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PRACA POGLĄDOWA
REVIEW

Understanding vitreomacular traction: Basic concepts and clinical implications

Zrozumienie trakcji szklistkowo-plamkowej – podstawowe pojęcia i znaczenie kliniczne

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ABSTRACT

The first references to vitreomacular traction (VMT) date back to 1953, when Samuel Rodman Irvine identified it as a potential cause of cystoid macular edema. Recently, the literature increasingly reports associations between VMT and other common ocular conditions, such as diabetic retinopathy and age-related macular degeneration (AMD). This review article explores the epidemiology, risk factors, pathophysiology, diagnostics, and treatment options for VMT. The mechanism of VMT development in the course of diabetic retinopathy differs slightly depending on whether it is proliferative or non-proliferative diabetic retinopathy. Exudative AMD is characterized by the development of macular neovascularization, which can lead to fluid leakage or hemorrhage in the macula. Recent studies also suggest a potential link between AMD and VMT. VMT and other vitreoretinal interface disorders represent a group of conditions with diverse underlying mechanisms and associated complications. Choosing an effective treatment strategy requires careful consideration of multiple clinical factors. To do this successfully, the physicians managing these cases must have a solid knowledge about this condition. This article provides an overview of the key concepts and current knowledge of VMT.

KEYWORDS

vitreoretinal interface disorders, vitreomacular traction, diabetic retinopathy, pars plana vitrectomy

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STRESZCZENIE

Pierwsze wzmianki o trakcji szklistkowo-plamkowej (*vitreomacular traction* – VMT) pochodzą z 1953 roku, gdy Samuel Rodman Irvine zidentyfikował ją jako potencjalną przyczynę torbielowatego obrzęku plamki. W ostatnich latach w literaturze coraz częściej pojawiają się doniesienia o powiązaniach między VMT a innymi powszechnymi schorzeniami oczu, takimi jak retinopatia cukrzycowa i zwyrodnienie plamki związane z wiekiem (*age-related macular degeneration* – AMD). Celem pracy jest omówienie epidemiologii, czynników ryzyka, patofizjologii, metod diagnostycznych oraz możliwości leczenia VMT. Mechanizm rozwoju VMT w przebiegu retinopatii cukrzycowej różni się w zależności od tego, czy mamy do czynienia z proliferacyjną czy nieproliferacyjną postacią retinopatii. Wysiękowa postać AMD charakteryzuje się rozwojem neowaskularyzacji plamkowej, prowadzącej do wysięku lub krwotoku w plamce. Najnowsze badania sugerują również możliwy związek między AMD i VMT. VMT oraz inne zaburzenia interakcji szklistkowo-siatkówkowych to grupa schorzeń o zróżnicowanych patomechanizmach i powikłaniach. Wybór skutecznej metody leczenia wymaga uwzględnienia wielu czynników klinicznych. Aby było to możliwe, lekarze zajmujący się tym zagadnieniem muszą posiadać solidną wiedzę. Niniejszy artykuł stanowi przegląd kluczowych zagadnień oraz aktualnego stanu wiedzy na temat VMT.

SŁOWA KLUCZOWE

zaburzenia interakcji szklistkowo-plamkowych, trakcja szklistkowo-plamkowa, retinopatia cukrzycowa, witrektomia pars plana

Introduction

The first references to vitreomacular traction (VMT) date back to 1953, when Samuel Rodman Irvine identified it as a potential cause of cystoid macular edema (CME) [1]. In 1967, Jaffe [2] formally introduced the term “vitreomacular traction syndrome” and classified it as a distinct ocular disease entity. Histopathological studies conducted by Reese et al. [3], in conjunction with these definitions, provided conclusive confirmation of VMT. These studies confirmed the presence of a partially detached posterior vitreous with persistent adherence to the internal limiting membrane (ILM) in the foveal region. The current definition of VMT describes it as an anomalous adhesion of the posterior hyaloid membrane (PHM) to the macular region. During the process of posterior vitreous detachment (PVD), this persistent adhesion can create tractional forces on the macula, leading to disruption of the vitreomacular interface (VMI) and structural abnormalities within the retina itself [4,5]. The recent literature increasingly reports associations between VMT and other common ocular conditions, such as diabetic retinopathy (DR) and age-related macular degeneration (AMD). The aim of this review is to provide a comprehensive overview of VMT, focusing on its epidemiology, risk factors, pathophysiology, diagnostic methods, and current treatment options.

Epidemiology

Menzler et al. [6] conducted a systematic review of the literature to determine the prevalence and incidence rates of symptomatic vitreomacular adhesion (SVMA). Based on data from seven studies that provided epidemiological insights into VMT, the average prevalence was estimated to be 1.263 cases per 100,000 people. The incidence of VMT was reported in only one study, with a rate of 0.56 cases per 100,000 people.

Risk factors

PVD is a physiological process that occurs in all individuals with age. Its prevalence has been reported at 24% among patients aged 50–59 years, increasing to 87% in those aged 80–90 years [7]. PVD can be classified as either normal or anomalous [8]. For normal PVD to occur, two key conditions must happen simultaneously: liquefaction of the vitreous gel and weakening of vitreoretinal adhesion [9]. However, when vitreous liquefaction occurs without a corresponding decrease in vitreoretinal adhesion – or if this adhesion becomes abnormally strong – it can result in anomalous PVD, which can lead to VMT and other vitreoretinal interface disorders [9]. Several factors have been associated with an increased risk of developing VMT, including advanced age [9,10], female sex in the postmenopausal period, high myopia, intraocular inflammation, ocular trauma, and even retinal vein occlusion (RVO) [11].

In addition, conditions such as DR [9] and neovascular/exudative age-related macular degeneration (nAMD) [12,13] can also induce vitreous changes that predispose patients to VMT. Notably, a study by Xie et al. [14] reported that the coexistence of VMT and nAMD may reduce the early efficacy of intravitreal anti-vascular endothelial growth factor (anti-VEGF) injections.

In addition, a few studies suggest that ocular trauma and certain ophthalmic surgical procedures may also serve as potential risk factors for VMT development [15].

Pathophysiology

The role of posterior vitreous detachment in vitreomacular traction

The vitreous body, located between the lens and the retina, is a transparent gel-like structure composed of approximately 99% water, 0.5% hyaluronic acid, and 0.5% collagen – mainly types II, V, IX and XI [7]. This composition, typical of younger individuals, ensures that the vitreous remains optically clear, allowing unobstructed transmission of light to the retina [16].

With aging, particularly between the ages of 70 and 90, the volume of the gel component of the vitreous decreases, while collagen deposits increase. Over time, a process known as syneresis (or synchysis) occurs, involving liquefaction of the vitreous along with thickening and coiling of collagen fibrils. Syneresis alone, however, is not sufficient to initiate PVD. A second key process is the weakening of adhesion between the posterior vitreous cortex and the ILM, which allows the liquefied vitreous to separate from the retinal surface [7]. Traditionally, it was believed that PVD begins at the posterior pole and progresses peripherally. However, more recent studies suggest that PVD may actually start in the perimacular region and then extend to the fovea, inferior mid-periphery, and optic disc [7,16]. If this sequence occurs smoothly and in balance, it is considered a normal PVD. In contrast, an imbalance – specifically, vitreous liquefaction without sufficient weakening of vitreoretinal adhesion – can result in various complications such as vitreous hemorrhage, retinal tears, retinal detachment, vitreoretinal traction syndrome, VMT, or macular holes [7]. Interestingly, postmenopausal women appear to be more prone to early vitreous liquefaction, likely due to a drop in estrogen levels during menopause [7,11]. Hormone replacement therapy in these patients has been associated with a reduced risk of macular hole formation, particularly when VMT is present. However, because of its widespread systemic side effects, hormone replacement therapy is generally not recommended for this indication [7,17].

Vitreomacular traction in diabetic retinopathy

The mechanism of VMT development in the course of DR differs slightly depending on whether it is a proliferative diabetic retinopathy (PDR) or a non-proliferative diabetic retinopathy (NPDR) [18]. Therefore, in this publication, these mechanisms will be described separately.

In NPDR, metabolic activity within the vitreous body is significantly different than that of a healthy eye, with glucose metabolism being particularly disrupted. There is a notable reduction in glycolytic metabolites, accompanied by up to a tenfold increase in products of the pentose phosphate pathway [19]. This shift reflects a compensatory response to oxidative stress caused by diabetes, with the pentose phosphate pathway becoming more active as a protective mechanism [20]. Advanced glycation end-products (AGEs) in the vitreous of eyes with NPDR contribute to hyaluronic acid degradation and vitreous liquefaction, which may lead to vitreoschisis or incomplete PVD [18,21]. Additionally, the arginine-to-proline pathway, polyol pathway, and ascorbic acid metabolism are also affected by the disease [18]. As NPDR progresses, changes in aspartate and linoleic acid levels become apparent. A drop in linoleic acid accompanied by a rise in aspartate may indicate a shift toward the proliferative form of the disease (PDR) [22]. In NPDR, not only are metabolic processes disrupted, but there is also increased secretion of connective tissue growth factor (CTGF), which is present even in preclinical stages of the disease and contributes to vitreous fibrosis in NPDR [23].

Pathological processes are not limited to the vitreous body. The ILM thickens due to excess fibronectin and various types of collagen; the concentration of extracellular matrix proteins is also elevated. Hsieh and Yang [18] observed that, in addition to typical findings seen in idiopathic VMT – such as thickening of the hyaloid membrane, multilayer traction, and intraretinal cysts – traction in NPDR was also associated with macular retinoschisis, lamellar macular holes, full-thickness macular holes (FTMHs), and foveal detachment. Incomplete PVD with anteroposterior or oblique traction vectors, combined with strong adhesion to the epiretinal membrane (ERM), can intensify traction. In NPDR, the posterior hyaloid frequently adheres to the macula at multiple sites, often extending to the midperipheral retina due to abnormal vitreoretinal adhesion or fibrovascular tufts [18]. Altogether, these changes result in significantly stronger vitreomacular adhesion in diabetic eyes, which promotes the development of persistent and robust VMT [18]. This traction is often strong enough to delay the occurrence of complete PVD in DR patients compared to healthy individuals [24].

In turn, PDR is characterized by neovascularization: the growth of abnormal new blood vessels on the optic disc and across the retinal surface [25]. Adhesions form at the vitreoretinal interface, between the vitreous and retinal vessels, in which the PHM serves as a scaffold for the development of neovascular complexes [26]. The adherence of the PHM to the retina, combined with vitreous contraction, can lead to retinal elevation and splitting of the outer plexiform layer, resulting in retinoschisis [27]. ILM also plays a role in supporting glial proliferation, which can contribute to macular edema and the recurrence of ERM [21]. PDR affects the vitreous and other eye structures through three linked pathways: angiogenic, inflammatory, and fibrotic [28]. PDR triggers a response similar to the wound-healing process, where neovascularization is accompanied by the infiltration of inflammatory cells and myofibroblasts into the retina, leading to fibrosis [23]. The breach of the blood–retina barrier caused by PDR can increase the levels of chemoattractants in the vitreous cavity. These attract glial and epithelial cells,

promoting their migration and contributing to VMT. Elevated levels of inflammatory and growth factors, such as interleukin-6 (IL-6), interleukin-8 (IL-8), interleukin-1 beta (IL-1β), vascular endothelial growth factor (VEGF), C-C motif chemokine ligand 2 (CCL2), endothelin-1 (EDN1), and tumor necrosis factor (TNF) can be observed in this process [29]. The chemokine CCL2, which plays a key role in retinal inflammation in diabetic eyes, also activates retinal microglia and macrophages in mice, disrupting the blood–retina barrier [26]. Elevated levels of IL-8 in the vitreous have been proven to worsen vision outcomes following vitrectomy in cases of PDR [30].

The most extensively studied growth factor in PDR is VEGF, which is produced by endothelial cells and the retinal pigment epithelium (RPE). VEGF reduces occludin levels and inhibits protein kinase C beta (PKC-β), increasing vascular permeability. Elevated VEGF levels are commonly observed in the vitreous of patients with PDR and diabetic macular edema [28]. In addition to the rise in pro-angiogenic and pro--inflammatory factors in PDR, there is also evidence of a reduction in anti-inflammatory factors, which further intensifies disease progression in the eye [28]. The enriched glial-stimulating environment in PDR increases the likelihood of vitreoretinal traction compared to NPDR. However, this does not mean that traction does not occur in DR without proliferation [18].

Vitreomacular traction in age-related macular degeneration

AMD remains the leading cause of vision loss in industrialized countries [31]. It is typically classified into two forms: dry AMD and nAMD. The exudative form is characterized by the development of macular neovascularization (MNV), which can lead to fluid leakage or hemorrhage in the macula [12]. Recent studies have suggested a potential link between nAMD and VMT [12,32,33]. Although the nature of this association is still not fully understood, most researchers agree that VMT alone is unlikely to initiate nAMD. It appears more likely that nAMD develops first, and that mechanical traction from VMT worsens the disease course [12,32]. The most commonly proposed pathophysiological mechanism is based on the theory that persistent adhesion between the vitreous and retina allows oxidative stress and pro--angiogenic cytokines to remain concentrated near the macula for prolonged periods. This extended exposure may promote the development of AMD [33]. It is important to point out that AMD is a multifactorial disease in which the crucial factor is chronic subretinal inflammation, called parainflammation or inflam-maging. Thus, the presence of vitreomacular adhesion additionally may be a risk factor and may contribute to the development of AMD [34,35]. Additionally, tractional forces and abnormal VMA can impair the diffusion of oxygen and nutrients to the macula. They may also sustain low-grade chronic inflammation and intensify macular edema caused by leaky MNV vessels [12,32,33]. Physiological PVD is thought to act as a natural defense mechanism by creating a space between the retina and vitreous body; this helps clear toxins and supports oxygen exchange [12,33]. Ondeş et al. [36] reported that PVD was significantly less common in eyes with AMD (33%) than in control eyes without the disease (50%). A study by Krebs et al. [37] demonstrated that complete PVD was significantly less frequent in eyes with nAMD (34.0%) compared to eyes with dry AMD (71.9%, $p = 0.00002$) and control eyes (60.7%, $p = 0.017$), suggesting a potential protective effect of PVD against the development of nAMD. However, when PVD is incomplete or when strong adhesion persists, these protective benefits are lost and the resulting traction may accelerate retinal degeneration and AMD progression [12]. Moreover, the presence of VMT might also interfere with the efficacy of anti-VEGF treatments in patients with nAMD, potentially limiting therapeutic outcomes [4,12].

Diagnosis and classification of vitreomacular traction

Optical coherence tomography

Optical coherence tomography (OCT) is a very important diagnostic tool with a significant influence on decisions regarding surgical treatment [38]. Currently, the most widely used and clinically relevant classification system is that developed by Duker et al. [5]. It is an anatomical classification based on OCT imaging that divides VMI into three stages: vitreomacular adhesion, VMT, and FTMH.

Vitreomacular adhesion

This is the first stage of VMI, in which partial abnormal separation of the vitreous has occurred in the perifoveal area, without retinal abnormalities. The OCT criteria required for a diagnosis of VMA include elevation of the cortical vitreous above the retinal surface and persistent vitreous attachment within 3 mm of the fovea. Based on the size of the adhesion, VMA can be classified as either focal (≤1,500 μm) or broad (>1,500 μm). Additionally, when classifying VMA, the coexistence of other retinal diseases – AMD, RVO, or diabetic macular edema – is described; in such cases, it is referred to as concurrent VMA. If it occurs without any coexisting conditions, it is referred to as isolated VMA [5].

Vitreomacular traction

This is the next stage of VMI, in which excessive traction on the macular area leads to anatomical disturbances in the contour of the foveal surface, the formation of intraretinal pseudocysts, elevation of the fovea from the RPE, or a combination of these three features. The diagnostic criteria for VMT on OCT require the visualization of all of the following

anatomical abnormalities on at least one B-scan OCT image: perifoveal separation of the vitreous cortex from the retinal surface, vitreous adhesion to the macula within 3 mm of the foveal center, and traction on the macular area causing structural changes such as distortion of the foveal contour, formation of intraretinal pseudocysts, elevation of the fovea from the RPE, or a combination of these findings. Another key criterion for a diagnosis of VMT is the preservation of retinal structural continuity, with no full-thickness disruption across all retinal layers. In terms of classification, the simplest approach divides VMT based on size of the adhesion into focal (≤1,500 μm) and broad (>1,500 μm) [5]. Steel et al. [39] developed a more detailed classification system for VMT known as WISPERR. It includes seven parameters used to assess focal VMT, aiming to provide a more comprehensive description, which may have prognostic value in future studies regarding spontaneous resolution or progression to FTMH. The WISPERR algorithm includes the following components: W – width of vitreomacular attachment; I – interface between the retina and the vitreous cavity; S – shape; P – pigment epithelium; E – elevation of the retinal surface from the RPE; R1 – inner retina; and R2 – outer retina [39] (Figures 1–3).

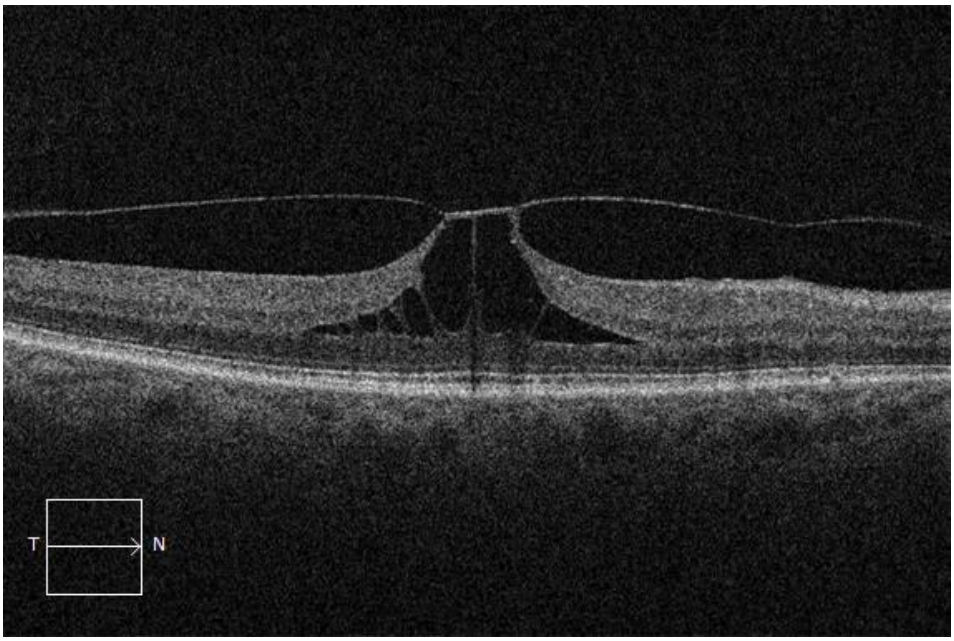

Fig. 1. Optical coherence tomography scan of the macula of the left eye, showing vitreomacular traction with loss of the foveal contour and intraretinal fluid

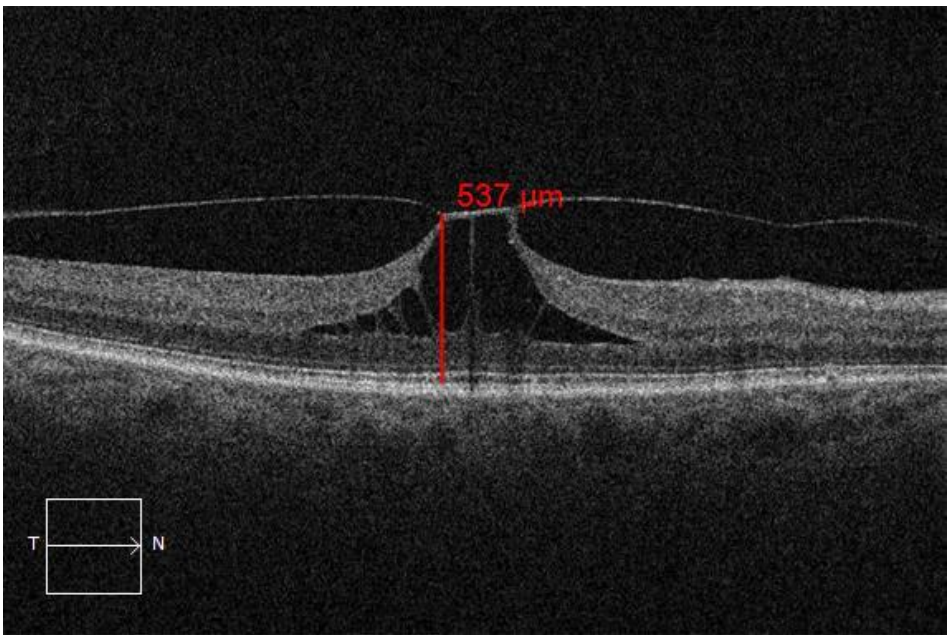

Fig. 2. Optical coherence tomography scan of the left eye, showing non-complete posterior vitreous detachment with retinal fraction and elevation of the retinal surface from the retinal pigment epithelium of 537 μm

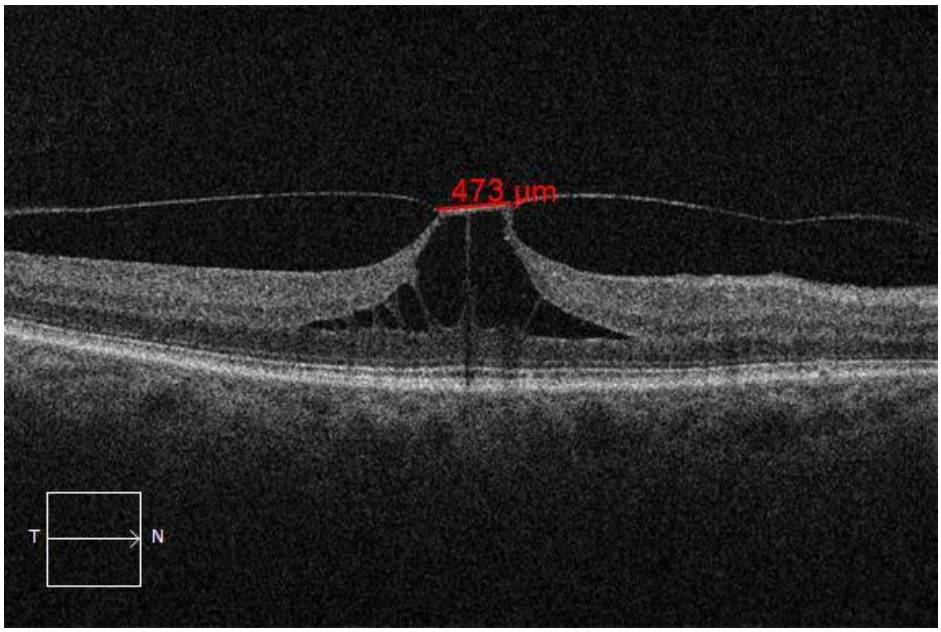

Fig. 3. Optical coherence tomography scan of the left eye, showing a width of vitreomacular attachment of 473 µm

Full-thickness macular hole

This is the final stage of VMI, in which VMT leads to a full-thickness disruption of all neurosensory retinal layers from the ILM to the RPE. The OCT criteria required to diagnose FTMH include visualization of a full-thickness defect involving all retinal layers on at least one B-scan OCT image [5]. FTMH can be classified according to the updated Gass [40] classification, which divides FTMH into five stages, of which only the last three describe the macular hole, while the first two correspond to VMA (stage 0) and VMT (stage 1 – impending macular hole). Stage 3 refers to a small FTMH with a hole diameter of less than 250 µm. Stage 4 is a medium FTMH with a diameter between 250 and 400 µm. The final stage, stage 5, describes a large FTMH with a diameter greater than 400 µm [5,40].

Dynamic B-scan ultrasonic examination

Dynamic B-scan ultrasonography is not the first-line diagnostic tool for detecting VMT. However, in cases where the vitreous or lens media are opaque, OCT imaging is impossible, or when OCT equipment is unavailable, ultrasonographic examination using 10- or 20-MHz probes can be a valuable alternative. In such cases, ultrasound may reveal a peripherally detached posterior hyaloid with persistent attachment over the posterior pole, allowing for an accurate assessment of the vitreoretinal interface [41].

Other imaging methods

Fluorescein angiography of the fundus can show capillary leakage in the macular area and at the optic disc, which may result from traction-related damage caused by VMT. However, such leakage is not always present [41]. This technique is not considered a reliable diagnostic method for evaluating the VMI.

Treatment methods for vitreomacular traction

Several treatment approaches for VMT have been described in the literature. The least invasive option is observation, allowing time for the traction to resolve spontaneously. More active interventions include enzymatic or pneumatic vitreolysis (PVL), while the most invasive method involves performing a pars plana vitrectomy (PPV) [42].

Spontaneous resolution of vitreomacular traction

Surprisingly, VMT spontaneous resolves relatively often [43]. In a frequently cited study by Hikichi et al. [44], which examined 53 eyes with VMT, complete PVD was observed in 6 eyes, corresponding to a spontaneous release rate of 11%. In a study conducted by Errera et al. [45] on 183 eyes of 159 patients with VMT without associated retinal diseases, the spontaneous release rate was reported at 20%. These studies suggest that, at best, 1 in 5 patients may experience spontaneous resolution of VMT. According to a study by Dimopoulos et al. [46], such low rates may be related to short follow-up periods. Their findings indicate that the likelihood of VMT resolution does not appear to decrease even after 12 months of observation. Certain clinical features can reduce the chances of spontaneous resolution. For example, a vitreomacular adhesion diameter greater than 400 µm has been shown to reduce the likelihood of spontaneous release by 99% compared to patients with smaller adhesion areas [43]. On the other hand, factors such as a wider angle between the nasal and temporal sides of the vitreoretinal interface, as well as a characteristic V-shaped configuration of the traction, are associated with a higher likelihood of spontaneous separation [42].

Observation is recommended in the early stages of the disease; however, spontaneous release does not occur in all patients and once symptoms appear or the disease progresses to macular hole, surgical intervention is advised [45].

Pharmacologic vitreolysis

In the context of pharmacological treatment for VMI, several vitreolytic agents are currently being investigated for their ability to induce PVD in cases of VMT. These agents are typically divided into two groups. Enzymatic agents include tissue plasminogen activator (tPA), plasmin, microplasmin, nattokinase, chondroitinase, hyaluronidase, and dispase. Non-enzymatic options include compounds such as vitreosolve and RGD-peptides (arginine-glycine-aspartate) [47]. Ocriplasmin, formerly known as microplasmin, is the most studied of these agents. Administered via intravitreal injection, it works by breaking down proteins such as collagen, fibronectin, and laminin: components responsible for vitreoretinal adhesion. This process leads to vitreous liquefaction and reduces vitreoretinal adhesion [48,49,50]. The best candidates for ocriplasmin injection are phakic eyes and patients ≤65 years of age, with no history of prior ocular surgery, no DR, no ERM, a VMA less than 1,500 μm, no macular pucker, and FTMH smaller than 250 μm. Relative indications for ocriplasmin use include FTMH with a diameter between 250 μm and 400 μm and VMT that meets the OCT criteria: small adhesion area and V-shaped VMT with wide angles [48,51]. Contraindications include FTMH >400 μm, myopia greater than 5 diopters, prior vitrectomy (within the past 3 months), previous intravitreal injection (within the past 6 months), history of rhegmatogenous retinal detachment, fibrocellular proliferation at the level of the ILM and ERM, and severe peripheral retinal degeneration [48,51]. Complications of treatment may include temporary reduction in visual acuity, photopsia, pupillary abnormalities, and lens subluxation. Retinal toxicity can present with various symptoms and structural changes such as retinal vessel narrowing, abnormal autofluorescence, accumulation of subretinal fluid, transient retinal detachment, and enlargement of the macular hole [48]. Ocriplasmin appears to be a useful agent in the treatment of VMT and FTMH; however, experts have not yet reached a consensus on whether it will become the standard of care [48].

Pneumatic vitreolysis

PVL is a non-surgical method for treating conditions associated with VMT. It involves intravitreal injection of an expanding gas, such as perfluoropropane (C3F8), into the eye with the goal of inducing PVD and thereby releasing the traction [52]. In a retrospective case series, Rodrigues et al. [53] described 15 eyes treated with intravitreal perfluoropropane gas injection, reporting VMT release in 40% of eyes at 1 month and in 60% of eyes within 6 months. They also observed higher success rates in patients with smaller VMA size, lower maximum macular thickness, and low vitreous face reflectivity. No retinal tears or other serious complications were reported among the 15 cases in this study. Chan et al. [54] conducted two studies involving 56 eyes: a randomized clinical trial (Protocol AG) comparing PVL to sham injection in VMT without FTMH and a single-arm study (Protocol AH) assessing PVL for closing small FTMHs. In Protocol AG, 78% of eyes in the PVL group achieved VMT release without the need for vitrectomy, compared to just 9% in the observation group after 24 weeks. In Protocol AH, PVL led to successful closure of FTMHs ≤250 μm in 29% of eyes after 8 weeks. Although hyaloid detachment occurred in nearly all cases, the macular hole closure rate remained lower than that achieved with vitrectomy (80% vs. 100%). Both protocols were terminated early due to a higher-than-expected incidence of complications, including retinal detachment and retinal tear. Across both studies, 7 out of 59 eyes (12%) treated with PVL developed retinal detachment (6 cases) or retinal tear (1 case). Other complications reported following PVL include endophthalmitis, development of new macular holes, traumatic cataract, cataract surgery, vitreous hemorrhage, and fluctuations in intraocular pressure. While PVL is theoretically safer, easier, and more cost-effective than surgical vitrectomy [52], current clinical data does not yet fully support its widespread use.

Pars plana vitrectomy

PPV is a traditional and widely used surgical method for treating vitreoretinal conditions such as VMT and macular hole [52,55,56]. The procedure involves several steps, beginning with the removal of the vitreous body [52]. A particularly important step is peeling of the ILM, and in some cases, the ERM as well. PPV with ILM peeling is considered a very safe procedure for treatment of macular hole, offering favorable anatomical and functional outcomes with a low rate of postoperative complications. ILM peeling significantly increases macular hole closure rates and is associated with a reduced need for reoperation and lower recurrence rates. Closure rates for macular holes after PPV with ILM peeling typically range from 90% to over 97%. However, this rate can be influenced by factors such as the size of the macular hole. In cases with smaller holes, the closure rates are higher (>96%) as compared to larger macular holes (40%–80%). The inverted ILM flap technique for macular hole closure shows higher anatomical success rate, ranging from 96% to 100%, even in challenging cases such as large idiopathic macular holes [56]. To improve visualization during membrane peeling, vital dyes such as brilliant blue or trypan blue are sometimes used [56]. Precise peeling of the ILM and ERM is described as the most effective method for treating VMT [57]. After vitreous removal, a gas tamponade (e.g., SF6 18%–20% or C3F8 12%–14%) is often used to stabilize the retina

and support closure of the macular hole. Following surgery, patients are frequently advised to maintain a face-down position for a specific period (e.g., 2–3 days). However, some sources suggest that hole closure often occurs within the first 24 hours after surgery, regardless of positioning, if wide ILM peeling and gas tamponade are performed [56]. As with any surgery, PPV carries potential risks, including retinal tears with or without detachment, cataract formation or progression, endophthalmitis, reopening of a closed macular hole, intraoperative retinal breaks, lamellar macular hole formation, lens dislocation, zonular damage, and elevated intraocular pressure [55,57]. According to a meta-analysis by Quiroz-Reyes et al. [57], PPV is associated with fewer serious complications than pharmacologic vitreolysis (with ocriplasmin) or PVL. It also appears to be more effective, with VMT release rates reaching 100% and macular hole closure rates of 95%, outperforming both alternative therapies. Indications for PPV include significant vision loss due to vitreomacular disorders, large VMTs (>1,500 μm), the presence of ERM (which anchors the vitreous to the retina and inhibits pharmacologic and pneumatic vitreolysis), macular holes larger than 400 μm, an absence of clear traction on imaging, and failure of other treatment methods [52,56]. In summary, PPV provides the most effective treatment approach for VMT [55] and is widely regarded as the gold standard in clinical practice [52].

Conclusions

VMT and other vitreoretinal interface disorders represent a group of conditions with diverse underlying mechanisms and associated complications. An optimal therapeutic strategy requires a comprehensive evaluation of multiple clinical parameters. Clinicians must possess an in-depth understanding of the underlying pathophysiology and natural course of the condition in order to make evidence-based decisions. This paper provides an overview of the key concepts and current knowledge about VMT, but it does not cover all aspects of the condition. Moreover, many aspects related to risk factors, pathogenesis, and treatment remain incompletely understood and require further research.

Authors' contribution

Study design – S. Kowalczyk, M. Guzikowski, S. Sirek, D. Wyględowska-Promieńska
Data collection – S. Kowalczyk, M. Guzikowski
Manuscript preparation – S. Kowalczyk, M. Guzikowski, S. Sirek, D. Wyględowska-Promieńska
Literature research – S. Kowalczyk, M. Guzikowski, D. Wyględowska-Promieńska
Final approval of the version to be published – D. Wyględowska-Promieńska

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