• Clinical Research •

Peripheral exudative hemorrhagic chorioretinopathy as a variant of polypoidal choroidal vasculopathy in a case series of Chinese patients

Jue-Xue Wang[1,2,3,4], *Nan Zhou*[1,2,3,4], *Li-Hong Yang*[5], *Wen-Bin Wei*[1,2,3,4]

[1]Beijing Tongren Eye Center, Beijing Ophthalmology & Visual Sciences Key Lab, Beijing Tongren Hospital, Capital Medical University, Beijing 100730, China
[2]Beijing key Laboratory of Intraocular Tumor Diagnosis and Treatment, Beijing Tongren Hospital, Capital Medical University, Beijing 100730, China
[3]Medical Artificial Intelligence Research and Verification Key Laboratory of the Ministry of Industry and Information Technology, Beijing Tongren Hospital, Capital Medical University, Beijing 100730, China
[4]Beijing Key Laboratory of Intelligent Diagnosis, Treatment and Prevention of Blinding Eye Diseases, Beijing Tongren Hospital, Capital Medical University, Beijing 100730, China
[5]Beijing Tongren Hospital, Beijing 100730, China
Correspondence to: Wen-Bin Wei. Beijing Tongren Eye Center, Beijing key Laboratory of Intraocular Tumor Diagnosis and Treatment, Beijing Ophthalmology & Visual Sciences Key Lab, Medical Artificial Intelligence Research and Verification Key Laboratory of the Ministry of Industry and Information Technology, Beijing Key Laboratory of Intelligent Diagnosis, Treatment and Prevention of Blinding Eye Diseases, Beijing Tongren Hospital, Capital Medical University, Beijing 100730, China. weiwenbintr@163.com
Received: 2025-08-28 Accepted: 2025-11-13

Abstract

● **AIM:** To evaluate the clinical features, diagnosis, treatment, and outcome of peripheral exudative hemorrhagic chorioretinopathy (PEHCR), a variant of polypoidal choroidal vasculopathy (PCV), in a case series of Chinese patients.

● **METHODS:** This study was retrospectively conducted from September 2018 to March 2025. Clinical examinations included color fundus photography, B-scan ultrasonography, fluorescein angiography (FA), indocyanine green angiography (ICGA), swept-source optical coherence tomography (SS-OCT), and optical coherence tomography angiography (OCTA), and two active or inactive subgroups and misdiagnosed cases were analyzed.

● **RESULTS:** Totally 19 patients (21 eyes) with a mean age of 54.3±9.4 (range, 36–68)y were included, with a majority of women (*n*=13, 68.4%). The mean follow-up period was 13±1.4 (range: 1–57)mo. Decreased visual acuity was the most frequent initial manifestation (17 eyes, 84.2%), and lesions were mainly distributed in the inferotemporal or temporal quadrant (14 eyes, 66.7%), with choroidal polyps and branching neovascular networks revealed by OCTA and ICGA. Nine patients had been previously misdiagnosed with choroidal melanoma, and 6 of them had massive vitreous hemorrhage (VH). PEHCR manifested along a spectrum ranging from active or inactive subretinal hemorrhagic forms to chronic fibrotic or atrophic forms. One patient experienced natural regression. Ten eyes received a mean of 4.7±1.1 (range: 3–7) intravitreal anti-vascular endothelial growth factor (VEGF) injections, two eyes underwent vitrectomy, and six eyes were treated with vitrectomy combined with anti-VEGF therapy. Best-corrected visual acuity (logMAR) in treated eyes (18 eyes) improved to 0.31±0.25 from the baseline of 1.50±0.75 (*P*<0.001).

● **CONCLUSION:** PEHCR is a variant of PCV. Chinese patients with PEHCR have a relatively younger age of onset. Anti-VEGF injections and/or vitrectomy are treatment options for lesion regression or dense VH to gain better visual outcomes.

● **KEYWORDS:** peripheral exudative hemorrhagic chorioretinopathy; peripheral polypoidal lesion; anti-vascular endothelial growth factor; Chinese

DOI:10.18240/ijo.2026.02.13

Citation: Wang JX, Zhou N, Yang LH, Wei WB. Peripheral exudative hemorrhagic chorioretinopathy as a variant of polypoidal choroidal vasculopathy in a case series of Chinese patients. *Int J Ophthalmol* 2026;19(2):312-319

INTRODUCTION

Peripheral exudative hemorrhagic chorioretinopathy (PEHCR) was initially described as a hematoma peripheral to the macular region by Reese and Jones[1] in 1961. Annesley[2] subsequently coined the term for this condition in 1980, which was characterized by hemorrhages in the subretinal or sub-retinal pigment epithelial (sub-RPE) space.

Previous studies have reported that PEHCR most likely affects older Caucasian women, and 31% of patients present with bilateral involvement[2-3]. The mean patient age ranged from 63.4 to 83y[3-5]. PEHCR is commonly asymptomatic and experiences natural regression, but it still has the potential to cause vision loss with dense vitreous hemorrhage (VH)[3].

The exact etiology of PEHCR has not yet been identified. Recent studies have found peripheral polyps on indocyanine green angiography (ICGA) in PEHCR and hypothesized that it could share a similar pathophysiological process with polypoidal choroidal vasculopathy (PCV) and be included in pachychoroid spectrum diseases[6-8]. PEHCR is a rare clinical entity that is easily underdiagnosed due to the challenges of peripheral retinal examination and failure to identify it in asymptomatic patients. It is often incorrectly identified as choroidal melanoma (CM) with a confusing, lump-like appearance. Approximately 13%-50% of patients are misdiagnosed with CM[2-3]. It is also the second most common pseudomelanoma, following choroidal nevus[9]. Given that the outcome and management of PEHCR are significantly different from those of choroidal tumors, a timely and accurate diagnosis is essential. Nowadays, there is no consensus regarding the treatment for PEHCR. In previous studies, most patients experienced stability or regression, and various therapeutic approaches have been attempted in cases of vision loss, active hemorrhage, and VH, including anti-vascular endothelial growth factor (VEGF) injection, laser photocoagulation, cryotherapy, and vitrectomy[2-3,10-12].

PEHCR has not been reported so far in China. The existing understanding is primarily dependent on case reports[3-4]. We conducted a retrospective analysis of patients diagnosed with PEHCR at Tongren Hospital over seven years in China. The main objective of this study is to investigate the general characteristics and clinical manifestations of PEHCR in Chinese patients. Therapeutic management and the corresponding outcomes were also evaluated.

PARTICIPANTS AND METHODS

Ethical Approval This retrospective case series included patients diagnosed with PEHCR at Beijing Tongren Hospital between September 14, 2018, and March 6, 2025. This study complied with the tenets of the Declaration of Helsinki and was approved by the Institutional Review Board and Medical Ethics Committee at Beijing Tongren Hospital (TRECKY2018–056-GZ (2022)−07).

Participants The inclusion criteria were patients diagnosed with PEHCR based on the presence of choroidal and retinal peripheral lesions caused by subretinal or sub-RPE hemorrhage. The location of the peripheral region was beyond the vortex vein ampullae, and the maculae were not involved. The exclusion criteria were other ocular or systemic diseases leading to subretinal hemorrhage or exudation, such as CM, osteoma, metastatic tumors, wet age-related degeneration, uveitis, and intraocular inflammation, which were all carefully and differentially diagnosed.

Demographic Information and Clinical Examinations Basic patient information, medical history, and family history were obtained during the first consultation. Paraclinical examinations included color fundus photography, B-scan ultrasonography, fluorescein angiography (FA), ICGA, swept-source optical coherence tomography (SS-OCT), and optical coherence tomography angiography (OCTA). Each patient's diagnosis was confirmed based on the combination of the characteristics of the lesions observed by fundus examination and the typical vascular anomalies observed on angiography, which presented as polyps or abnormal vascular morphological networks. All suspected patients underwent B-scan ultrasonography and positron emission tomography computerized tomography (PET-CT) to rule out CM, based on its specific patterns: mushroom shape, ciliary body extension, and choroidal excavation. B-scan ultrasonography also helped evaluate patients with VH, whose fundus examinations were obscured. SS-OCT and OCTA were critical in locating lesions and in the follow-up assessment of their activity.

Treatment and Follow-up The therapy and outcomes of each patient were also analyzed. For eyes with active subretinal or sub-RPE hemorrhagic lesions, intravitreal anti-VEGF therapy with either ranibizumab 0.5 mg or aflibercept 2 mg was recommended. Follow-up visits were scheduled for the first day and one month after injection to monitor changes in lesion activity and hemorrhage absorption. If no significant improvement was observed after three consecutive injections, the dose was increased to ranibizumab 1.0 mg or aflibercept 3.0 mg. Follow-up intervals were tailored according to individual clinical stability.

Vitrectomy was performed on eyes with dense VH that obscured the fundus view and showed no improvement for 1–2wk. All procedures were performed at Beijing Tongren Hospital by the same skilled vitreoretinal surgeon. Postoperative evaluations were scheduled for the first day, one week, and one month. Additional anti-VEGF injections were administered if active lesions were detected during the follow-up period.

Visual acuity and ocular complications were recorded during each visit. Clinical improvement and treatment outcomes were assessed using fundus photography, SS-OCT, OCTA, and B-scan ultrasonography, when required.

Statistical Analysis Statistical analysis was conducted using SPSS software (version 27.0; IBM Corp., Armonk, NY, USA). Best-corrected visual acuity (BCVA) was documented using Snellen notation and converted to the logarithm of the

Table 1 General information of patients with peripheral exudative hemorrhagic chorioretinopathy

Case	Gender	Age, y	Affected eye	Baseline BCVA	Lesion location	VH	Baseline diagnosis	Treatment	Last-visit BCVA
1	M	61	OS	FC	Multifocal	Yes	ARN	IV	20/66
2	F	41	OD	FC	Temporal lower	Yes	CM	PPV	20/200
3	M	45	OD	20/66	Upper nasal	Yes	CM	PPV+IV	20/30
4	F	45	OS	20/25	Temporal lower	No	CM	Observation	20/25
5	F	59	OS	FC	Temporal lower	Yes	CM	PPV+IV	20/100
6	M	43	OU	HM (OS)	Temporal lower (OU)	Yes	CM	PPV+IV (OS)	20/100
7	M	65	OU	FC (OU)	Temporal (OU)	Yes	CM	IV (OD), PPV (OS)	20/30 (OU)
8	F	62	OS	FC	Upper nasal	No	PEHCR	IV	20/30
9	F	52	OD	HM	Temporal	Yes	VH	PPV+IV	20/30
10	F	67	OD	20/66	Temporal	No	PEHCR	IV	20/30
11	F	53	OS	20/80	Upper	No	PEHCR	IV	20/40
12	F	50	OD	HM	Temporal	No	PEHCR	IV	20/40
13	F	65	OS	20/66	Temporal	No	PEHCR	IV	20/30
14	F	56	OD	20/200	Multifocal	No	Choroidal matastsis	IV	20/25
15	F	53	OS	20/80	Lower nasal	No	CM	IV	20/40
16	F	68	OD	20/100	Temporal	No	PEHCR	IV	20/40
17	F	36	OS	HM	Temporal	Yes	VH	PPV+IV	20/25
18	M	63	OS	HM	Temporal lower	Yes	CM	Unknown	Unknown
19	M	48	OD	20/400	Upper nasal	Yes	VH	PPV+IV	20/25

F: Female; M: Male; OD: Right eye; OS: Left eye; OU: Both eyes; FC: Finger counting; HM: Hand motion; BCVA: Best corrected visual acuity; VH: Vitreous hemorrhage; ARN: Acute retinal necrosis; CM: Choroidal melanoma; PEHCR: Peripheral exudative hemorrhagic chorioretinopathy; IV: Intravitreal injections of anti-vascular endothelial growth factor; PPV: Pars plana vitrectomy.

minimum angle of resolution (logMAR) for statistical analysis. logMAR values were assigned to the severe vision impairment situation as follows: 2.0 for finger counting (FC) and 2.3 for hand motion (HM). Data are presented as the mean±standard deviation, and the Wilcoxon signed-rank test was used for data analysis. Statistical significance was defined as $P<0.05$.

RESULTS

Demographic Information and Baseline Examinations

This study included 19 patients (21 eyes). General patient characteristics are shown in Table 1. The patients had a mean age of 54.3±9.4 (range:36-68)y, with women accounting for the majority (68.4%, 13/19). Bilateral involvement was found in two patients. The mean follow-up duration was 13±1.4 (range: 1–57)mo. All 19 patients were Chinese. Most patients did not have systemic comorbidities (89.5%, 17/19); only two patients had systemic hypertension. Decreased vision was the most common initial symptom, accounting for 85% (17/20) of eyes, followed by visual field obstruction, photopsia, and darkness. The mean BCVA value at the first visit was 1.47±0.77 logMAR (20 eyes). Specifically, BCVA (logMAR) was ≤0.30 in one eye, 0.30–1 in 7 eyes, and >1 in 12 eyes.

On fundus examination, the lesions were located in the supranasal (3 eyes), inferonasal (1 eye), and mainly in the temporal or inferotemporal (14 eyes, 66.7%) quadrants of the peripheral retina. The lesions were between the vortex vein ampullae and ora serrata in 12 eyes, and between the macula and vortex vein ampullae in 9 eyes, with the posterior margin of the lesions extending to the macular region in 4 eyes (23.5%, 4/17). The total circumferential extent of the lesions had an average range of 2.7±1.8 clock hours (median: 2.5, range: 0.5–7; n=17 eyes). The lesions appeared yellow-white in 10 eyes, orange-colored in two eyes, non-pigmented in two eyes, and were accompanied by uneven brownish pigmentation in three eyes. Subretinal and sub-RPE hemorrhages were the most common findings and were detected in all eyes by SS-OCT, followed by retinal exudation or drusen in 4 eyes (4/16). Various amounts of VH were observed in 10 eyes, with the fundus obscured in eight eyes. FA and ICGA were performed on 12 patients. Blocked fluorescence was observed, followed by fluorescein staining in the lesion at the late phase of FA. Abnormal choroidal vascular networks were observed during the late phase of ICGA. Peripheral polypoidal lesions on ICGA were observed in 10 of 12 patients (83.3%).

Diagnosis, Treatment and Clinical Course Nine patients were referred to our hospital with misdiagnosed CM (Table 1), and six of them had massive VH. The misdiagnosed lesions mostly appeared as yellowish-white, elevated masses, with or without fresh hemorrhage (Figure 1A). B-scan ultrasonography revealed inhomogeneous reflectivity of low-to-moderate intensity with or without a blood signal and contrast agent inside the lesion (Figure 1B). OCTA revealed polypoid choroidal telangiectasia (Figure 1C).

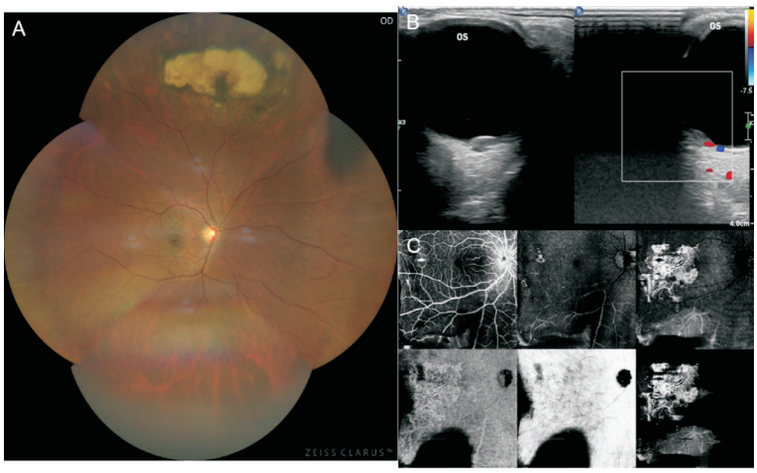

Figure 1 Representative images of patients misdiagnosed with choroidal melanoma A: Color fundus photograph showing a yellowish-white elevation in the upper nasal quadrant of the right eye (Case 3); B: B-scan ultrasonography showing a localized elevated lesion on the inferior sclera of the left eye, with hypoechoic to isoechoic signal and contrast agent inside the lesion, without blood flow signals (Case 4); C: SS-OCTA showing polypoid choroidal telangiectasia (Case 14). SS-OCTA: Swept-source optical coherence tomography angiography.

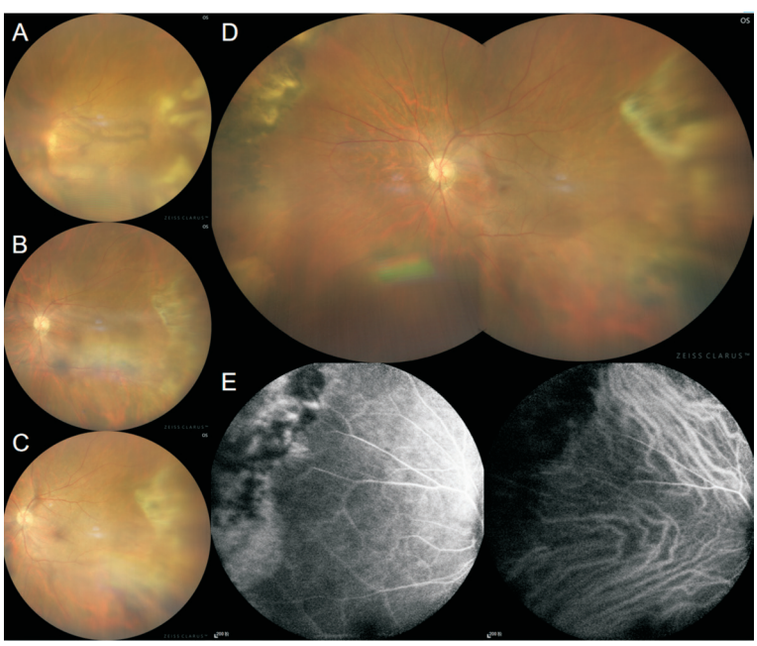

Figure 2 Color fundus photographs and FA/ICGA of Case 1 A 61-year-old man presented with decreased vision, redness, and pain. Multiple focal yellow-white retinal lesions were noted in the periphery, and he was diagnosed with acute retinal necrosis syndrome. Aqueous humor testing revealed that all tested viral nucleic acid tests were negative. FA and ICGA were conducted, and he was identified as PEHCR. It respectively showed the conditions of the patient at the first visit (A), after two injections of ranibizumab (B), after three injections (C), and after 6mo of the fifth injection (D). FA showed hyperfluorescent leakage, and ICGA revealed masking fluorescence of corresponding areas with polypoidal lesions at the edge (E). FA: Fluorescein angiography; ICGA: Indocyanine green angiography; PEHCR: Peripheral exudative hemorrhagic chorioretinopathy.

Two patients with multifocal lesions were misdiagnosed with acute retinal necrosis and choroidal metastases. The patient misdiagnosed with acute retinal necrosis is shown in Figure 2. The patient received intravitreal injections of ranibizumab (IVR). The VH was significantly resolved by subsequent follow-up. The patient underwent IVR five times in total, once a month. The lesions remained stable for more than six months, and vision improved to 20/66 at the last follow-up.

Based on observation of the cases in our series, PEHCR could be divided into two types according to the characteristics of its clinical course: inactive hemorrhagic type and active hemorrhagic type. The active hemorrhagic form was predominant in 20 eyes at baseline. PEHCR manifested along a spectrum ranging from active or inactive hemorrhagic forms to chronic fibrotic or atrophic forms.

Of 19 eyes in the series, only one (Case 4) underwent

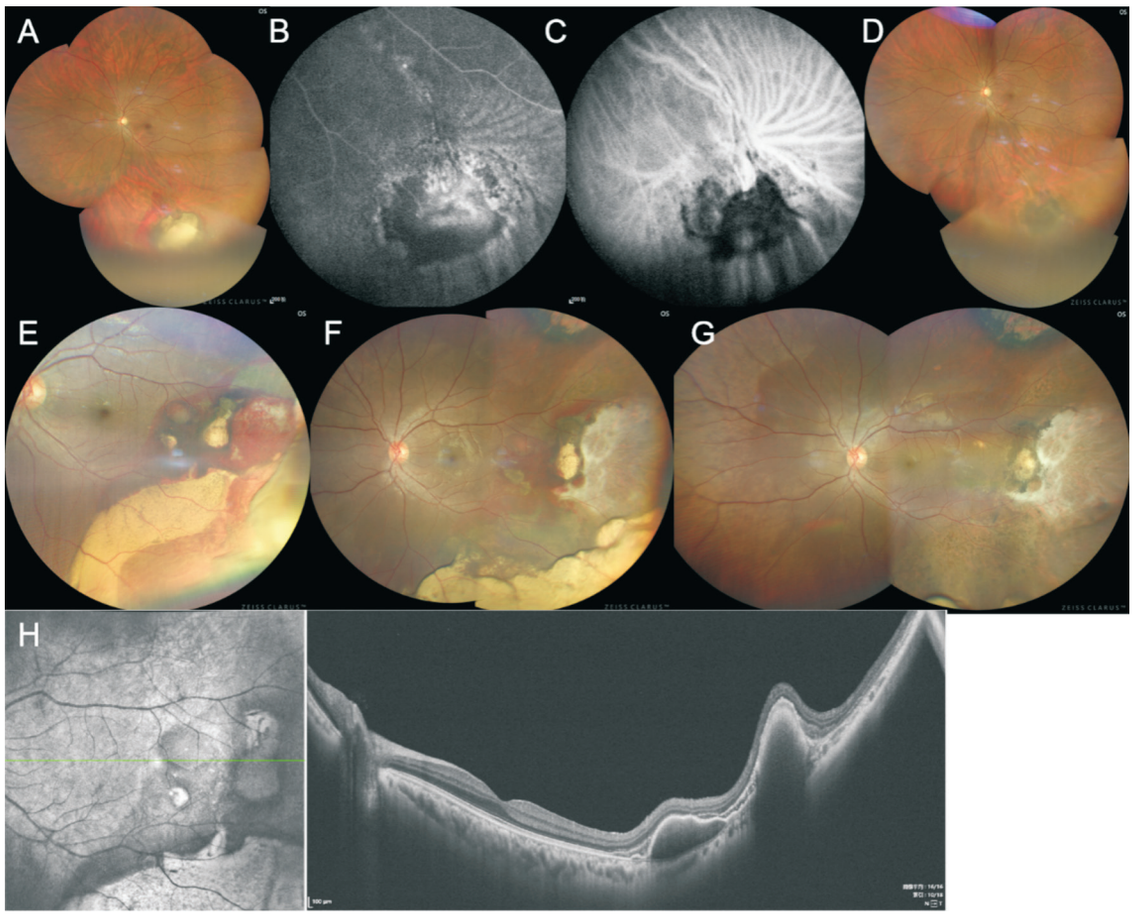

Figure 3 Representative images of patients with inactive hemorrhagic type and active hemorrhagic type A-D: A 45-year-old female patient (Case 4) experienced natural regression. Color fundus photography showing a yellowish-white elevated lesion with a small amount of hemorrhage (A); FA showing irregular hyperfluorescence at the corresponding lesion (B), and ICGA showing a polypoid hyperfluorescent lesion (C). After three months of follow-up, the accumulated hemorrhage had been absorbed, leaving residual pigmented scar lesions (D). E-H: A 36-year-old female patient (Case 17) received combined therapy. The fundus photo after vitrectomy revealing a large yellowish-white elevated lesion in the temporal retina, accompanied by a large amount of hemorrhage (E); the lesion regressed, and hemorrhage was absorbed one month after IVR (F); five months after three injections, the accumulated hemorrhage had been absorbed, leaving behind RPE degeneration and atrophy lesions (G); SS-OCT showing sub-RPE hemorrhage (H). FA: Fluorescein angiography; ICGA: Indocyanine green angiography; IVR: Intravitreal injections of ranibizumab; RPE: Retinal pigment epithelium; SS-OCTA: Swept-source optical coherence tomography angiography.

observation. This eye showed a yellowish-white elevated lesion with a small amount of subretinal hemorrhage (Figure 3A). FA (Figure 3B) revealed irregular hyperfluorescence in the corresponding lesion, and ICGA (Figure 3C) showed a polypoid hyperfluorescent lesion. After three months of follow-up, the accumulated hemorrhage was absorbed, leaving residual pigmented scar lesions (Figure 3D).

We performed three treatment options for 18 eyes: anti-VEGF injection in 10 eyes, vitrectomy in 2 eyes, and vitrectomy combined with anti-VEGF therapy in 6 eyes. There were significant differences in BCVA between baseline and the last follow-up in all treated patients (1.50±0.75 *vs* 0.31±0.25, $P<0.001$). The subretinal or sub-RPE hemorrhage was resolved at subsequent follow-up, and only residual yellowish, flattened hemorrhagic foci remained.

In total, 16 eyes received anti-VEGF injections, of which 13 were treated with aflibercept and 3 with ranibizumab. Anti-VEGF mono-therapy was the most frequently used therapy and administered in patients with obviously decreased vision, a little VH that did not obscure the fundus view, and fresh hemorrhage, with a mean of 4.7±1.1 injections delivered. BCVA significantly improved after treatment (1.22±0.75 *vs* 0.25±0.12, $P=0.005$).

Vitrectomy was performed in 2 eyes with visual acuity of FC and FC before surgery, and 20/200 and 20/30 after surgery, respectively. Vitrectomy combined with anti-VEGF injections was performed in 6 eyes. BCVA in the combined therapy group revealed a significant visual improvement (1.79±0.73 *vs* 0.33±0.29, $P=0.028$). The typical fundus photo after vitrectomy revealed a large yellowish-white elevated lesion, accompanied by a large amount of fresh hemorrhage (Figure 3E). Subsequently, the patient received IVR. The lesions

regressed during the follow-up period, and RPE degeneration and atrophy lesions occurred (Figure 3F, 3G). SS-OCT identified that the accumulated residual hemorrhage was located sub-RPE layer (Figure 3H).

DISCUSSION

The present study revealed that PEHCR in Chinese patients had a mean age of 54.3y and predominantly affected women. Most of the patients did not have systemic comorbidities. The most common initial symptom was a decreased BCVA. The lesions presented as typical subretinal or sub-RPE hemorrhages and were mainly located in the inferotemporal and temporal quadrants. Notably, more than half of the patients were referred for an incorrect diagnosis of a malignant choroidal tumor. All patients, except one, received treatment with anti-VEGF injections or vitrectomy and had a good vision outcome.

Consistent with previous findings, the majority of affected individuals were female, accounting for over 65%, whereas the sample size in this study was insufficient to determine a sex predilection[13-14]. Notably, the average age of onset was significantly younger than that reported in Caucasians. As previously reported, PEHCR occurred predominantly in elderly Caucasian patients with a mean age of over 70y[3,11,13]. Hypertension, hypercholesterolemia, and diabetes are the most frequently reported metabolic risk factors for PEHCR[13,15]. However, corresponding to the younger onset age, systemic risk factors in Chinese patients were not obvious, with only two patients having hypertension. Most patients presented with poor vision, and ocular symptoms were observed in all patients. In contrast, asymptomatic patients accounted for 26%–89% in previous studies[3,15]. This could be caused by the high proportion of the active hemorrhagic type at baseline. Almost half of the patients (47.6%) were affected by VH. This feature is consistent with findings reported in Koreans and Indians[4,14-15].

PEHCR presents with various clinical manifestations, including subretinal hemorrhage and VH, sub-RPE hemorrhage or RPE alterations or detachments, drusen and retinal exudation, and exudative retinal detachment. In this study, the most prevalent manifestations were subretinal and sub-RPE hemorrhage. Retinal exudation and drusen were not common in this study, accounting for only 25% of the eyes. This proportion was similar to that observed in Shields *et al*' study[3]. Single isolated lesions were most frequently observed (85.7%, 18/21), with the temporal or inferior temporal quadrant being the dominant location. This is consistent with the findings of previous studies[12-13].

Based on this study, we concur with Shroff *et al*[6] that PEHCR is a peripheral variant of PCV. ICGA is the gold standard for diagnosing PCV, revealing typical polypoidal hyperfluorescent lesions and a branching neovascular network[16]. In our analysis, over 80% of patients demonstrated typical polypoidal hyperfluorescent lesions on ICGA. Some small polypoidal lesions were not observed in the remaining patients, possibly because of their peripheral location.

As PEHCR is a benign disease, it is critical to differentiate it from choroidal tumors. In this study, we summarized the following causes for the 11 misdiagnosed patients: more than half of the patients had dense VH, which interfered with fundus examination, and lesions were located in the extremely peripheral area of the retina, making them difficult to observe. Subretinal lesions, with highly misleading appearances, may present as single or multiple elevated masses or as multiple scattered yellow-white plaque-like lesions in the retinal periphery, mimicking retinal necrotic foci. Typical features of multimodal imaging assist in differentiating PECHR from CM. SS-OCT confirmed that the hemorrhage was located sub-RPE layer, and pigment epithelial detachment was observed. Statistical analysis verified that pigment epithelial detachment, as one of the diagnostic features of PEHCR, exhibited a high level of sensitivity [0.93; 95% confidence interval (CI) 0.80–0.98][11]. The typical mushroom shape, ciliary body extension, and choroidal excavation sign on B-scan were most specific (1.00; 95%CI 0.91–1.00) for reliable differentiation between PEHCR and CM[11].

In our series, PEHCR lesions were divided into two types: inactive and active hemorrhagic. Two distinct forms were observed during the clinical course: the active form could transition to the inactive form, and both may either resolve or progress to stable fibrotic or atrophic lesions. It has been reported that the resolution of subretinal hemorrhage at different stages is a characteristic of PEHCR rather than CM[17]. It should be noted that regression indicates gradual absorption of residual subretinal and/or sub-RPE hemorrhage, but follow-up observations are still necessary due to the uncertainty of recurrent active hemorrhage. This classification is based on the current activity of the lesion and does not represent a long-term state.

This study provides new insights into PEHCR treatment. To date, there is no consensus on the treatment for PEHCR owing to spontaneous regression in some patients. In Shields *et al*'s study[3], lesions in 89% of patients experienced regression or stability, and none received anti-VEGF treatment. This is consistent with Goldman *et al*'s study[7], in which most eyes experienced a benign course with spontaneous resolution. However, anti-VEGF treatment was reported to exhibit favorable outcomes in cases of macular involvement, and early intervention was suggested before the macula was affected by the long-term visual results[18]. In our study, only one female patient experienced spontaneous regression. Most patients received anti-VEGF treatment with significantly improved

Table 2 Literature review for reports of peripheral exudative hemorrhagic chorioretinopathy

Author, y	Eyes/patients	Race	Gender (M/F)	Mean age, y	Treatment			
					Anti-VEGF	Vitrectomy	Natural regression	Others[a]
Annesley[2], 1980	32/27	Caucasian 96%, African 4.00%	44.4%/55.6%	69.9		6.25%	84.4%	9.35%
Shields[3],2009	173/146	Caucasian 99%, Asian 1%	33%/67%	80			89%	
Mantel[19], 2009	56/45	Caucasian 100%	31.1%/68.9%	77.3		Not mentioned		
Kim[20], 2010	4/4	Asian 100%	0/100%	76	25%	25%	25%	25%
Mantel[8], 2012	48/40	Caucasian 100%	45%/55%	78.1		Not mentioned		
Goldman[7], 2013	10/8	Caucasian 75%, African 25%	87.5%/12.5%	70	30%	30%	40%	
Seibel[18], 2016	9/8	Not clear	87.5%/12.5%	76	66.7%	33.3%		
Cebeci[21], 2016	21/12	Not clear	33.3%/66.7%	82.4	23.8%		76.2%	4.8%
Glatz[22], 2017	8/8	Not clear	37.5%/62.5%	81.6		75%	12.5%	
Goel[4], 2019	5/5	Asian 100%	60%/40%	63.4		100%		
Vandefonteyne[12], 2020	84/69	Caucasian 96.2%, African 3.8%	not clear	77.4	36.6%	19.50%	31.8%	34.2%
Zicarelli[5], 2021	50/35	Caucasian 100%	26%/74%	83	62%		36%	38%
Shroff[6], 2021	14/9	Not clear	44.4%/55.6%	75.1		Not mentioned		
Larrea[13], 2022	39/23	Not clear	26.1%/73.9%	79.3	38.5%	15.4%	35.9%	10.3%
Safir[10], 2022	35/32	Not clear	43.75%/56.25%	79	31.43%		68.57%	
Choi[23], 2023	30/30	Not clear	27%/73%	68		Not mentioned		
Choi[14], 2023	33/29	Asian 100%	24.1%/75.9%	70.9	51.5%	39.4%	21.2%	
Sodhi[11], 2023	40/40	Caucasian 93%, African 8%	43%/58%	80	5%	5%	90%	
Lee[15], 2025	43/36	Asian 100%	33%/67%	70.1	56.8%	18.9%	14%	2.7%
Kritfuangfoo[24], 2025	14/14	Caucasian 78.6%, African 21.4%	35.7%/64.3%	77	35.7%	57.1%	28.6%	35.7%

[a]Others: Laser photocoagulation, cryotherapy, and other treatment. M/F: Male/female; VEGF: Vascular endothelial growth factor.

BCVA. Patients with thick VH underwent vitrectomy and had satisfactory visual outcomes. Therefore, we believe that anti-VEGF injections are beneficial for lesion regression and better visual outcomes in patients with PEHCR. For patients with dense VH, vitrectomy as a treatment option can achieve better vision.

Our initial literature search yielded 75 studies, of which 20 were finally included. Studies with a sample size of three or fewer and articles not in English were excluded. The results are summarized in Table 2[2-8,10-15,18-24]. Among the included studies, 308 patients (366 eyes) were reported. The largest sample size was obtained from a study by Shield *et al*[3], which enrolled 143 patients (173 eyes), with Asian patients accounting for only 1%. Four studies specifically focused on Asian patients, involving 74 patients (86 eyes), including one conducted in India and three in Korea. Additionally, three studies conducted in Türkiye, India, and Korea, including a total of 51 patients (65 eyes), did not explicitly specify the ethnic distribution, and they likely predominantly involved Asian individuals. Notably, no studies have been reported in China. This study fills this gap by providing the first report of Chinese patients with this condition.

Our study has several limitations. Obviously, it had a relatively small sample size and an inherently retrospective design. The period of follow-up was not long enough. The genetic background and environmental risk factors were not thoroughly investigated. However, it established a valuable foundation for further research on pathogenesis and optimal treatment strategies for PEHCR.

ACKNOWLEDGEMENTS

Data Availability Statement: All data relevant to the study are included in the article.

Foundations: Supported by the National Natural Science Foundation of China (No.82220108017; No.82141128); The Capital Health Research and Development of Special (No.2024-1-2052); Science & Technology Project of Beijing Municipal Science & Technology Commission (No.Z201100005520045); Sanming Project of Medicine in Shenzhen (No.SZSM202311018).

Conflicts of Interest: Wang JX, None; **Zhou N,** None; **Yang LH,** None; **Wei WB,** None.

REFERENCES

1 Reese AB, Jones IS. Hematomas under the retinal pigment epithelium. *Trans Am Ophthalmol Soc* 1961;59:43-79.

2 Annesley WH Jr. Peripheral exudative hemorrhagic chorioretinopathy. *Trans Am Ophthalmol Soc* 1980;78:321-64.

3 Shields CL, Salazar PF, Mashayekhi A, *et al*. Peripheral exudative hemorrhagic chorioretinopathy simulating choroidal melanoma in 173 eyes. *Ophthalmology* 2009;116(3):529-535.

4 Goel N. Vitreous hemorrhage as the presenting feature of peripheral

exudative hemorrhagic chorioretinopathy in Indian eyes. *Indian J Ophthalmol* 2019;67(3):419-423.

5 Zicarelli F, Preziosa C, Staurenghi G, *et al*. Peripheral exudative haemorrhagic chorioretinopathy: a widefield imaging study. *Br J Ophthalmol* 2021;105(10):1410-1414.

6 Shroff D, Sharma M, Chhablani J, *et al*. Peripheral exudative hemorrhagic chorioretinopathy-a new addition to the spectrum of pachychoroid disease. *Retina* 2021;41(7):1518-1525.

7 Goldman DR, Freund KB, McCannel CA, *et al*. Peripheral polypoidal choroidal vasculopathy as a cause of peripheral exudative hemorrhagic chorioretinopathy: a report of 10 eyes. *Retina* 2013;33(1):48-55.

8 Mantel I, Schalenbourg A, Zografos L. Peripheral exudative hemorrhagic chorioretinopathy: polypoidal choroidal vasculopathy and hemodynamic modifications. *Am J Ophthalmol* 2012;153(5):910-922.e2.

9 Roca-Cabau MA, Kumaran N, Asencio-Durán M, *et al*. Pseudomelanoma diagnosis in a tertiary ophthalmologic centre in Spain. *Can J Ophthalmol* 2024;59(6):e763-e767.

10 Safir M, Zloto O, Fabian ID, *et al*. Peripheral Exudative Hemorrhagic Chorioretinopathy with and without treatment-Clinical and multimodal imaging characteristics and prognosis. *PLoS One* 2022;17(9):e0275163.

11 Sodhi GS, Singh N, Wrenn J, *et al*. Peripheral hemorrhagic chorioretinopathy: differentiating features from choroidal melanoma. *Ocul Oncol Pathol* 2023;9(1-2):1-8.

12 Vandefonteyne S, Caujolle JP, Rosier L, *et al*. Diagnosis and treatment of peripheral exudative haemorrhagic chorioretinopathy. *Br J Ophthalmol* 2020;104(6):874-878.

13 Larrea J, Sánchez-Ávila RM, Villota-Deleu E, *et al*. Clinical characteristics of peripheral exudative hemorrhagic chorioretinopathy in a referral center in Spain. *Arch Soc Esp Oftalmol* 2022;97(11):603-611.

14 Choi S, Lee SC, Byeon SH, *et al*. Peripheral exudative hemorrhagic chorioretinopathy in Asian populations. *Retina* 2023;43(5):762-766.

15 Lee H, Choi YJ, Lee SM, *et al*. Clinical characteristics of peripheral exudative hemorrhagic chorioretinopathy easily misdiagnosed at initial presentation in Korean patients. *Sci Rep* 2025;15(1):30693.

16 Sen P, Manayath G, Shroff D, *et al*. Polypoidal choroidal vasculopathy: an update on diagnosis and treatment. *Clin Ophthalmol* 2023;17:53-70.

17 Shields JA, Mashayekhi A, Ra S, *et al*. Pseudomelanomas of the posterior uveal tract: the 2006 Taylor R. Smith Lecture. *Retina* 2005;25(6):767-771.

18 Seibel I, Hager A, Duncker T, *et al*. Anti-VEGF therapy in symptomatic peripheral exudative hemorrhagic chorioretinopathy (PEHCR) involving the macula. *Graefes Arch Clin Exp Ophthalmol* 2016;254(4):653-659.

19 Mantel I, Uffer S, Zografos L. Peripheral exudative hemorrhagic chorioretinopathy: a clinical, angiographic, and histologic study. *Am J Ophthalmol* 2009;148(6):932-938.e1.

20 Kim YT, Kang SW, Lee JH, *et al*. Peripheral exudative hemorrhagic chorioretinopathy in Korean patients. *Jpn J Ophthalmol* 2010;54(3):227-231.

21 Cebeci Z, Dere Y, Bayraktar Ş, *et al*. Clinical features and course of patients with peripheral exudative hemorrhagic chorioretinopathy. *Turk J Ophthalmol* 2016;46(5):215-220.

22 Glatz W, Schörkhuber M, Pöschl EM, *et al*. Persistent vitreoretinal adhesion in eyes with peripheral exudative haemorrhagic chorioretinopathy. *Acta Ophthalmol* 2017;95(1):e82-e83.

23 Choi EY, Kim HR, Jung J, *et al*. Bilateral macular choroidal abnormalities with drusenoid deposits in patients with unilateral peripheral exudative hemorrhagic chorioretinopathy. *Retina* 2023;43(1):120-129.

24 Kritfuangfoo T, Li Y, Mieler WF. Clinical characteristics and treatment outcomes of breakthrough vitreous hemorrhage in peripheral exudative hemorrhagic chorioretinopathy (PEHCR). *Clin Ophthalmol* 2025;19:2821-2833.