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ORIGINAL RESEARCH

Clinical Characteristics and Treatment Outcomes of Breakthrough Vitreous Hemorrhage in Peripheral Exudative Hemorrhagic Chorioretinopathy (PEHCR)

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Purpose: To evaluate the clinical characteristics and treatment outcomes of breakthrough vitreous hemorrhage secondary to peripheral exudative hemorrhagic chorioretinopathy (PEHCR).

Methods: This retrospective study included 14 eyes from 14 patients with vitreous hemorrhage secondary to peripheral retinochoroidal mass lesions. Data collected included demographic profiles, clinical presentation, multimodal imaging findings, and treatment outcomes following pars plana vitrectomy (PPV), intravitreal anti-VEGF injections, or laser photocoagulation.

Results: The median age at presentation was 83 years (range, 58–91), with nine females (64.3%). Median presenting visual acuity (VA) was 1.3 logMAR (range, 0.3–2.7). All patients had normal intraocular pressure. Bilateral PEHCR was observed in 50%, though hemorrhage occurred unilaterally. Unifocal lesions were present in 71.4%, with a mean lesion thickness of 3.4 mm (range, 1.5–6.8 mm). Dense vitreous hemorrhage obscuring posterior pole details was seen in eight eyes (57.1%) and required PPV. The remaining six eyes, with moderate hemorrhage, improved spontaneously without surgery. Intravitreal anti-VEGF therapy was administered in five eyes for macular involvement or to prevent recurrent hemorrhage. At a median follow-up of 11.7 months (range, 3–63), median VA improved to 0.36 logMAR (range, 0.1–2.0). The mean VA gain was 0.76 logMAR in the vitrectomy group ($p = 0.004$) and 0.55 logMAR in eyes without macular involvement ($p = 0.024$). However, five eyes (35.7%) had final VA $\leq$ 20/200 due to macular pathology consistent with age-related macular degeneration or polypoidal choroidal vasculopathy-like changes.

Conclusion: PEHCR with breakthrough vitreous hemorrhage is a rare but important diagnostic consideration in patients presenting with peripheral retinochoroidal mass-like lesions. PPV and intravitreal anti-VEGF therapy may improve visual outcomes in these cases. However, visual recovery may be limited in cases with macular involvement due to irreversible retinal damage. Early diagnosis and tailored management are essential to optimize outcomes and avoid misdiagnosis.

Keywords: peripheral exudative hemorrhagic chorioretinopathy, vitreous hemorrhage, choroidal mass, vitrectomy, choroidal melanoma

Introduction

Peripheral exudative hemorrhagic chorioretinopathy (PEHCR) was first described by Reese and Jones in 1961,[1] with Annesley later naming the condition in 1980.[2] It is a typically bilateral, asymmetric retinal degenerative disorder that predominantly affects the elderly population. The condition is characterized by retinal pigment epithelial detachment (PED), subretinal and sub-retinal pigment epithelial (RPE) hemorrhages or exudates, and may present with fibrotic masses located outside the macular region.[2–4] Although its exact etiology remains unknown, PEHCR shares features with both age-related macular degeneration (AMD) and polypoidal choroidal vasculopathy (PCV).[3,5,6]

PEHCR is one of the primary conditions that mimics choroidal melanoma, as hemorrhagic retinal lesions can appear dark and elevated, leading to frequent misdiagnosis as intraocular tumors.[3,7,8] Consequently, patients are often referred to ocular oncologists for further evaluation. Differentiating PEHCR from choroidal melanoma is critical due to the distinct

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management strategies and prognoses associated with each condition. Shields et al identified several diagnostic features that are suggestive of PEHCR, including exudation, atrophic changes in the RPE, hypofluorescence on fluorescein angiography, absence of intrinsic vascular pulsations and sentinel vessels on slit-lamp biomicroscopy, lack of ciliary body extension, and the presence of clot retraction clefts on echography. Additionally, bilateral involvement (with findings in the non-presenting eye) further supports a diagnosis of PEHCR over melanoma.[3,8]

Although many cases of PEHCR are self-limiting, vision loss can occur due to subretinal hemorrhage or exudation extending from the periphery toward the macula, with breakthrough vitreous hemorrhage occurring in approximately 10–20% of cases, a significant contributor to visual impairment.[7,9,10] In cases complicated by breakthrough vitreous hemorrhage, where fundus examination is obscured, echography may mimic a choroidal mass due to the accumulation of hemorrhagic blood, making differential diagnosis and management particularly challenging. The management of PEHCR is predominantly observational, as spontaneous improvement is commonly observed.[3] In rare instances, the disease may progress aggressively, resulting in extensive subretinal or vitreous hemorrhage that necessitates pars plana vitrectomy and surgical evacuation of blood. Despite the clinical significance of these severe manifestations, research on this aggressive presentation of PEHCR remains limited.

This study evaluated the clinical characteristics and treatment outcomes, including vitrectomy, intravitreal anti-VEGF therapy, and laser photocoagulation, in patients with aggressive PEHCR who presented with breakthrough vitreous hemorrhage, a relatively uncommon manifestation. Additionally, the study sought to identify risk factors associated with the aggressive progression of this condition.

Materials and Methods

This retrospective study included patients diagnosed with PEHCR who were referred for for vitreous hemorrhage with a peripheral retinochoroidal mass lesion, over concern of a possible unspecified choroidal mass or a choroidal lesion resembling a uveal melanoma. A total of 14 eyes from 14 patients, selected from referral cases between October, 2018, and September, 2024, at the University of Illinois Chicago, were included. The study adhered to the principles of the Declaration of Helsinki was approved by the Institutional Review Board and ethics committee of University of Illinois at Chicago (approval number: Study2023-1435). Informed consent was waived due to the retrospective nature of the study. No identifiable patient data or images are included in this publication.

Exclusion criteria encompassed cases with incomplete ocular history, follow-up periods shorter than 3 months, and any history or signs of trauma, inflammation, infection, tumor, drug toxicity, congenital or developmental anomalies, or other ocular or systemic conditions associated with intraocular hemorrhage, fluid accumulation, or exudation.

Data collected for each patient included age at the initial consultation, symptom onset, gender, ethnicity, presence of unilateral or bilateral ocular involvement, medical history, family and trauma history, and current medications. For each affected eye, best-corrected visual acuity (VA) and findings from slit-lamp and fundus examinations at the initial and final follow-up visits were recorded. Given that fundus examinations were obscured by vitreous hemorrhage in all cases, preoperative evaluations included echography, and all treatments for PEHCR were documented.

Surgery and Postoperative Assessment

For eyes with dense vitreous hemorrhage, where clinical examination and imaging strongly suggested PEHCR, vitrectomy was recommended if VA remained low without improvement for 2–6 weeks. All procedures were performed at the University of Illinois Chicago, a tertiary academic referral center. All surgeries were performed by experienced vitreoretinal surgeons using the Alcon Constellation system (Alcon, USA). A standard three-port, 25-gauge vitrectomy was performed under monitored anesthesia care (MAC). The primary purpose of the surgery was to remove vitreous hemorrhage. In eyes with active lesions, intravitreal anti-VEGF was administered. No cases required additional surgical intervention for subretinal or subchoroidal hemorrhage. The surgical approach involved an initial core vitrectomy, followed by meticulous shaving of the vitreous base, facilitated by the Zeiss Resight visualization system with scleral indentation. Intraoperative laser photocoagulation was applied in selected eyes with suspected areas of active choroidal neovascularization not involving the macular region. At the conclusion of the procedure a fluid-air exchange was

performed to remove residual hemorrhage, and the eye was subsequently filled with balanced salt solution. No cases required gas or silicone oil for tamponade.

Postoperative follow-up visits were scheduled for the first day, one week, monthly thereafter, and then extended based on the stability of clinical conditions. Additional intravitreal anti-VEGF injections were administered in cases of active disease progression, particularly with macular involvement. Visual acuity and any complications were documented at each visit, while OCT and echography were utilized to monitor clinical improvement and treatment outcomes.

Statistical Analysis

Snellen visual acuity measurements were converted to logarithm of the minimum angle of resolution (logMAR) values. Poor visual acuity outcomes were assigned logMAR values as follows: 2.0 for counting fingers, 2.3 for hand motion, 2.5 for light projection, 2.7 for light perception, and 3.0 for no light perception. To evaluate visual outcomes following treatment with vitrectomy, intravitreal anti-VEGF injections, and in relation to macular involvement, multilevel linear regression with random intercepts and random slopes was performed. A p-value of less than 0.05 was considered statistically significant.

Results

Demographic Characteristics at Presentation

Breakthrough vitreous hemorrhage secondary to PEHCR was diagnosed in 14 eyes from 14 patients. Diagnosis in all cases was confirmed through clinical identification of characteristic lesions, supported by imaging techniques such as echography and angiography. Of these patients, nine (64.3%) were female, with a median age at presentation of 83 years (mean 77; range 58–91). The majority of patients were Caucasian, with three identifying as African American (21.4%).

Comorbid conditions included hypertension in 13 patients (92.9%), diabetes mellitus in seven (50%), chronic kidney disease in six (42.9%), atrial fibrillation in three (21.4%), and coronary artery disease in one (7.1%). Three patients (21.4%) were on systemic anticoagulation therapy with apixaban or warfarin for atrial fibrillation (Table 1). Vitreous hemorrhage predominantly affected the left eye, occurring in 10 of 14 cases. Bilateral involvement was observed in seven patients (50.0%); however, vitreous hemorrhage was present in only one eye per patient. At presentation, nine eyes (64.3%) were pseudophakic, three (21.4%) had a concurrent diagnosis of non-exudative AMD, and three (21.4%) showed mild to moderate non-proliferative diabetic retinopathy (NPDR). None of the patients had a history of ocular trauma or prior intravitreal anti-VEGF injections.

Clinical Characteristics at Presentation

The main clinical characteristics at presentation are summarized in Table 1. Since this series exclusively included cases of breakthrough vitreous hemorrhage, the majority of patients presented with visual loss in 10 patients (71.4%), floaters in seven patients (50.0%), and visual field defects in five patients (35.7%). The median duration from symptom onset to presentation was 17.5 days (mean, 21.8; range, 7–45 days).

The primary referral diagnosis was non-clearing vitreous hemorrhage with a peripheral retinochoroidal mass lesion, raising concern for a possible unspecified choroidal mass in eight eyes (57.1%) and suspected uveal melanoma in six eyes (42.9%). Median visual acuity at presentation was 1.3 logMAR (mean, 1.3; range, 0.3–2.7). At presentation, eight eyes (57.1%) exhibited dense vitreous hemorrhage, resulting in a completely obscured view of the retina and optic disc. The remaining six eyes (42.9%) presented with a moderate degree of vitreous hemorrhage, allowing partial visibility of some retinal vessels and the optic disc. One patient (Case 1) experienced breakthrough hemorrhage into the anterior chamber.

Imaging Findings

At initial presentation, all patients underwent comprehensive A- and B-scan echography due to obscured fundus examination resulting from breakthrough vitreous hemorrhage. Once visualization improved, optical coherence tomography (OCT) was performed for all patients, with fluorescein angiography (FFA) and indocyanine green angiography (ICGA) conducted in selected cases without contraindications.

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Table 1 Characteristics and Treatment Outcomes of Patients with Peripheral Exudative Hemorrhagic Chorioretinopathy (PEHCR) and Vitreous Hemorrhage

Case	Gender, Age	Systemic Diseases (Anticoagulant/ Antiplatelet Drugs)	VA of Affected Eye at Baseline	Degree of VH at Baseline	Laterality	Thickness of Mass from Echography (mm)	No. of Lesion Quadrant/ Macular Involvement	Treatment			VA of Affected Eye After Treatment	Thickness of Lesion from Echography (mm) at Last Follow-Up	Follow Up Duration (Months)
								PPV	No. of IVT	Laser			
1	F, 90	HT, AF, CKD (Apixaban)	LP	Dense	Bilat	6.8 (multifocal)	Y	Y	0	N	CF	3.0 (unifocal)	63
2	F, 61	HT, DM (none)	20/50	Moderate	Bilat	2.0 (unifocal)	N	N	0	N	20/25	1.5 (unifocal)	33
3	M, 80	HT, DM (none)	20/40	Moderate	Unilat	1.5 (unifocal)	N	N	0	N	20/25	0.8 (unifocal)	58
4	M, 82	HT, DM (none)	20/40	Moderate	Bilat	2.2 (unifocal)	N	N	0	Y	20/40	n/a	3
5	F, 60	DLP (none)	20/400	Dense	Unilat	3.7 (unifocal)	N	Y	0	Y	20/40	2.6 (unifocal)	3
6	M, 91	HT, AF, CKD ban, aspirin)	HM	Dense	Bilat	4.5 (unifocal)	Y	Y	1 (Bevacizumab)	Y	CF	1.5 (unifocal)	10
7	M, 86	HT, CAD, CKD (warfarin)	CF	Dense	Bilat	3.5 (multifocal)	Y	Y	3 (2 bevacizumab then aflibercept)	Y	CF	2.0 (multifocal)	13
8	F, 89	HT, DM (none)	20/400	Dense	Bilat	2.0 (multifocal)	Y	Y	0	N	20/400	n/a	37
9	F, 70	HT, DM (none)	20/40	Moderate	Bilat	4.0 (unifocal)	N	N	0	N	20/25	2.0 (unifocal)	33
10	F, 82	HT, DM (aspirin)	20/70	Moderate	Unilat	2.0 (unifocal)	N	N	8 (aflibercept)	N	20/50	n/a	16
11	F, 70	HT, DM, CKD (aspirin)	HM	Dense	Unilat	2.0 (unifocal)	N	Y	0	N	20/60	n/a	3
12	M, 90	HT, CKD (none)	20/400	Moderate	Unilat	4.2 (multifocal)	Y	N	3 (bevacizumab)	Y	20/40	3.9 (multifocal)	8
13	F, 64	HT (none)	20/200	Dense	Unilat	3.3 (unifocal)	N	Y	0	N	20/50	n/a (unifocal)	7
14	F, 58	HT, DM, CKD (aspirin)	HM	Dense	Unilat	5.3 (unifocal)	Y	Y	3 (bevacizumab)	Y	20/200	3.5 (unifocal)	5

Abbreviations: F, Female; M, Male; HT, Hypertension; VH, Vitreous hemorrhage; DM, Diabetes Mellitus; AF, Atrial Fibrillation; CKD, Chronic Kidney Disease; DLP, Dyslipidemia; CAD, Coronary Artery Disease; VA, Visual Acuity; LP, Light Perception; HM, Hand Motion; CF, Counting Fingers; Bilat, Bilateral; Unilat, Unilateral; PPV, Pars Plana Vitrectomy; IVT, Intravitreal Injection; Y, Yes; N, No; n/a, not applicable; No, number.

Table 2 Imaging Findings of Patients with Peripheral Exudative Hemorrhagic Chorioretinopathy (PEHCR) and Vitreous Hemorrhage

Modality and Findings	Number (%)
Ocular echography (n=14)	
A-scan internal reflectivity	
Low	5 (35.7)
Intermediate	8 (57.1)
High	1 (7.1)
B-scan configuration	
Dome	6 (42.9)
Plateau	8 (57.1)
Mushroom	0
B-scan acoustic heterogeneity	
Heterogenous	9 (64.3)
Homogenous	5 (35.7)
B-scan acoustic quality	
Hollow	3 (21.4)
Intermediate	10 (71.4)
Solid	1 (7.1)
B-scan other features	
Cleft between subretinal blood and choroid	6 (42.9)
Choroidal excavation	0
Related fundus features (n=14)	
Subretinal fluid	7 (50)
Subretinal hemorrhage	11 (78.6)
Serous pigment epithelial detachment (PED)	6 (42.9)
Hemorrhagic PED	12 (85.7)
Subretinal exudate	9 (64.3)
Retinal pigment epithelium (RPE) hyperplasia or atrophy	11 (78.6)
Optical coherence tomography (OCT)	
Macular findings (n=14)	
Subretinal fluid	2 (14.3)
Subretinal hemorrhage	1 (7.1)
Serous PED	1 (7.1)
Hemorrhagic PED	0
Fibrosis/scarring	4 (28.6)
Pachychoroid features	8 (57.1)
Periphery through lesions (n=6)	
Subretinal fluid	3 (50)
Subretinal hemorrhage	0
Serous PED	4 (75)
Hemorrhagic PED	4 (75)
Fibrosis/scarring	0
Fluorescein angiography (FFA) (n=6)	
Leakage at late phase	2 (33.3)
Staining	3 (50)
Pooling	1 (16.7)
Hypofluorescence from blockage	5 (83.3)
RPE atrophy	6 (100)

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A summary of imaging findings is presented in Table 2. The most common features were plateau-shaped elevations on B-scan (8 eyes, 57.1%) and intermediate to solid acoustic patterns (11 eyes, 78.6%). Clot retraction clefts were observed in 6 eyes (42.9%), while no intrinsic vascular pulsations or choroidal excavation were detected. A-scan most frequently showed intermediate to high reflectivity (9 eyes, 64.3%). Multiple lesions (2–3 lesions) were observed on B-scans in four eyes (28.6%), with a mean maximum thickness of 3.4 mm (range 1.5–6.8 mm).

After improved visualization, either through surgical intervention or spontaneous clearing, fundus examination revealed that PEHCR lesions presented as peripheral retinochoroidal mass lesions in all affected eyes. Lesions extended over more than one quadrant in eight eyes (57.1%), involving up to three quadrants. The temporal region was most commonly affected, seen in nine eyes (64.3%), followed by two eyes nasally (14.3%), two eyes inferiorly, and one eye superiorly (7.1%). The lesions were typically located between the equator and ora serrata in 57.1% of cases. Associated fundus features included hemorrhagic PED in 12 eyes (86%), serous PED in six eyes (43%), subretinal hemorrhage in 11 eyes (79%), subretinal fluid in seven eyes (50%), subretinal exudate in nine eyes (64%), and RPE hyperplasia or atrophy in 11 eyes (79%). Active macular involvement was observed in two eyes, presenting with hemorrhage or exudative changes. Among the seven patients with bilateral PEHCR, the contralateral eye (without vitreous hemorrhage) showed PEHCR lesions, all located on the temporal side. These lesions were mostly small, unifocal, and confined to no more than one quadrant, with leakage observed on FFA. Only one of these eyes had active macular involvement, necessitating an anti-VEGF injection.

Intravenous wide-field FFA was performed in six eyes. Of these, two eyes (33%) demonstrated late leakage, three eyes (50%) showed staining, and one eye (17%) exhibited pooling. Patchy blockage of choroidal fluorescence, associated with subretinal hemorrhage, sub-RPE hemorrhage, or RPE hyperplasia, was observed in five eyes (83%). All imaged eyes showed peripheral angiographic changes suggestive of RPE hyperplasia or atrophy. ICGA was performed in two eyes, with no polyps observed in either case.

OCT was performed on all patients. Macular involvement was identified in six of 14 eyes, with active lesions in two eyes presenting as subretinal fluid, subretinal hemorrhage, or serous PED. Four eyes showed inactive macular involvement, characterized by outer retinal loss, subretinal fibrosis, and RPE alterations. Pachychoroid features were noted in six eyes (43%), including increased choroidal thickness, the presence of pachyvessels, and attenuation of the inner choroid. Peripheral lesion imaging with OCT, performed in six eyes, revealed hemorrhagic PED in four eyes, serous PED in four eyes, and subretinal hemorrhage in three eyes.

Management

Systemic blood pressure was controlled in all patients, given its potential contribution to the severity of vitreous hemorrhage in PEHCR. All patients with non-clearing, dense vitreous hemorrhage, resulting in a completely obscured view of the retina and optic disc (eight eyes, 57.1% of total patients), underwent PPV for blood evacuation after a period of observation without intravitreal injection. The observation period was determined through a collaborative decision between the patient and the surgeon, with a median time from presentation to surgery of 32 days (mean 53; range 16–170). One patient (Case 1) was observed for 170 days due to a high systemic risk at presentation. After her condition stabilized, she proceeded with vitrectomy (Figure 1).

In the vitrectomy group, the median initial VA at presentation was 2.15 logMAR (range 20/200 to light perception). Intraoperative findings revealed resolving vitreous hemorrhage in most eyes, with fresh hemorrhage noted in only one eye. The majority of cases (five eyes, 63%) revealed non-active PEHCR lesions, characterized by fibrosis at elevated lesions and hemoglobinized hemorrhage, while three eyes (38%) presented with fresh, active lesions. Intraoperative intravitreal anti-VEGF injections with bevacizumab were administered in three eyes with active lesions involving or threatening the macula. Endolaser photocoagulation was performed in five eyes. All eyes underwent vitrectomy for blood evacuation, and none required subretinal hemorrhage drainage. Balanced salt solution was used as the tamponade agent in all cases, with no gas or oil tamponade required.

Six patients presented with a moderate degree of vitreous hemorrhage and relatively good visual acuity (range 20/40 to 20/400), and therefore, no surgical intervention was deemed necessary. After a period of observation, spontaneous improvement in vitreous hemorrhage occurred, with median visual acuity improving to 0.2 logMAR (range 20/25 to 20/

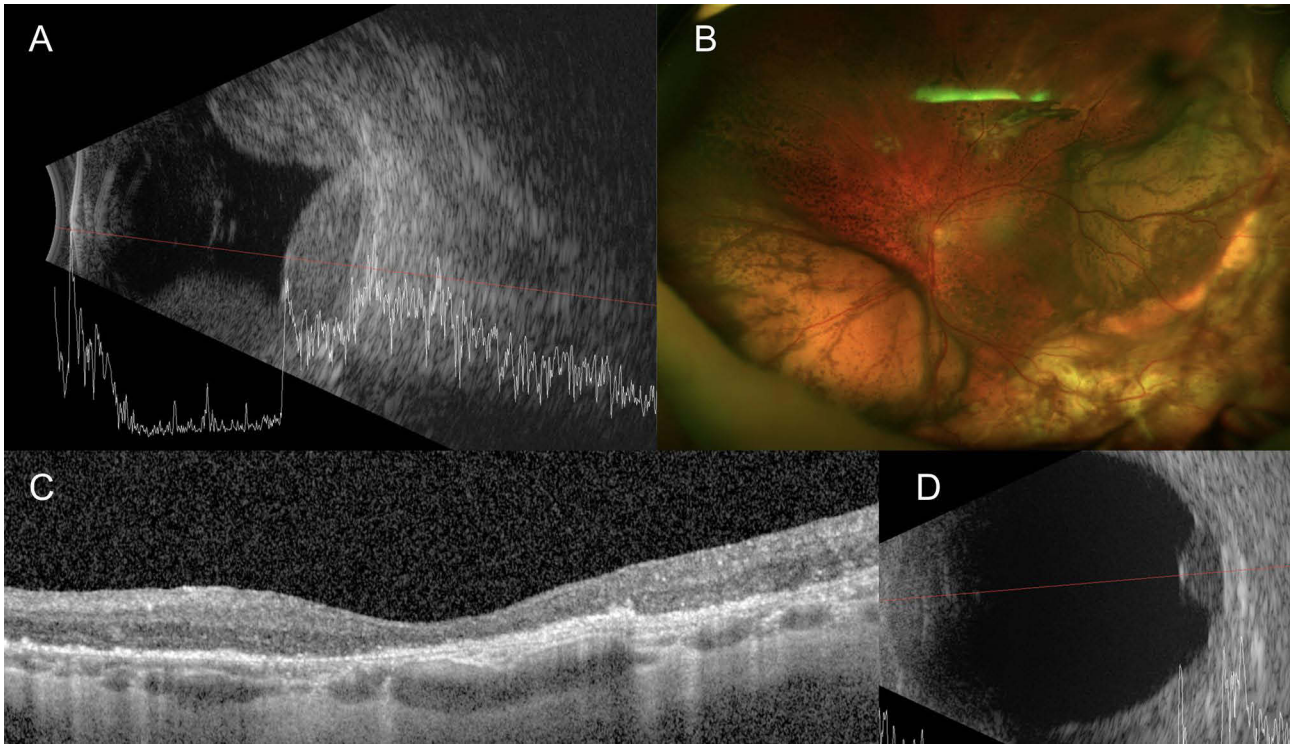

Figure 1 Case 1 (**A**) Echography of the left eye in a patient presenting with dense vitreous hemorrhage. B-scan shows multiple dome-shaped, mass-like lesions, and A-scan demonstrates moderate internal reflectivity. (**B**) Wide-field color fundus image taken one week after vitrectomy reveals multiple PEHCR lesions involving three quadrants with generalized RPE alterations. No active hemorrhage is observed. (**C**) Optical coherence tomography (OCT) of the fovea one-week post-vitrectomy shows no intraretinal or subretinal fluid. Hyperreflective thickening of the outer retina, loss of the ellipsoid zone and external limiting membrane, and pachyvessels are noted. (**D**) Five months after vitrectomy, B-scan echography shows significant regression of PEHCR lesions, reducing from multifocal to a single lesion with marked decreases in size and thickness, without any additional treatment after surgery.

50) in all cases (Figure 2). Two eyes received intravitreal anti-VEGF injections within 2 weeks after onset of symptoms: Case 10, with macula-threatening lesions, showed improvement from 20/70 to 20/50 after eight aflibercept injections. Case 12, initially at 20/400 due to macular involvement, improved to 20/40 following three bevacizumab injections, with stable lesions and no recurrence observed (Figure 3).

The median follow-up period was 11.7 months (mean 21, range, 3–63). At the final follow-up, the median VA improved to 0.36 logMAR (mean 0.7; range, 0.1–2.0) from an initial presentation of 1.3 logMAR (mean 1.3; range 0.3–2.7). Multilevel linear regression with random intercepts and random slopes was used to evaluate visual outcomes across different treatment groups. The vitrectomy group showed a significant mean VA improvement of 0.76 logMAR (range, 0.25–1.26; $p = 0.004$), while the non-vitrectomy group showed a smaller, non-significant gain of 0.18 logMAR (range, –0.77 to 0.41; $p = 0.055$). Regarding anti-VEGF injections, five of the 14 eyes received intravitreal anti-VEGF, with an average of 3.6 injections per eye (range 1–8). VA improved by 0.55 logMAR in both the injected and non-injected groups, with p-values of 0.134 and 0.042, respectively.

Of the 8 eyes without macular involvement, the mean final VA was 0.3 logMAR (range 0.1–0.4), compared to 1.4 logMAR (range 0.3–2.0) in eyes with macular involvement, though this difference was not statistically significant ($p = 0.995$). Both groups showed VA improvement after treatment, with a mean improvement of 0.55 logMAR in the non-macular involvement group ($p = 0.024$) and in the macular involvement group ($p = 0.052$). Despite these improvements, five of the 14 eyes had final VA worse than or equal to 20/200 due to permanent macular damage, confirmed by OCT, which demonstrated disruption of the outer retinal layers. During the follow-up period, regression or stability of the PEHCR lesion was observed in all of 14 eyes. Although imaging changes were less prominent than clinical findings, a trend toward increased scarring and a reduction in both the number and size of lesions was noted over time. The median lesion thickness decreased to 2.2 mm (range 0.8–3.9) from an initial 3.4 mm (range 1.5–6.8) as measured by echography. No progression, recurrence, or new lesions were observed in any of the 14 eyes in this study.

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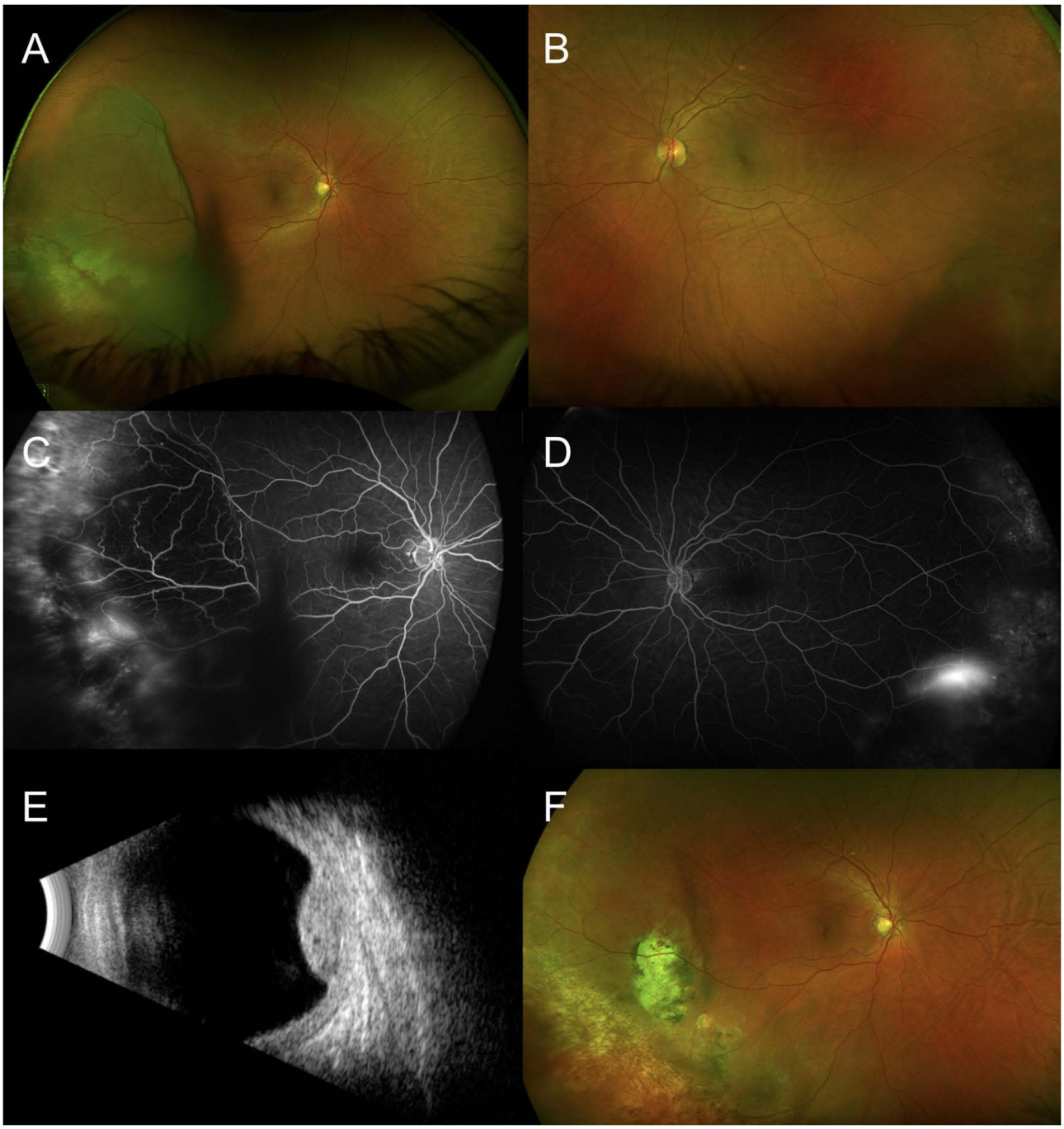

Figure 2 Case 9 (**A** and **B**) Wide-field color fundus images of both eyes at initial presentation. The right eye shows a large unifocal PEHCR lesion in the temporal area with exudation, no active hemorrhage, and no macular involvement. The left eye demonstrates an area of hemorrhage in the inferotemporal far periphery. (**C** and **D**) Wide-field fluorescein angiography (FA) reveals blockage from the lesions and hyperfluorescence due to late leakage at the peripheral areas corresponding to the lesions in both eyes. (**E**) Echography shows a dome-shaped PEHCR lesion with slight heterogeneity and no evidence of choroidal excavation. (**F**) At 2 years of follow-up without additional treatment, both eyes show stable regression of PEHCR lesions with chorioretinal atrophy and fibrosis. RPE alterations are also observed.

Discussion

This study examined 14 eyes with this rare and aggressive presentation, focusing on diagnostic approaches and visual outcomes following treatment. The mean age of patients in our series was 77 years, aligning with other studies on PEHCR.[3,9] Our cohort demonstrated a higher prevalence of systemic vascular comorbidities compared to other series,[9,11,12] with nearly all patients had a history of systemic hypertension, and approximately half had diabetes mellitus and chronic kidney disease.

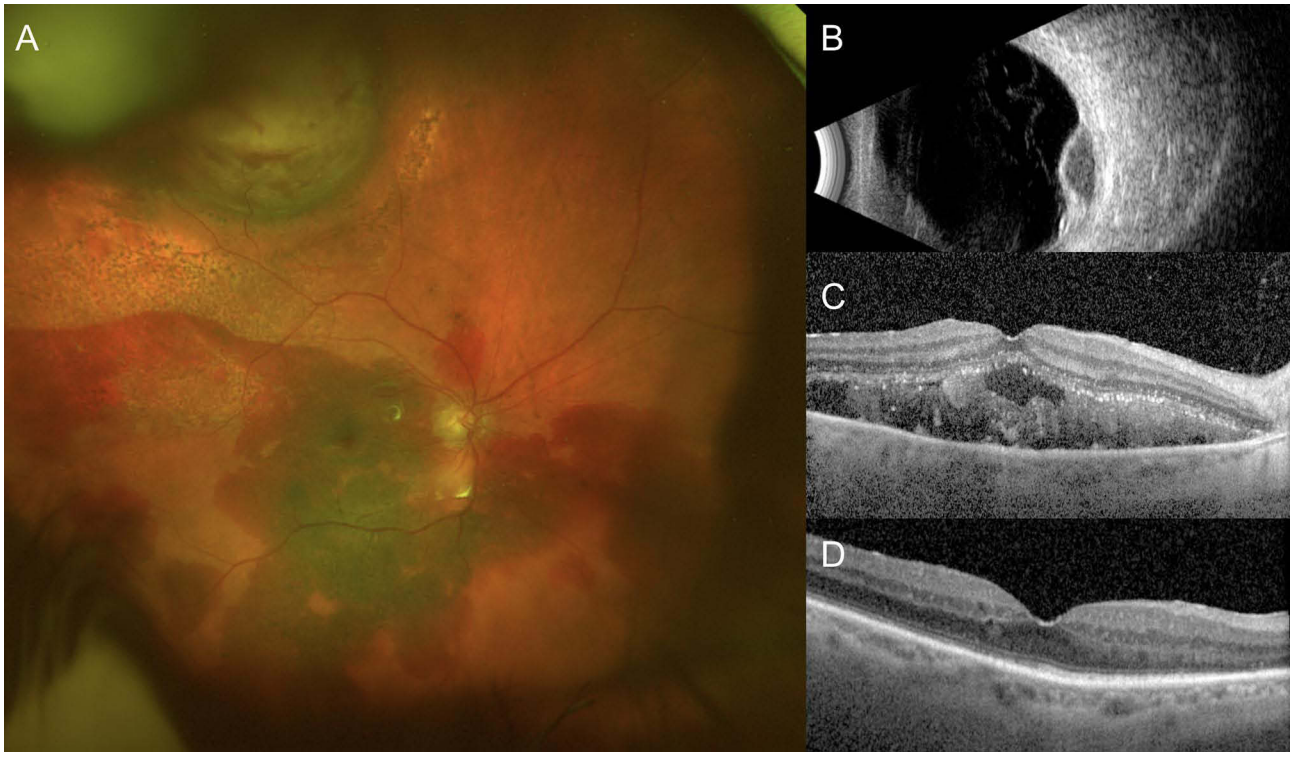

Figure 3 Case 12 (A) Wide-field color fundus image of the right eye after spontaneous improvement of vitreous hemorrhage shows a PEHCR lesion in the superotemporal region with surrounding RPE alterations. Subretinal and sub-RPE hemorrhages are present, involving the macula. (**B)** B-scan echography reveals an echolucent, mass-like lesion corresponding to the PEHCR lesion seen in A. (**C)** OCT demonstrates subretinal fluid and hyperreflective subretinal lesions corresponding to hemorrhages. (**D)** After three monthly bevacizumab injections, OCT shows resolution of the subretinal fluid and hemorrhage.

Similar to previous studies, systemic hypertension was commonly observed in about half of patients,[3,10] though the prevalence in our cohort was notably higher. This strong association with vascular risk factors may suggest a role in the pathogenesis or progression of PEHCR, particularly in cases with more aggressive presentations.

In our cohort, all patients presented with moderate to dense vitreous hemorrhage causing significant visual loss. In contrast, Shields et al reported 42% of PEHCR eyes simulating choroidal melanoma were asymptomatic, likely due to broader inclusion criteria. This selection bias in our study explains the worse presenting visual acuity (mean 1.3 logMAR) compared to prior reports.[3,9,10] Bilateral disease was observed in half of the patients in our cohort, a higher frequency than previously reported rates of 9–37%.[3,4,9,13] B-scan ultrasonography revealed a mean PEHCR lesion thickness of 3.4 mm (range 1.5–6.8 mm), slightly exceeding values reported in other studies.[3,9]

Notably, all three patients on anticoagulant therapy (either apixaban or warfarin for atrial fibrillation) presented with bilateral PEHCR and severely dense vitreous hemorrhage, necessitating PPV in each case. Additionally, three of the four patients on antiplatelet therapy (aspirin 81 mg) also developed dense vitreous hemorrhage that required PPV. Macular involvement was present in all eyes of patients on anticoagulant therapy, likely contributing to the generally poor final visual outcomes observed in this subgroup. Previous studies have reported that vitreous hemorrhage is a recognized intraocular hemorrhagic complication associated with anticoagulant or antiplatelet medications, particularly when multiple agents are used in combination.[14–17] Our findings support this association, suggesting that anticoagulant therapy may exacerbate hemorrhagic manifestations in PEHCR, potentially leading to a more aggressive disease course and a greater likelihood of requiring surgical intervention.

Of the 14 patients in our cohort, all were referred with breakthrough vitreous hemorrhage and retinochoroidal mass lesions. Previous studies have reported that vitreous hemorrhage can occur in 10–20% of PEHCR cases,[7,9,10] with vitrectomy required in up to 7%.[3] The diagnosis of PEHCR is primarily clinical, relying on careful examination of the retinal fundus. However, in our cohort, the presence of vitreous hemorrhage obscured fundus visualization, making echography essential for diagnosis. At initial presentation, echography revealed peripheral retinochoroidal mass lesions

in all cases, with a mean maximum thickness of 3.4 mm. Most lesions were located temporally (64%), between the equator and the ora serrata (57%), with extension over more than one quadrant in 8 eyes (57%). In comparison, the largest studies on choroidal melanoma have shown that melanoma thickness correlates with tumor location, with the majority of lesions located between the macula and the equator (78%) and a smaller proportion (17%) between the equator and the ora serrata. Mean melanoma thickness varied by location: 4.3 mm for tumors situated between the macula and the equator, and 7.0 mm for those between the equator and the ora serrata. Moreover, the majority of melanomas affected less than one quadrant and rarely present bilaterally.[18] Importantly, choroidal melanoma does not frequently cause breakthrough vitreous hemorrhage, which can aid in distinguishing it from PEHCR in cases of suspected malignancy.

Additionally, Ultrasonographic findings in our cases supported the diagnosis of PEHCR, with features such as clot retraction clefts, absence of intrinsic vascular pulsations or choroidal excavation, and intermediate-to-solid acoustic patterns with moderate to high reflectivity. While not pathognomonic, these findings are suggestive of PEHCR and may assist in differentiating it from choroidal melanoma. Ultrawide-field FA in our cases revealed irregular late hyperfluorescence in the peripheral retina, with 33% of cases showing late leakage that could indicate neovascularization, sometimes accompanied by persistent hypofluorescence due to masking from hemorrhagic lesions (Figure 4). Previous studies have reported peripheral neovascularization detected by FA in a wide range of 3–35% of cases.[3,4,10] This variability may result from the masking effect of large hematomas, which can obstruct the visualization of underlying choroidal neovascularization. Consequently, the extent of peripheral choroidal neovascularization contributing to PEHCR might be underestimated. Moreover, PEHCR has been suggested as a potential peripheral subtype of PCV due to shared clinical features. Recent studies utilizing ultrawide-field indocyanine green angiography (ICGA) have detected peripheral polyp-like choroidal telangiectasia and abnormal choroidal vascular networks in PEHCR.[4,5] This has led to a proposed classification system that categorizes cases based on the presence or absence of polyps.[5] According to Vandefonteyne et al, 58.3% of eyes examined with ICGA showed polyps.[10] However, in

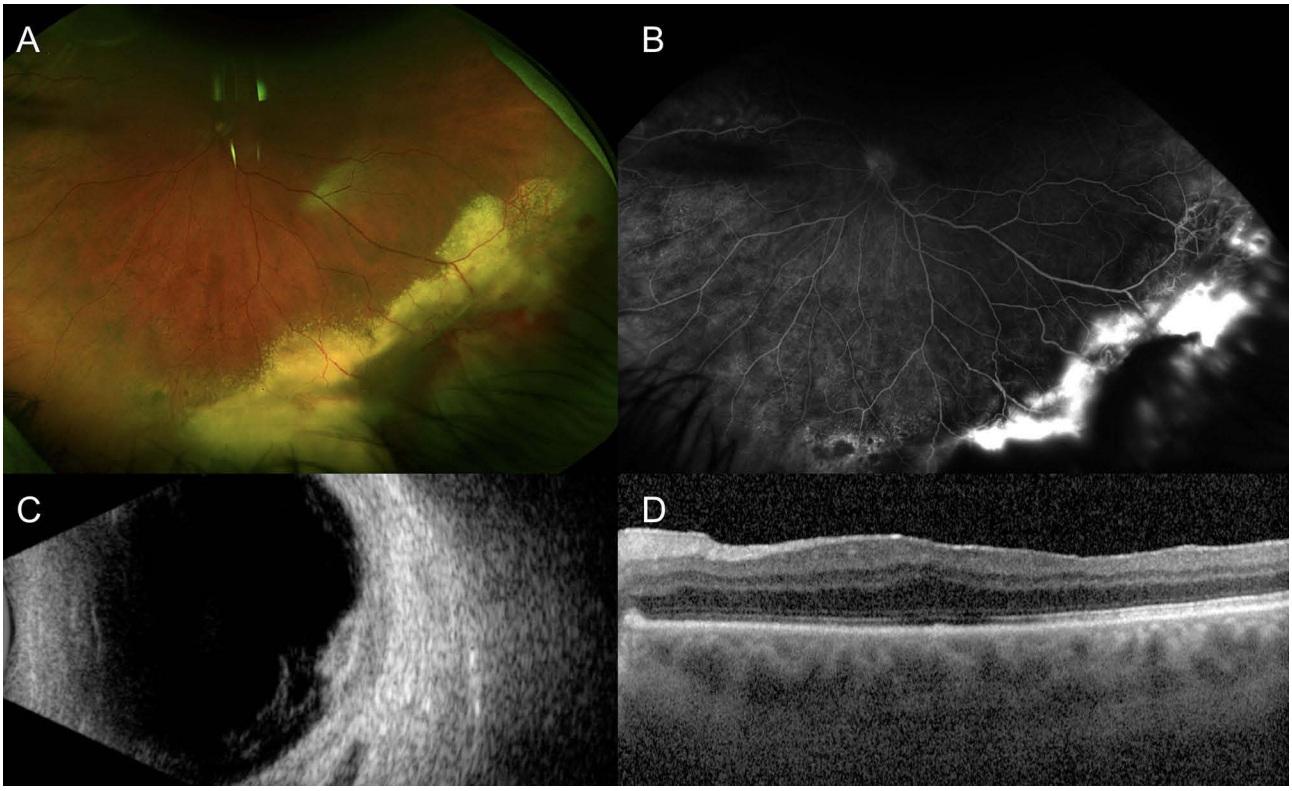

Figure 4 Case 13 (A) Wide-field color fundus image taken one week after vitrectomy shows inferotemporal lesions with subretinal and sub-RPE hemorrhage, accompanied by surrounding subretinal exudation. **(B)** Wide-field fluorescein angiography (FA) demonstrates late hyperfluorescence and leakage from peripheral choroidal neovascularization. **(C)** Echography reveals a plateau-shaped lesion with heterogeneous echogenicity. **(D)** OCT image one week after vitrectomy shows no macular involvement, intact outer retinal layers, and thickened choroidal thickness.

our series, only two patients underwent ICGA, and neither presented with polyps, which may suggest an underestimation due to potential blockage by hematomas. Given the tendency of PEHCR lesions to affect the far periphery, performing widefield ICGA could be beneficial in detecting these peripheral abnormalities more effectively.

OCT was performed in all cases, revealing maculopathy in 6 of 14 eyes. Two eyes showed active lesions with subretinal fluid, hemorrhage, and serous PED, while four exhibited chronic changes, including outer retinal loss, subretinal fibrosis, and RPE disruption. These findings reflect the progressive nature of PEHCR and its potential for irreversible macular damage. Interestingly, pachychoroid features were observed in 6 eyes (43%), which aligns with the hypothesis that PEHCR may have a pathophysiological link with pachychoroid-driven conditions such as PCV. Pachychoroid features, including a thickened choroid and dilated choroidal vessels, are often associated with increased vascular permeability, which could contribute to hemorrhagic manifestations in PEHCR. This observation raises the possibility that pachychoroid disease spectrum characteristics may predispose patients to PEHCR, potentially influencing its severity and progression. OCT provides crucial visualization in PEHCR, identifying treatable active maculopathy and chronic changes for prognosis. Its routine use supports comprehensive assessment and early detection of treatable lesions.

As previously described, the most common findings after the resolution of vitreous hemorrhage in our cohort were hemorrhagic PED and subretinal hemorrhage, which occurred at higher rates compared to other studies that included a broader spectrum of PEHCR presentations.[3,10] This suggests that these features may be associated with a higher risk of developing breakthrough vitreous hemorrhage. Similar observations have been reported in studies of PCV, where hemorrhagic PED and hemorrhagic retinal detachment were identified as significant risk factors for breakthrough vitreous hemorrhage.[19]

PEHCR typically follows a spontaneously regressive course, making observation the preferred management approach.[3,9,10] However, treatment becomes necessary in cases with breakthrough vitreous hemorrhage or active maculopathy. Suggested treatments, such as laser photocoagulation and anti-VEGF injections, have not demonstrated statistically significant benefits.[3,10] In PEHCR cases with breakthrough vitreous hemorrhage, only one small series has been reported, involving five eyes in Indian patients, all treated with vitrectomy.[20] The study reported that satisfactory visual and anatomic outcomes could be achieved with PPV to clear the hemorrhage, with no additional treatment required, even on long-term follow-up. In our study, eight eyes underwent vitrectomy, with additional intravitreal anti-VEGF injections administered in cases involving macular involvement. In this group, VA improved by an average of 0.76 logMAR (range 0.25–1.26), with statistically significant results. Additionally, laser photocoagulation was also performed in half of 14 patients, primarily as intraoperative endolaser treatment. Although there are no standardized recommendations for laser use in PEHCR, some studies suggest applying laser photocoagulation when lesions extend toward the posterior pole or to limit the progression of peripheral choroidal neovascularization.[10,21] Although cryotherapy has been reported as an adjunctive treatment for PEHCR in selected cases, its efficacy remains uncertain, with limited data available from case series.[9,10] Some studies have described its use for peripheral lesions with minimal threat to the macula; however, no significant visual benefit has been demonstrated. In our cohort, cryotherapy was not utilized, as management focused on vitrectomy, intravitreal anti-VEGF, and laser photocoagulation based on degree of hemorrhage, lesion activity and anatomical involvement.

In our cohort, anti-VEGF therapy was administered in select cases with macular involvement, either intraoperatively or during follow-up. While visual improvement was observed, the effect did not reach statistical significance, likely due to the small sample size and the presence of permanent outer retinal damage in some cases with macular involvement. This aligns with recent trends in the use of intravitreal anti-VEGF therapy for PEHCR, particularly for cases with macular-threatening lesions or substantial exudation.[9,11,21,22] Seibel et al were the first to report a case series on anti-VEGF therapy in PEHCR, emphasizing its benefit when peripheral lesions threaten the macula and recommending close monitoring similar to exudative AMD.[22] Additionally, a recent systematic review concluded that anti-VEGF is effective in managing PEHCR, particularly in cases with macular exudation, and highlighted the importance of urgent initiation to stabilize the disease.[23] In our cohort, two non-vitrectomy eyes treated within two weeks of symptom onset showed favorable responses, supporting the value of early intervention. However, pre-existing macular damage may limit recovery, underscoring the need for timely and individualized treatment.

Among the eight eyes without macular involvement, the mean final VA was 0.3 logMAR, compared to 1.4 logMAR in eyes with macular involvement. The non-macular involvement group demonstrated a significant mean improvement of 0.55 logMAR after treatment ($p = 0.024$). However, in five of the 14 eyes with macular involvement, the final VA was

worse than or equal to 20/200, primarily due to irreversible macular damage. Poor visual outcomes have also been documented in untreated submacular hemorrhages associated with conditions such as PCV or AMD, supported by experimental evidence showing that subretinal blood can lead to retinal toxicity and damage.[24–26] These findings emphasize the importance of early intervention in cases with submacular hemorrhage to minimize the risk of permanent visual impairment and optimize outcomes.

During a median follow-up of 11.7 months, none of the 14 eyes in this study showed progression or new lesions. These results align with other studies, which report a high rate of disease regression, reaching nearly 90%.[3,9] Based on current literature, PEHCR appears to have a generally favorable prognosis for most patients, particularly in the absence of macular involvement.

PEHCR should be considered in the differential diagnosis of vitreous hemorrhage associated with a choroidal mass to ensure accurate management and avoid misdiagnosis. Differentiating PEHCR cases with breakthrough vitreous hemorrhage from other choroidal pathologies, such as melanoma, can be challenging but is essential for appropriate treatment. Given the rarity of this disease and its uncommon presentation, along with the limited sample size in our study, further research with larger cohorts is needed to establish standardized treatment protocols and to evaluate the role of adjunctive therapies in managing severe presentations of PEHCR.

Conclusion

PEHCR with breakthrough vitreous hemorrhage represents a rare but significant manifestation. Vitrectomy and intravitreal anti-VEGF therapy appear to enhance visual outcomes in patients with breakthrough vitreous hemorrhage secondary to PEHCR. However, visual recovery may be limited in cases with macular involvement due to irreversible retinal damage, underscoring the importance of early intervention and individualized management strategies.

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