflect on the findings of previous literature. Findings may also give a chance to challenge the literature. Overall, the final phase should build a narrative about what essential things the different themes reveal about the topic. (Nowell et al., 2017.)

5 RESULTS

This narrative literature review consists of 9 studies, presented in Table 5. Selected studies included three systematic reviews (Blindbaek & Grauslund, 2015; Gishti et al., 2019; Neffendorf et al., 2017), one systematic review and meta-analysis (Garg et al., 2022), one retrospective study (Driban & Chhablani, 2022), one retrospective cohort study (Seider et al., 2022), one prospective surveillance study (Mitry et al., 2011), one cross-sectional analysis of cohort study participants (Liew et al., 2021), and one population-based longitudinal study (Yang et al., 2018). Two of the selected studies were made in the United States, and the rest were made in the United Kingdom, China, Netherlands, Denmark, Scotland, Australia, and Canada. Study participant sizes vary from 932 to 8416.

5.1 Complications of Posterior Vitreous Detachment

In four of nine studies (Driban & Chhablani, 2022; Gishti et al., 2019; Mitry et al., 2011; Seider et al., 2022), the main focus was on findings and complications of acute PVD. Two studies concentrated more on complications of early-stage PVD and the epiretinal membrane (Liew et al., 2021; Yang et al., 2018). Although epiretinal membrane might occur at any stage during the PVD process, it was grouped to belong to early-stage findings in this chapter.

Complications of acute PVD

The study by Driban et al. (2022) aimed to analyse the typical presentation of acute PVD. They conducted a retrospective analysis of medical records collected at the University of Pittsburgh Medical Center with acute PVD from January 2015 to June 2020. Overall, 4692 eyes from 2346 participants were analysed. 60.5% of the participants were females, and the average age was 62.8. 25.8% of the patients (N=605) had any ocular findings on the fundus exam, and 6.8% had bilateral findings. The main findings included pigmentation (N = 184, 7.8%), lattice degeneration (N = 158, 6.7%), tear (N = 131, 5.6%), and hole (N = 122, 5.2%). 26 patients (1.1%) had unilateral RRD. Thus, retinal breaks or RRD occurred in 11.9% of the patients. Small numbers of other retinal findings, such as chorioretinal scarring (2.9%), pavingstone degeneration (1.9%), cobblestone degeneration (1.6%), nevus (1.4%), and subretinal fluid (1.0%) were also present in study participants. (Driban & Chhablani, 2022.) Lattice degeneration is a well-known risk factor for retinal breaks and RRD, but their relations were not reported in this study. Additionally, the study patients' vitreous status (pigment or haemorrhage) was not reported.

Seider et al. (2022) aimed to evaluate the risk factors for retinal tear or RRD associated with acute, symptomatic PVD in a large comprehensive eye care setting. They conducted a retrospective cohort study of

8305 patients who presented with symptoms of acute unilateral PVD. Study participants were examined in Kaiser Permanente Northern California (KPNC). KPNC provides comprehensive eye care, including retina subspecialty services. The KPNC electronic medical record was queried to capture patients examined in 2018. 448 in 8305 (5.4%) patients were diagnosed with retinal tear, and 335 (4.0%) were diagnosed with RRD. Only a tiny proportion (2.7%) of patients developed a late retinal or RRD within the first year after the diagnosis of uncomplicated PVD. The median time for late complications was 22 days. The mean age of study participants was 63.8 years, and women were represented more than men (61.2% vs. 38.8%). In women, acute PVD peaked slightly earlier (mean age 63.1 vs. 64.7 years). Most patients experienced floaters (94.6%) and little less than half flashes (44.9%). Approximately half of the patients with an RRD reported a peripheral scotoma (51.3%). (Seider et al., 2022.)

The systematic review by Gishti et al. (2019) aimed to assess the associations between different symptoms related to PVD and the risk of developing retinal tears. They searched articles written in English within the time frame 1996–2017, using MEDLINE, Embase (via Embase.com), and the Cochrane Controlled Trials Register. 13 articles that fulfilled the selection criteria were extracted by two authors using predefined data fields, including study quality indicators. Overall, 13 studies included 2651 participants (eyes) with a mean age of 62.2 years. Patients who only reported flashes developed retinal tears in 5.3% of the cases, and patients who reported only floaters had retinal tears in 16.5%. Both symptoms were associated with retinal tears in 20% of the cases. Vitreous or retinal haemorrhage and pigment cells at the initial examination or at the follow-up period (4-8 weeks) were associated with retinal tears in roughly one-third (30%) of the patients. If both flashes and floaters were present and the initial examination had no findings of retinal breaks, these patients had retinal tears at the follow-up in 3.9% of the cases. (Gishti et al., 2019.)

The prospective surveillance study made by Mityr et al. (2011) aimed to describe RRD's predisposing pathology and clinical features. In their research, all 1202 incident cases of RRD were recruited as a part of the Scottish Retinal Detachment Study between November 1, 2007, and October 31, 2009. Clinical information was available on 1130 patients. The annual incidence of horseshoe tear (HST) related RRD was 9.45 per 100,000 people. PVD was present in 87.6% of cases (990/1130), and HST accounted for 98.5% of this group (975/990). Giant retinal tear accounted for only 1.5% of the cases (15/990). In half of the HST-related RRD cases, a single HST was responsible for subsequent RRD, and half had multiple HSTs. PVD was absent in 12.4% of patients (140/1130). These comprised round hole (RH) related RRD in 40% (56/140), retinal dialysis in 47.8% (67/140), and retinoschisis RRD in 12.1% (17/140) of the cases. RH-related RRD occurred often in young patients with high myopic correction (42.8%, <40 y.). Trauma was associated with retinal dialysis in 55.2% of the cases. Vitreous haemorrhage was reported to be present in 10 % of the HST-related RRDs. In HST-related RRDs where PVD was present, retinal breaks were most often (81.7%) in the superior half of the retina (superotemporal 56% and superonasal 25.7%). (Mityr et al., 2011.)

Complications of early-stage PVD

Liew et al. (2021) aimed to describe the prevalence, risk factors, and associations of vitreoretinal interface (VRI) abnormalities in a population-based study of older adults in Australia. They conducted a cross-sectional analysis of 1149 participants, followed for 15 years, starting from 1992. At the baseline, white persons 49 or older were included. After the follow-up period, 905 participants (1791 eyes) had gradable OCT scans from at least one eye. 451 of 905 participants (49.8%) showed VRI abnormalities. 33.6% of participants had vitreomacular adhesion (VMA), of which 22% were focal, and 78% were broad-based. Vitreomacular traction (VMT) was present in 1.6% of participants, of which 76% were focal and 24% were broad. 21.4% had epiretinal membrane (ERM), and both full-thickness macular hole (FTMH) and lamellar macular hole (LMH) were present in 0.7% of participants. FTMH was large (>400 μ m) in every observed case. The mild epiretinal membrane was present in 207 eyes and moderate to severe only in 35 eyes. Thus, the prevalence of a more severe form of ERM was 3.1%. Moderate to severe ERM reduced the BCVA for two ETDRS lines (9.2 letters) and FTMH for five lines (26 letters). Mild ERM, VMA, VMT, and LMH did not affect visual acuity. (Liew et al., 2021.)

The study by Yang et al. (2018) aimed to assess the incidence and progression of epiretinal membranes. They conducted a population-based longitudinal study of 4439 participants at the beginning of 2001. Participants were followed for ten years. Overall, fundus photographs of 2476 participants were available after the follow-up period. The age range was from 51 to 93 years, and the mean axial length was 23.3 ± 0.9 mm. ERM developed in 208 patients during the follow-up time. Thus, the cumulative incidence of ten years was 8.4%. 2.5% were preretinal macular fibrosis (PMF) and 5.9% were cellophane macular reflex (CMR). Newly developed ERMs were mainly unilateral (76% vs. 24%). Age was a significant factor for ERM development, as only 3.1% of younger than 60 developed ERM during the follow-up time, and the incidence of patients over 60 years old was at least 10.0% in every age group. The incidence peaked between 70 to 79 years (14.4%). There was no significant difference between the genders (men 7.5% and women 9.1%). (Yang et al., 2018.)

5.2 Risk Factors of Anomalous Posterior Vitreous Detachment

Risk factors for anomalous PVD findings were evaluated in seven studies. Five were more focused on the risk factors of acute-stage PVD, and two on the risk factors of early-stage PVD.

Seider et al. (2022) found several ocular and non-ocular risk factors for anomalous PVD during acute PVD. Their study grouped risk factors as variables before and during the examination. Blurred vision, male sex, age under 60-year, a family history of RD, prior keratorefractive surgery or cataract surgery, and duration of symptoms (<1 week) were all variables reported before the examination, which were associated with higher

risk of RT or RRD. Surprisingly, symptoms of flashes were said to be mildly protective against RT and RRD. Vitreous pigment, vitreous haemorrhage, lattice degeneration, and visual acuity worse than 20/40 were the main elements associated with a high risk of RT or RRD during the examination. Significantly, the presence or absence of vitreous pigment was reported to be highly predictive of acute anomalous PVD. 12.4% of patients had late RT or RRD within one year after initial presentation. The most predictive factors for late events were vitreous haemorrhage at the initial examination, lattice degeneration, and a history of RT or RRD in the fellow eye. Only 0.7% of the late complications occurred if none of these factors were present. Also, myopic refractive error was reported as a risk factor for RT or RRD, whereas hyperopic refractive error (>+1 dpt) was a protective factor. Highly myopes (>-6 dpt) had PVD on average at 58.2 years and hyperopes roughly ten years later. (Seider et al., 2022.)

Gishti et al. (2019) evaluated symptoms of acute PVD and their relation to the risk of having retinal tears. The most significant symptom-based risk factors for a retinal tear were reported to be several floaters (ten or more) and a cloud-like obscuration in the visual field. Symptoms of floaters and flashes in tandem were reported to carry a higher risk than isolated symptoms. Isolated symptoms of floaters (16.5%) had a three-fold risk of developing retinal tears compared to isolated flashes (5.3%). Patients who reported isolated, subjective blurring of vision were not at high risk of having tears, but in contrast, blurring of vision in tandem with flashes and floaters was associated with a high risk of developing retinal tears. (Gishti et al., 2019.)

Gishti et al. (2019) also demonstrated that the risk of RT persisted for at least four to eight weeks after the onset of initial symptoms. Retinal, or vitreous haemorrhage and pigment cells were the most predictive factors for RT during the eye examination, as they were associated with retinal tears in up to 30% of the cases. (Gishti et al., 2019.) Also, Blindbaek et al. (2015) highlighted in their study that retinal tears identified in the follow-up were all found in the patient with the incomplete initial examination. Furthermore, obscuring elements, such as vitreous haemorrhage, is a significant risk factor for retinal breaks at the presentation and during the following months. (Blindbaek & Grauslund, 2015.)

Mitry et al. (2011) collected thorough details of 1130 patients with RRD. PVD was present in 990 cases (87.6%). They found that myopia, pseudophakia, lattice degeneration, and other peripheral retinal degeneration were all ocular-related risk factors for PVD-associated RRD. Non-ocular risk factors included trauma (blunt or head), age, male gender, first-degree relative with a history of RRD, and syndromic disease (e.g., Marfan or Stickler's). One of five patients (20.2%) was pseudophakic; the mean time after cataract surgery to RRD was 3.28 years (1.06-7.23 years). 17 % of the pseudophakic patients also had previous YAG capsulotomy, and the mean time after YAG to RRD was two years (0.8-6.1 years). More than half of the patients with PVD-associated RRD had at least -1 dpt of myopic refractive error, and 16.8% had more than -6 dpt. Trauma was part of the RRD in every tenth case (10.2%), and in 73 patients, PVD was present (73/990). The mean time between trauma and RRD was 3.48 months (0.36-8.86 months), and lattice degeneration

was present in traumatic RRD eyes in 10.3% of the cases. In total, lattice degeneration was present roughly in every fifth (19.3%) RRD eye with associated PVD, and other peripheral degenerations were present in 10.5% of the cases. Lattice was significantly linked with high myopia, as 22.7% of the lattice eyes had at least -6 dpt. 6.8% of patients reported a first-degree relative with a history of RRD. These patients were, on average, 1.1 dpt more myopic (0.85mm in axial length) compared to the RRD population without a family history of RRD. 60% of the PVD-related RRDs occurred in the age group between 50 and 70 years old, and males were slightly more often affected (60% vs. 40%). Only a small number of the cases had the syndromic disease (8/1130), including one diagnosed with Stickler's syndrome, and in two cases, Stickler's syndrome was suspected. One of the eight cases had Marfan's syndrome with a subluxated lens. (Mitry et al., 2011.)

Driban et al. (2022) hypothesized that environmental factors, such as increased sun exposure, physical activity in warmer weather, and dehydration, might impact the nature of PVD. In their study, male patients did not show any significant seasonal tendency, but female and older patients had a slight association with PVD presentation during the spring and summer months. Females had increased rates of retinal tears and decreased rates of retinal holes compared to men. Lattice degeneration is a well-known risk factor for retinal breaks, and it was present in 6.7% of the patients, 63.3% unilateral and 36.7% bilateral. (Driban & Chhablani, 2022.) Their study did not mention the association between lattice degeneration and subsequent retinal breaks and RRD.

In the studies by Liew et al. (2021) and Yang et al. (2018), the focus was on vitreoretinal interface abnormalities of the older population. Liew et al. (2021) found that VRI abnormalities are highly related to age. VMA was found to be more prevalent with the younger generation, as one in three cases (34%) younger than 75 years old had VMA. ERM was more prevalent in patients older than 75 years, as ERM was present in 16.5% of the cases in the age group of 75 to 84 years and then remained stable. VMT was least common in the age group older than 85 years, and FTMH was slightly more common in this age group, but otherwise, VMT, FTMH, and LMH had very similar prevalence between the age groups. VMA was slightly more common in males (29.6% vs. 21.4%), but other VRI abnormalities did not differ by sex. Liew et al. (2021) also found that myopia and cataract surgery are risk factors for ERM formation. (Liew et al., 2021.) Yang et al. (2018) had similar findings about ERM. They found that the incidence of ERM increases with age, and the peak age was between 70 and 79 years. ERM was highly related to the previous cataract surgery and PVD. (Yang et al., 2018.)

5.3 Prognosis of Anomalous Posterior Vitreous Detachment

Four of nine studies evaluated the prognosis of PVD-related complications. Blindbaek et al. (2015) assess the progression of retinal breaks, Garg et al. (2022) evaluate the spontaneous release of VMT, and one part

of the study by Yang et al. (2018) was to evaluate the progression of ERM. Neffendorf et al. (2017) assessed the efficacy and safety of ocriplasmin treatment for symptomatic VMA compared to no treatment, sham, or placebo.

Retinal breaks

The systematic review by Blindbaek et al. (2015) aimed to examine the need for follow-up after PVD regarding retinal breaks. The second aim was to evaluate the indication of prophylactic treatment in asymptomatic and symptomatic breaks (either holes or tears). After four screening levels, 13 studies were identified to fill the inclusion criteria. The search included searches from PubMed and Medline and two additional manual searches. Searching was limited to include studies only in English and published before 2012. In total, 13 selected studies had 2612 eyes. The initial examination, when symptomatic acute PVD occurred, was found to reveal 85–95% of retinal breaks. Thus, 5 to 15% of the breaks were not identified; therefore, a follow-up examination is necessary, especially in the incomplete initial examination. For example, vitreous haemorrhage might be obscuring the view to the peripheral retina, and not all the breaks are not identified. They also found that asymptomatic retinal breaks progressed to RRD in 0-13.8% and symptomatic in 35 to 47% of the cases. In the cases where prophylactic treatment was executed (either laser photocoagulation or cryopexy), the cumulated incidence of RRD was between 2.1 and 8.8%. (Blindbaek & Grauslund, 2015.)

Symptomatic vitreomacular adhesion

The study by Garg et al. (2022) aimed to evaluate predictive factors of spontaneous VMT release. They conducted a systematic literature search from Ovid MEDLINE, Embase, and Cochrane Library. Overall, 12 studies and 934 eyes were included in the systematic review. Three of the 12 studies (N=104) were prospective, and nine were retrospective (N=830). The mean age of the participants was 70.0 years, 37.2% of patients were men, and the mean follow-up was 22.0 months. Four studies included only symptomatic VMT patients and the information on patients who were symptomatic or asymptomatic was unavailable in eight studies. 29% of the eyes (272/934) went through spontaneous VMT release, and 71% (662/934) had persistent VMT. The mean release time was 15.3 months and release improved BCVA on average from Snellen 20/44 to 20/32. Three predictive factors were found for spontaneous VMT release: diameter of VMT, absence of ERM, and eye laterality (right eye). A small VMT diameter was the only significant marker for spontaneous release. The mean diameter of spontaneous release was $292.3 \pm 192.9 \mu\text{m}$. (Garg et al., 2022.)

The Cochrane systematic review by Neffendorf et al. (2017) aimed to assess the efficacy and safety of ocriplasmin treatment. The comparison group included patients with no treatment, sham, or placebo. Their review included four RCT studies with a total of 932 participants. All participants were adults between the ages of 18 and 97. Symptomatic VMA was diagnosed using OCT, and BCVA was 20/25 or worse. Symptomatic VMA was defined as visual loss due to foveal damage caused by abnormal VMT. Symptomatic VMA

included isolated VMT, impending MH, and MH with persistent VMT. Neffendorf et al. (2017) found that without enzymatic vitreolysis (Ocriplasmin), roughly one in four patients (26.5%) with symptomatic VMA require vitrectomy within six months. Furthermore, without treatment, significant improvement in best-corrected visual acuity (three lines or more) occurred only in 6% of the eyes within six months, and spontaneous closure of macular hole occurred only in 12.3% of the cases within two years. Approximately one in ten patients had spontaneous release of VMA within 28 days. Ocriplasmin treatment was more efficient in every tested group, but still, up to 20% of patients required vitrectomy within six months. (Neffendorf et al., 2017.)

Epiretinal membrane

One aim of the study by Yang et al. (2018) was to evaluate the progression of epiretinal membranes. The evaluation was made according to fundus photographs during the ten-year follow-up period. Fundus photographs of 64 eyes were part of the evaluation process. Progression of ERM was defined by an enlargement of the ERM area by more than 25% or a shift from CMR to PMF. Regression was evaluated by a decrease of more than 25% in the area, by change from PMF to CMR, or by disappearance of ERM. CMR was found to remain stable (35.7%) or regress (42.9%) in most cases. Only one of five (21.4%) eyes with CMR progressed. PMF eyes showed a progression in 38.9% of the cases, 27.8% remained stable, and 33.3% regressed. Overall, approximately one-third of the eyes with ERM showed progression, one-third remained stable, and one-third regressed. (Yang et al., 2018.)

Table 5. Studies selected for the narrative literature review

No.	Study	Aim of the study	Research method and sample size	Key findings
1	Complications of Acute Posterior Vitreous Detachment Seider et al. (2022) USA	To evaluate the risk factors for retinal tear (RT) or rhegmatogenous retinal detachment (RRD) associated with acute, symptomatic posterior vitreous detachment (PVD) in a large comprehensive eye care setting.	Retrospective cohort study N= 8305	RT: 5.4% (448/8305) RRD: 4.0% (335/8305) Late RT or RRD: 2.7% (208/7522) Late RT or RRD mainly occurred if initial examination revealed vitreous haemorrhage, lattice degeneration, or other eye had a history of RT or RRD.

2	<p>Symptoms related to posterior vitreous detachment and the risk of developing retinal tears: a systematic review</p> <p>Gishti et al. (2019) Netherlands</p>	<p>To assess the associations between different symptoms related to PVD and the risk of developing retinal tears.</p>	<p>Systematic review</p> <p>N= 2651</p>	<p>Risk of having tears:</p> <p>Flashes: <u>5.3 %</u></p> <p>Floater: <u>16.5%</u></p> <p>Several floaters increase the risk.</p> <p>Floater and Flashes: <u>20%</u></p> <p>Haemorrhage and pigment cells: <u>30%</u></p>
3	<p>Prophylactic treatment of retinal breaks--a systematic review</p> <p>Blindbaek et al. (2015) Denmark</p>	<p>To examine the need of follow-up after posterior vitreous detachment (PVD)–with regard to retinal breaks as well as the indication of prophylactic treatment in asymptomatic and symptomatic breaks.</p>	<p>Systematic review</p> <p>N=13 studies, 2612 eyes</p>	<p>Asymptomatic retinal breaks progressed to RRD in <u>0-13.8%</u>, symptomatic <u>35-47%</u>. With prophylactic treatment only <u>2.1-8.8%</u>.</p> <p>Follow-up is needed if the initial examination is incomplete.</p>
4	<p>The Predisposing Pathology and Clinical Characteristics in the Scottish Retinal Detachment Study</p> <p>Mitry et al. (2011) Scotland</p>	<p>To describe the predisposing pathology and clinical features of all incident cases of rhegmatogenous retinal detachment (RRD) recruited in Scotland during a 2-year period.</p>	<p>Prospective surveillance study</p> <p>N=1130</p>	<p>PVD was present in <u>87.6%</u> of RRD cases. HST accounted for <u>98.5%</u> of PVD-related RRD cases.</p> <p>Critical predisposing factors were ocular trauma, previous cataract surgery, family history, and lattice degeneration.</p>
5	<p>Clinical findings in acute posterior vitreous detachment</p> <p>Driban et al. (2022) USA</p>	<p>To analyze the typical presentation of acute posterior vitreous detachment (PVD), including demographics and prevalence of various treatable findings in the same and fellow eye.</p>	<p>Retrospective study</p> <p>N=2346</p>	<p>After acute PVD</p> <p>RT: <u>5.6%</u></p> <p>Retinal hole: <u>5.2%</u></p> <p>RRD: <u>1.1%</u></p> <p>Prevalence of</p> <p>Lattice degeneration: <u>6.7%</u></p> <p>Retinal pigmentation:</p>

				<u>7.8%</u>
6	Prevalence of Vitreoretinal Interface Disorders in an Australian Population Liew et al. (2021) Australia	To describe the prevalence, risk factors, and associations of vitreoretinal interface (VRI) abnormalities in a population-based study of older adults.	Cross-sectional analysis of cohort study participants. N=1149	Prevalence of VMA: <u>33.6%</u> VMT: <u>1.6%</u> ERM: <u>21.4%</u> FTMH: <u>0.7%</u> LMH: <u>0.7%</u> (mean age, 76.1±6.9 years)
7	Predictive factors of spontaneous release of vitreomacular traction: A Systematic Review and Meta-Analysis Garg et al. (2022) Canada	To review predictive factors of spontaneous vitreomacular traction (VMT) release.	Systematic Review and Meta-Analysis N=934	Spontaneous release of VMT= <u>29%</u> The mean release time was 15.3 months; the mean age was 70. Predictive factors for spontaneous release: small VMT diameter (292.3 ± 192.9 µm), absence of epiretinal membrane.
8	Ocriplasmin for symptomatic vitreomacular adhesion Neffendorf et al. (2017) UK	To assess the efficacy and safety of ocriplasmin compared to no treatment, sham or placebo for the treatment of sVMA.	Systematic review N=932	Spontaneous resolve of (without treatment) SVMA= <u>9.7%</u> (≤28 days) MH= <u>12.3%</u> (1-24 months) Requirement for PPV without treatment <u>26.5%</u> (≤6 months)
9	Ten-year cumulative incidence of epiretinal membranes assessed on fundus photographs. The Beijing Eye Study 2001/2011 Yang et al. (2018) China	To assess the 10-year incidence and progression of epiretinal membranes (ERMs).	Population-based longitudinal study N=4060	Cumulative (10 years) incidence of ERM= <u>8.4%</u> PMF= <u>2.5%</u> CMR= <u>5.9%</u> CMR remained stable at <u>35.7%</u> , regressed <u>42.9%</u> , and progressed <u>21.4%</u> . PMF remained stable at <u>27.8%</u> , regressed <u>33.3%</u> , and progressed <u>38.9%</u> .

5.4 Synthesis

The literature review aimed to answer the research questions about complications of PVD and the prognosis of anomalous PVD. Research questions were developed to support the process of creating Finnish guidelines. In four of nine studies, the main focus was on findings and complications of acute PVD. Two studies concentrated more on complications of early-stage PVD and epiretinal membrane. Four of nine studies evaluated the prognosis of PVD-related complications. Additionally, seven of nine studies covered risk factors of anomalous acute PVD or early-stage PVD. Risk factors were divided into ocular and non-ocular risk factors. Especially during acute PVD, knowledge of risk factors is essential while assessing the need for follow-up examination. Therefore, the theme of risk factors was considered necessary in relation to the overall research development, even if it did not directly answer either of the research questions.

Acute Posterior Vitreous Detachment

In the studies by Driban et al. (2022) and Seider et al. (2022), the focus was on the findings of patients who presented to eyecare practise with symptoms (flashes and floaters) of acute PVD. In both studies, the rate of retinal tears was similar (5.6% vs. 5.4%), but the study by Seider et al. (2022) had a significantly higher number of RRD (4.0% vs. 1.1%) at the initial examination. Additionally, Driban et al. (2022) reported that 5.2% of the patients had retinal holes. In general, PVD-associated retinal holes do not carry a significant risk of RRD. Still, the number of holes increases the prevalence of overall retinal breaks to 10.8%, and in tandem with RRD, complications were 11.9% in the study by Driban et al. (2022). The overall prevalence of complications in the study by Seider et al. (2022) was slightly less, 9.4%. Seider et al. (2022) reported time for late complications; the mean time was 22 days. Late retinal tears, or RRD, occurred only in 1.8% of the patients. These results suggest that the overall number of retinal breaks and RRD associated with PVD may be less than the previous literature has assumed. In the previous Case series and Meta-analysis made by Coffee et al. (2007), the rate of retinal tears was discovered to be 21.7% (8.2% to 47.6%), which is significantly higher compared to the rate in the studies by Seider et al. (2022) and Driban et al. (2022). In turn, the prevalence of late complications in the same survey was the same as in the study by Seider et al. (2022). This finding supports the current best practice that only a minority of the patients with acute PVD are at risk of developing complications during the following weeks and months after the initial presentation.

Mitry et al. (2011) found that PVD is associated with RRD in almost nine out of ten cases (87.6%), and the causative reason for RRD was most often PVD-related HST, as it counted in 98.5% of the RRDs. Only 1.5% of the RRDs started from PVD-related giant retinal tears. In their study, round hole related RRDs occurred in young myopic patients, and these cases were not associated with PVD. According to Mitry et al. (2011), these findings were not in line with previous literature, as the proportion of PVD-related RRD was much higher in their study. Several clinical trials that were made during the '70s, the '80s, and the '90s revealed that HST was a causative reason for RRD in 50% to 61% of cases, and an atrophic round hole was causative

in 12% to 21% of RRD cases. In contrast, in the study by Ferrera et al. (2023), PVD was present in 9 of 10 RRD cases (89.2%), and HST was the causative reason for RRD in 82% of the cases. According to these results, the amount of PVD-related RRD is likely higher than previously reported.

Risk factors of acute anomalous PVD, either ocular or non-ocular, were covered in five studies. Acute PVD is related to the cardinal symptoms of flashes and floaters. Surprisingly, Gishti et al. (2019) and Seider et al. (2022) found that isolated flashes are not often related to complicated PVD. Seider et al. (2022) even stated that flashes may be a protective factor for retinal tears. Flashes are signs that the vitreous is pulling the sensory retina; according to the author's knowledge, many ophthalmologists are concerned with these symptoms. Nevertheless, Seider et al. (2022) and Gishti et al. (2019) have not been the only researchers with such findings. Also, in the previous study by Bond-Taylor et al. (2017), symptoms of flashes were not found to increase the risk of retinal tears. In contrast, Gishti et al. (2019) found that floaters' symptoms significantly increase the risk of retinal tears compared to the risk of flashes (16.5% vs. 5.3%). In their study, the increasing number of floaters was found to carry a higher risk of having tears than just a single floater. Ten or more floaters and/or cloud-like obscuration in the visual field were found to carry the highest risk for tears. Seider et al. (2022) had similar findings about cloud-like obscuration, as in their study, approximately half of the patients who complained of a peripheral scotoma were diagnosed with RRD at the initial examination. Interestingly, Seider et al. (2022) and Gishti et al. (2019) had different findings about the blurring of vision. Seider et al. (2022) reported that subjective blurring of vision was associated with complicated PVD, but Gishti et al. (2019) reported that blurry vision did not increase the risk significantly.

It has been well stated in previous literature that myopia, lattice degeneration, previous cataract surgery, and family history of RD are all significant predisposing factors for developing complicated acute PVD. These statements were supported in the studies by Seider et al. (2022) and Mitry et al. (2011). In Mitry's study, more than half of RRD cases had a myopic refractive error (>-1 dpt), every fifth had undergone cataract surgery, roughly every fifth (18.7%) had lattice degeneration, and 7% of RRD cases had a first-degree relative with the history of RRD. Also, male gender was demonstrated to be a risk factor in both studies. Head or ocular trauma was present in every tenth case in the study by Mitry et al. (2011). Although hereditary collagen-vascular disorders, such as Stickler and Marfan, are mentioned to be risk factors in the literature (see Ch. 2.3), these syndromic factors were present only in 0.7% (8/1130) of the cases in the study by Mitry et al. (2011). One interesting note from Mitry's study is that RRD patients with a family history of RRD were significantly more myopic than RRD patients without a family history. This result could suggest that maybe myopia, especially high myopia itself, is a more significant factor for predicting RRD than the family history of RRD. Although the risk related to the male gender was covered in two studies, in the study by Drihan et al. (2022), women had more retinal tears, and men had more retinal holes. If considering previous literature, this finding is controversial. According to Steel (2014), symptomatic retinal tears have a high rate of progression to retinal detachment ($>50\%$), especially if the vitreoretinal traction is present. Retinal holes are not

associated with persistent vitreous traction; thus, the risk of vitreous fluid entering the subretinal space is lower. Mitry et al. (2011) state that RH-related RRDs often occur without PVD. Patients are younger (<40 years) and more myopic than those with PVD-related RRDs.

Risk factors mentioned during the eye examination included pigment in the vitreous, vitreous, or retinal haemorrhage, and significantly reduced visual acuity (<20/40). Blindbaek et al. (2015), Seider et al. (2022), and Gishti et al. (2019) all found that vitreous haemorrhage at the initial examination carries a high risk for complicated PVD. Although, sometimes vitreous haemorrhage can also result from small retinal vessel damage. In the study by Mitry et al. (2011), one in ten cases of PVD-associated RRD had a vitreous haemorrhage. In the study by Gishti et al. (2019), vitreous and/or retinal haemorrhage was found to carry the second highest risk of complicated PVD after multiple floaters and cloud-like obscuration. Blindbaek et al. (2015) found that the retinal tears identified at follow-up were all found in cases where a full retinal examination was impossible due to obscuring elements, such as vitreous haemorrhage. The presence of vitreous pigment was covered in two studies, Seider et al. (2022) and Gishti et al. (2019). Seider et al. (2022) demonstrated that the presence or absence of vitreous pigment is highly predictive of the existence of complicated PVD. Gishti et al. (2019) stated that the vitreous pigment appears almost immediately after retinal break formation. Therefore, it's a good predictor for a complicated PVD at the presentation or follow-up examination. Pigment, along with vitreous haemorrhage, may also be part of the floater phenomenon. In Seider's study, significantly reduced visual acuity was mentioned to be a predictive factor for complicated PVD, but this finding was not covered in any other research. Their study did not state if the visual acuity was lowered due to significant vitreous haemorrhage or macula off RRD, which could reduce visual acuity significantly.

The prognosis of complicated PVD has not been studied or covered in many previous studies. One reason for this may be ethical. It would be unethical to follow some cases which have a high rate of progression to the worst outcome. On the other hand, it is crucial to understand the prognosis behind each finding of complicated PVD for optometrists who are not allowed to treat patients. Blindbaek et al. (2015) covered 13 studies and 2612 eyes in their Systematic Review (SR). They found that initial examination reveals 85-95% of retinal breaks, and follow-up examination is critical, especially if the initial examination has been incomplete due to obscuring elements. They also found that asymptomatic retinal breaks do not often progress into RRD (0-13.8%). In the study by Byer (1982), which was included in their SR, none of the 231 eyes with asymptomatic retinal breaks led to RRD during a one-year follow-up period. Eyes were all phakic, and both non-fellow eyes and eyes with retinal detachment were included. (Byer, 1982.) According to Blindbaek et al. (2015), a story is entirely different with symptomatic retinal breaks, as they were found to progress into RRD in 35 to 47% of the cases. According to Steel (2014), more than half of the retinal tear cases with persistent vitreoretinal traction end up having RRD. Therefore, especially symptomatic retinal tears need prophylactic treatment. As mentioned earlier, horseshoe tears are responsible for RRDs in up to 98.5% of PVD-associated cases. Prophylactic treatment with either laser photocoagulation or cryopexy has significantly

decreased the incidence of RRD. The incidence of RRD decreases with prophylactic treatment from 35-47% to 2.1-8.8%; thus, prophylactic treatment should be considered with symptomatic retinal breaks. In contrast, even with prophylactic treatment, the incidence of RRD is not much lower than untreated asymptomatic retinal breaks (0-13.8% vs. 2.1-8.8%). Therefore, no definite conclusion has been made regarding the prophylactic treatment of asymptomatic retinal breaks. According to the author's knowledge, asymptomatic retinal breaks are often treated depending on the resources at hand in Finland. Also, the follow-up of asymptomatic retinal breaks needs resources, and if prophylactic treatment reduces the possibility of RRD even slightly, the treatment decision may be justified.

Early-Stage Posterior Vitreous Detachment

Two of nine studies concentrated on complications of early-stage PVD and epiretinal membrane. In the study by Liew et al. (2021), almost half of the participants (49.8%) showed some vitreoretinal interface abnormalities. The mean age of the participants was 76.1±6.9 years. One-third of the (33.6%) participants had VMA, of which 22% were focal and 78% were broad-based. VMT was present in 1.6% of participants, in which, 76% were focal, and 24% were broad. 21.4% of the participants had ERM. The prevalence of mild form was 18.3% and moderate to severe form 3.1%; only 5.4% of the participants had bilateral ERM findings. Full-thickness macular hole (FTMH) and lamellar macular hole (LMH) were present in 0.7% of participants. Some of these findings are in line with the previous study by Meuer et al. (2015). This study's age profile was similar (63–102 years). Meuer et al. (2015) found a similar prevalence of VMT (1.6%) and just a slightly lower prevalence of VMA (26.1%). One-third of the participants (34.1%) had ERM, whereas, in the study by Liew et al. (2021), only one-fourth had ERM. Meuer et al. (2015) also found the prevalence of LMH to be higher (3.6%), but in contrast, the prevalence of FTMH was lower (0.4%). In the study by Quinn et al. (2020), the participants were younger (mean age 62 years), and the prevalence of VRI findings was much lower. The prevalence of VMA was 22.6%, VMT 0.5%, MH 0.3% and ERM 7.6%. These findings suggest that VRI abnormalities are highly age dependent. A relatively low number of people under 60 have VRI findings, but the prevalence of VRI abnormalities is much higher with people closer to or over 70 years of age. It is essential to understand that even if half of the older patients might have some VRI findings, only a tiny proportion experience a reduced visual acuity. Furthermore, OCT imaging has increased the understanding of PVD-related VRI abnormalities, but the severity of OCT findings does not always correlate with patient's complaints and visual acuity.

Risk factors for VRI abnormalities were covered in two of nine studies. Yang et al. (2018) concentrated on ERM, and the study by Liew et al. (2021) covered VMA, VMT, ERM, FTMH, and LMH. Both studies had similar findings about ERM, as the prevalence of ERM increased with older age. Yang et al. (2018) found that the peak age of ERM was between 70 and 79 years, and ERM was relatively rare in patients younger than 60 years. In the study by Liew et al. (2021), ERM peaked slightly later (75 to 84 years), and then the incidence remained stable. This finding is consistent with the current knowledge that PVD is the most

apparent reason for developing idiopathic ERM (Jonhson 2010). ERM may occur during any stage of PVD, and after developing, approximately one in three cases regress or disappear. Therefore, the prevalence of ERM progresses as the population gets older. Furthermore, both studies found that a higher incidence of ERM was also related to previous cataract surgery and complete PVD. Liew et al. (2021) also reported that myopia was associated with ERM and a lower VMA prevalence. Previous literature has reported that myopia exacerbates the onset of PVD by one year of a diopter of myopic refractive error (Yonemoto *et al.*, 1994). With older age, this leads to a higher prevalence of ERM and lower levels of VMA. Liew et al. (2021) also found that male sex was associated with a higher prevalence of VMA. This finding was supported by the previous study of Shao et al. (2013). According to Tozer et al. (2014), lower levels of hyaluronic acid in the vitreous may be why women acquire earlier PVD. Therefore, it makes sense that older male patients have more VMA findings than older female patients.

The prognosis of early-stage anomalous PVD was covered in three of nine studies. Garg et al. (2022) covered the spontaneous release of VMT, Yang et al. (2018) covered the prognosis of ERM, and the complete release of symptomatic VMA was covered in the study by Neffendorf et al. (2017). According to Johnson (2010), early-stage PVD is usually chronic and occult and progresses slowly over months or even years until complete vitreopapillary separation. This hypothesis was supported in the study by Garg et al. (2022, as they found that one in three patients underwent spontaneous release of VMT, and the mean time for this was 15.3 months. Release is more likely to occur if the VMT diameter is small and there is no ERM present. Neffendorf et al. (2017) found that spontaneous release of symptomatic VMA occurs only in one of ten cases during the first month (28 days). These findings suggest that observation may be the best choice for patients with moderate and tolerable symptoms because pars plana vitrectomy is neither risk-free. Release of VMT may occur after a long period. One part of the study by Yang et al. (2018) was to observe the prognosis of ERM. They found that approximately one-third of the eyes with ERM showed progression, one-third remained stable, and one-third regressed. These findings are very much in line with a previous study by Fraser-Bell et al. (2003). Fraser-Bell et al. (2003) found that existing epiretinal membranes progressed, regressed, or remained stable during the five years in 28.6%, 25.7%, and 38.8% of eyes. Cellophane maculopathy progressed to a more severe pucker in 9.3% of the cases, approximately half compared to the finding (23.4%) in the study by Yang et al. (2018). Fraser-Bell et al. (2003) had many more participants, and therefore, the progression rate of cellophane maculopathy to PMF is probably way less than one in five cases.

Conclusion

In conclusion, posterior vitreous detachment can lead to severe deterioration of vision during the early-stage or in acute PVD. The most emergency pathological condition related to PVD is RRD, which is associated with PVD in almost 90% of the cases. The progress of RRD starts from the retinal break and, most often, from horseshoe tears (up to 98.5%). The annual incidence of HST-related RRD is 9.45 per 100,000 people. Retinal breaks occur between 5-10% of the cases with symptomatic acute PVD. Some amount of RRD (1-

4%) might be present already in the initial examination during the acute stage. Several risk factors are associated with PVD-related RRD. The most important risk factors include myopia, lattice degeneration, previous cataract surgery, YAG capsulotomy, first-degree relative with RRD history, trauma (blunt or head), age (50-70 years), and male gender. The most significant symptoms are floaters and a cloud-like obscuration in the visual field. Fewer floaters carry a lower risk of retinal breaks and vice versa. Flashes do not significantly increase the risk of having retinal breaks, and isolated flashes rarely cause harm. The risk during the acute PVD lasts at least 4-8 weeks after the onset of symptoms. Late retinal breaks occur in up to a few percent of the cases, and the most predictive factors for late events are vitreous haemorrhage or pigment granules at the initial examination, lattice degeneration, and a history of RT or RRD in the fellow eye. If none of these risks is present, the risk is very low (0.7%), and a follow-up examination may not be necessary. Early-stage PVD may lead to symptomatic VMA, which includes several pathological conditions (VMT, MH+VMT, LMH). These conditions may have mild symptoms or lead to significantly deteriorated vision. Spontaneous release of VMT occurs in up to one in three cases, but the mean release time is more than 15 months, and only one in ten eyes with symptomatic VMA go through spontaneous release within one month. Ocriplasmin eases the release process, but 20% of the eyes still need pars plana vitrectomy. Epiretinal membrane is relatively common in older populations and may occur during any stage of PVD. Prevalence peaks at the age of 70 years and then remains stable. Up to 20% of the population older than 70 have some form of ERM. A minority of the ERM cases have severe PMF, which may cause significant vision problems and need vitrectomy. ERM is most often stable or regress. CMR progresses to PMF in up to one in five eyes, and PMF shows progression in one in three eyes.

6 DEVELOPMENT PHASE

The development phase of this Thesis included making clinical guidelines for Finnish optometrists on how to assess and manage patients with posterior vitreous detachment (PVD). The guideline is divided into three sections: **patient history/anamnesis**, **ocular examination**, and **patient education**. The theoretical background covers managing and treating PVD complications; therefore, the guideline does not include these sections. The purpose of the guideline is to create a uniform procedure for Finnish optometrists. The guideline is based on the theoretical background, literature review, and existing valid international guidelines. Theoretical background includes essential information on PVD and gives optometrists a comprehensive baseline for clinical work. The theoretical background explains some procedures more thoroughly; therefore, the guideline does not include a step-by-step technical approach to every procedure. In addition, the experience as an optometrist and the author's knowledge of the facilities that are often available in Finnish optometric settings were used while writing this clinical guideline.

At the time of this thesis, the Finnish Current Care Guideline (Käypähoito suositus) does not include procedures to assess and manage PVD or its complications. Therefore, only international evidence-based guidelines were used to create the Finnish guideline for optometrists. Legislations that automatically influence the work of Finnish optometrists were also noted when selecting the clinical procedures for the guideline. Guidelines must comply with valid legislation; therefore, cooperation (either consultation or referral) with an ophthalmologist is mandatory for patients with current or suspected ocular disease.

Clinical assessment procedures were selected from four evidence-based international guidelines:

- American Optometric Association (AOA), Care of the Patient with Retinal Detachment And Related Peripheral Vitreoretinal Disease 2004
- American Academy of Ophthalmology (AAO), Posterior Vitreous Detachment, Retinal Breaks, and Lattice Degeneration Preferred Practice Pattern 2020
- American Academy of Ophthalmology (AAO), Idiopathic Epiretinal Membrane and Vitreomacular Traction Preferred Practice Pattern 2020
- The College of Optometrists (The professional body of optometry in the United Kingdom), Vitreomacular Traction and Macular Hole Clinical Management Guideline 2022

At the time of this Thesis, there is no practical way to prevent the physiological elements that lead to PVD. Although PVD is often harmless, it may have devastating consequences. Therefore, all the effort in optometric settings should be allocated to find the anomalous features of PVD. Anomalous PVD may lead to

permanent visual deterioration or even vision loss. Proper patient care aims to prevent patients' functional impairment and maintain good quality of life. Due to legislation and the rights of the Finnish optometrists, it can be assumed that most of the cases with symptomatic PVD in optometric practise will be patients with otherwise healthy eyes. Therefore, this guideline does not consider anomalous PVD, which may be caused by other eye diseases or eye surgery. The only exception is cataract surgery because it is a common procedure, and depending on local policies, some optometrists may often encounter patients with previous cataract surgery.

By this guideline, optometrists are able to:

- To recognise different stages of PVD and the possible complications it may lead to.
- To know which patients, need to be referred to the ophthalmologist.
- To educate the patient about the visual changes and possible complications due to different stages of PVD.

Optometrists should always understand their capabilities and limitations. Therefore, referral to college or an ophthalmologist with expertise and experience in assessing the condition is recommended.

6.1 Patient History / Anamnesis

Anamnesis and patient history are essential parts of the PVD protocols. Anamnesis should be done according to Andersson's new Patient History Questionnaire (Andersson, 2022). In addition, the following elements should have particular attention:

- age (>65 years)
- refractive history
 - myopia
- history of previous eye surgery (particularly lens exchange or cataract surgery)
- previous retinal detachment
- family history of retinal detachment
- previous ocular trauma
- genetic disorders (e.g., Marfan or Stickler syndrome)
- vision problems/symptoms
 - Do you see flashing lights?
 - isolated or concurrently with floaters?
 - monocular or binocular?

- Do you see floaters?
 - single or multiple?
 - in which pattern do they appear?
- Have you noticed a loss of vision or decreased visual acuity?
- Have you noticed a loss of visual field or a shadow?
- Have you noticed metamorphopsia, micropsia, macropsia, or diplopia?

Keynotes: The hallmark features of acute PVD are floaters and flashing lights. Isolated flashing lights or a single floater do not carry as high risk for anomalous PVD as multiple floaters or concurrent symptoms. Early-stage PVD with vitreomacular traction (VMT) may cause metamorphopsia or reduce VA. Epiretinal membrane (ERM) may reduce VA or cause metamorphopsia and even diplopia. The prevalence of PVD increases with age. More than half of nontraumatic RRDs occur in myopic eyes. Particularly, high myopes (>-6 dpt) are at risk for retinal tears during PVD. Myopia and cataract, or lens exchange surgery, speed up the PVD developing process and increase the complication risk.

6.2 Ocular Examination

No symptoms can reliably distinguish a normal PVD from an anomalous PVD; therefore, a comprehensive adult eye examination is mandatory. Comprehensive adult eye examination should be done according to the Finnish Guideline for the optometrist's good examination practice, Hyvä optometristin tutkimuskäytäntöohjeistus. Depending on the symptoms and individual risk factors, an eye examination should be done within one week or earlier. The list of ocular examination is not all-inclusive, and different parts should be emphasized on a case-by-case basis. The following elements should have particular attention:

- best corrected visual acuity for both eyes, distance, and near
- pupillary responses
- confrontation visual field examination (to rule out retinal detachment)
- IOP measurement
- Amsler grid
- biomicroscopy
 - assessment of the anterior chamber
 - inflammatory cells
 - assessment of vitreous with dilated pupils
 - pigment or haemorrhage (anterior vitreous)
 - Weiss ring
 - PVD: collapse – without collapse – partial PVD – no PVD

- assessment of macula with dilated pupils
 - ERM: cellophane maculopathy – preretinal macular fibrosis. If preretinal macular fibrosis is present, consult an ophthalmologist.
 - VMT: loss of foveal depression and yellowish foveal spot or ring (100-350 μm). Use Watzke–Allen to rule out macular hole. If VMT is without a macular hole, consult an ophthalmologist.
 - MH: small– medium – large. If the macular hole is present, urgent refer to an ophthalmologist.
- assessment of peripheral fundus with fully dilated pupils (3-mirror contact lens)
 - retinal tears (urgent refer to an ophthalmologist)
 - ◆ shape and size
 - ◆ operculated or tractional
 - ◆ subretinal fluid
 - peripheral retinal holes (consult an ophthalmologist)
 - ◆ atrophic and/or pigmented
 - ◆ operculum
 - retinal detachment (OCULAR EMERGENCY, immediately refer to an emergency unit)
 - peripheral retinal degeneration (patient education)
 - ◆ lattice
 - ◆ snailtrack
 - ◆ chorioretinal atrophy
 - ◆ other degeneration
- drawing of peripheral findings (or photo documentation)
- if any other eye disease is suspected during the examination, refer the patient to the ophthalmologist

Keynotes: Many patients with retinal tears have blood and/or pigmented cells in the anterior vitreous, which is an essential part of the examination. Approximately half of the retinal tears develop into retinal detachment without treatment. Retinal detachment, if not just localized, is always an ocular emergency, and patients need immediate treatment. RAPD may be present in massive retinal detachment. Retinal detachment may also cause visual field loss, iritis, and lower the IOP. Peripheral retinal holes do not carry a high risk of developing into retinal detachment but should be co-managed with an ophthalmologist. Peripheral retinal degenerations are common findings in adult patients. Particularly, lattice degeneration in myopic eyes increases the risk of PVD-related retinal tears. Early-stage PVD may cause VMT and subsequent macular hole. VMT commonly causes slight symptoms or may be symptomatic. A macular hole decreases the VA significantly and rarely gets better without treatment. Idiopathic ERM may occur in either a mild form

(cellophane maculopathy) or a more severe fibrotic form (preretinal macular fibrosis), which might need surgical intervention if the patient has significant visual complaints.

The examiner should always consider other causes of vitreous cells or debris, such as uveitis, infection, inflammation, or neoplasia. ERM may also be caused by various reasons, such as retinal vascular occlusions, diabetic retinopathy, intraocular tumours, or inflammatory mediators; therefore, secondary causes must also be considered.

OCT examination is not mandatory, but it is a valuable tool to provide information on the size of a macular hole and the presence of VMT and epiretinal membrane. An optometrist should consider referring a patient to a colleague with the equipment in case of hesitation. OCT helps a cooperation with an ophthalmologist. OCT also gives information on the stages of PVD, which may be impossible to recognize with a slit-lamp examination before the complete PVD occurs. OCT imaging may as well be used for patient education.

6.3 Patient Education

During the initial examination, all the patients should be educated about the severity of acute PVD and the symptoms of retinal detachment and retinal tears. Prompt reaction to the symptoms increases the possibility of successful retinal reattachment surgery. Particular attention to education should be remembered with patients of any risk factors, such as high myopia or a family history of RRD. It is recommended to have a follow-up examination **within four to six weeks** after the initial examination. Especially patients with particular risk factors, incomplete initial examination, or either pigment or haemorrhage in the vitreous, are prone to develop late retinal breaks. If none of these factors is present, the risk of having late breaks is relatively small, and there may be no need for a follow-up examination. All the patients should be advised to return immediately to further evaluation if they experience the following:

- increase in floaters
- increase in flashing lights
- reduced visual acuity
- curtain or shadow in the visual field
- sudden onset of tiny black specks

Patients with early-stage PVD or epiretinal membrane should be encouraged to regular monocular **Amsler grid** testing for increased symptoms (metamorphopsia, central scotoma, reduction in VA). If the symptoms get worse and patients cannot cope with their daily requirements, they need to be referred to an ophthalmologist for further consideration of treatment.

7 DISCUSSION

The first aim of the Thesis was to create a clinical guideline for Finnish optometrists. It is an essential part of the care process to understand all the stages of PVD, and therefore, this guideline includes both early-stage PVD and acute PVD. To the author's knowledge, the PVD guideline, which includes all stages, has not been made before. Naturally, the fact that Finnish optometrists cannot treat the complications of PVD due to legislation enabled this guideline without too extensive content.

In acute symptomatic PVD, 80-90 % of the cases do not need more than observation. Optometrists can ease the workload and release the labour input of ophthalmologists to more urgent eyecare. Optometrists, with their clinical knowledge and skills, can also be very helpful in managing or co-managing cases of VMT and ERM with ophthalmologists before there is a need to treat these conditions. Only a small proportion of macular holes recover without treatment; therefore, an urgent referral to an ophthalmologist is always needed. Treatment protocols and routines of retinal breaks vary depending on the resources. Finnish optometrists cannot decide which kind of breaks to treat and observe; thus, also all the operculated findings should be referred for further evaluation.

New responsibilities also bring new requirements. The optometrist should continually develop their clinical skills and understand their limitations for patient safety. Patients should be referred to another colleague or an ophthalmologist if limitations are exceeded. During acute PVD, particular attention must be paid to those with pigment or blood cells in the anterior vitreous. An examination of the anterior vitreous does not need more than basic slit-lamp microscopy skills, which all optometrists can do. Finding pigment or haemorrhage could be a minimum requirement with proper anamnesis (risk factors and symptoms) and patient education. By managing this combination, many harmless cases could be assorted from those in danger of developing retinal tears and detachment. It is also essential to understand that even with proper examination, some retinal breaks may be missed or have not been developed yet. Therefore, patient education plays a vital role in the care process of acute PVD.

How can organisations support the work of optometrists? Time resources given to optometrists in many organisations need to be rethought. Acute PVD needs peripheral retinal examination with fully dilated eyes, which may be impossible for most optometrists to organize in the current workflow. Time allocation should support the comprehensive optometric eye evaluation so that all the required examinations can be conducted appropriately. Facilities for thorough eye examination and PVD evaluation already exist in optical stores. Binocular indirect ophthalmoscopy with scleral indentation is often kept a gold standard peripheral retinal examination method. Still, a 3-mirror contact lens is much more suitable for Finnish optical store

settings, and therefore, it is the recommended method. Also, the amount of OCT equipment in Finland is rising. OCT eases the recognition and management of patients with early-stage PVD or epiretinal membrane. Furthermore, with the help of OCT images, it is easy to consult ophthalmologists.

At the time of this thesis, social and healthcare and many other working areas are struggling with labour resources. Optometrists with clinical education could ease the patient load in eye care settings. The help of optometrists could be allocated more thoroughly to identify patients who need treatment or medication from those who can just be observed. Following the guideline, optometrists will have a uniform way of evaluating PVD. This leads to a better level of patient care throughout the field of Finnish optometry

7.1 Strengths and Limitations

This research development also had some inevitable limitations. First, the initial idea of this thesis was planned for more than a year. During the writing process of theoretical background, instructors were replaced by the school, and the original plans were not eligible anymore. One more part was added to the research development. This reorganisation was labour-intensive and frustrating. Second, this research development was done alone. The narrative literature review would have needed another author (Polit & Beck, 2017). Appropriate key search terms for looking the proper studies alleviate the searching process, but another pair of eyes would have increased the trustworthiness while looking for suitable study options. Third, the selection of the studies could have been done more systematically and using evaluation tools to justify the reliability of the chosen studies. Additionally, the literature search was made only from databases. A grey literature could have revealed some hidden evidence (Paez, 2017). Still, it is doubtful in the case of posterior vitreous detachment; therefore, the decision was made not to seek unpublished sources.

A narrative literature review may be conducted without strict rules; therefore, it has been criticised for lagging and contradicting the available evidence. If a narrative literature review is conducted unsystematically, it is more likely to be ruled by the author's decisions. (Montori et al., 2003.) For the trustworthiness of the thesis, possible issues of the chosen method were kept in mind during the whole writing process. The search process was conducted with an Oulu University of Applied Sciences information specialist. All the key search terms, used databases, and inclusion and exclusion criteria were reported appropriately to increase the trustworthiness. In addition, the expertise and guidance of the instructors were also an essential part of overall trustworthiness.

Although some minor issues may be identified with the narrative literature review, the same problems do not concern the development phase of clinical guidelines. The development phase of the thesis was not only conducted due to the results of the narrative literature review but also included evidence from international

guidelines. The chosen literature for international guidelines has undergone a rigorous evaluation process and will also increase the reliability of Finnish clinical guidelines.

7.2 Ethicality

This Master`s thesis complies with ethical norms by the Finnish Advisory Board on Research Integrity. According to TENK (2012), "In order for research to be ethically acceptable and reliable and for its results to be credible, the research must be conducted according to the responsible conduct of research." (Finnish Advisory Board on Research Integrity, 2012.) This sentence was kept in mind and complied with during the research development process.

The Thesis was conducted and reported according to the principles of Oulu University of Applied Sciences. This research development did not include any participants as objects of study or pharmaceuticals; therefore, special research permits and agreements were unnecessary in this Thesis.

Furthermore, all the references were made carefully to give full respect and credit to other researchers' work (TENK, 2012). Some of the materials used in the theoretical background were relatively old. Hence, the original sources were searched whenever it was reasonable to achieve. Appropriate permission was asked for the figures used in the theoretical background. For five of seven figures, publishers responded to the requests, and permission was given; for one figure, the publisher and author did not respond to requests. That figure was decided to be used by the policies of Oulu University of Applied Sciences. One figure was published in an open-access public article, so reuse without special permission was allowed.

The author had no conflicts of interest or other commitments that would have been relevant to research (TENK, 2012). The Finnish Ethical Board of Optometry (OEN) ordered guideline for assessing the posterior vitreous detachment from the Oulu University of Applied Sciences because it has the will to create a uniform procedure across the Finnish optometrists. This subject seemed one of the most exciting guidelines to be made, and therefore, it was chosen by the author.

To create this research development, a scholarship of 1500 euros was applied and received from the foundation of Bror Biese. The scholarship was used for daily expenses.

7.3 Conclusion

As large age groups of the Finnish population are getting older, the prevalence of age-related eye illnesses is increasing rapidly. This sets a significant challenge to the Finnish social and healthcare system. Finnish

optometrists are not allowed to treat patients with eye illnesses, but they can help to screen patients who need further evaluation. PVD is part of the natural aging process of the eye and is often a harmless phenomenon; therefore, the screening is enough for most patients. Overall, a proper allocation of optometrists' resources will enable the Finnish eye care system to focus the effort of ophthalmologists on those who really need it. Creating a guideline for Finnish optometrists on how to assess PVD appropriately is the first step to starting this process.

Finnish optometrists have all the basic elements to perform a comprehensive adult eye examination. However, the level of optometrists varies significantly in the field, and maybe not all are interested in comprehensive eye examinations with such dedication as desired. New responsibilities also mean a great requirement to educate oneself further and use the learned skills. That is the only way Finnish optometrists can get through a changing working environment.

As a sequel to this thesis, a pilot project on how well Finnish optometrists can accomplish all the necessary examinations for comprehensive PVD evaluation is recommended. A similar study has been made in the UK, and British colleagues did very well in that survey. Even though the skills of Finnish optometrists are variable, the author believes that Finnish optometrists could also cope well with similar challenges.

ABBREVIATION LIST

PVD:	Posterior vitreous detachment
APVD:	Anomalous posterior vitreous detachment
VMT:	Vitreomacular traction
VMA:	Vitreomacular adhesion
SVMA:	Symptomatic vitreomacular adhesion
RRD:	Rhegmatogenous retinal detachment
RT:	Retinal tear
HST:	Horseshoe tear
RH:	Round hole
ILM:	Internal limiting membrane
ERM:	Epiretinal membrane
MH:	Macular hole
FTHM:	Full-thickness macular hole
LMH:	Lamellar macular hole
RAPD:	Relative afferent pupillary defect
VA:	Visual acuity
BCVA:	Best corrected visual acuity
PMF:	Preretinal macular fibrosis
CMR:	Cellophane macular reflex
URGENT:	Within one week
EMERGENCY:	Within 24 hours

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