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Hidden in the Periphery: A Case Report on Peripheral Exudative Hemorrhagic Chorioretinopathy

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ABSTRACT

Introduction: Peripheral exudative hemorrhagic chorioretinopathy is an uncommon degenerative peripheral retinal condition characterized by multilayered hemorrhaging, exudation, or both that is beneath the retinal pigment epithelium or sensory retina. Its exact etiology is not well understood.

Case Report: A 91-year-old Caucasian male presented for a routine eye exam without visual complaints. His medical history included prediabetes, anemia, arrhythmia, atrial fibrillation, chronic kidney disease, hypertension, hyperlipidemia, and a history of bladder cancer. His ocular history included pseudophakia, dry eye syndrome, hypertension without ocular complications, and refractive error with presbyopia. His dilated fundus exam revealed extensive multilayered temporal peripheral hemorrhaging in the left eye. Over time, the hemorrhaging gradually resolved with residual peripheral fibrosis in the left eye.

Conclusion: Peripheral exudative hemorrhagic chorioretinopathy should be considered in elderly patients presenting with peripheral multilayered hemorrhaging. Multimodal imaging helps in distinguishing peripheral exudative hemorrhagic chorioretinopathy from malignant and vascular mimickers. Most cases resolve with observation, although continued monitoring is warranted.

Keywords: choroid, exudation, hemorrhage, pseudomelanoma, retina, ultrawide imaging

INTRODUCTION

Peripheral exudative hemorrhagic chorioretinopathy (PEHCR) is an uncommon degenerative condition affecting the peripheral retina and choroid. It is characterized by subretinal and subretinal pigment epithelium (RPE) hemorrhaging, exudation, and, in later stages, fibrosis. The condition was first described by Reese and Jones in 1961 and later termed PEHCR by Annesley in 1980.[1-2] It is frequently misdiagnosed due to its resemblance to choroidal melanoma and other peripheral retinal disorders such as polypoidal choroidal vasculopathy or a retinal detachment.[1-2] PEHCR has also been described as a peripheral variant of age-related macular degeneration (AMD) and has been compared to peripheral polypoidal choroidal vasculopathy due to overlapping hemorrhagic features.[3-5] Most patients are asymptomatic unless hemorrhaging or exudation extends into the macula or a vitreous hemorrhage develops.[3] Given its similar appearance to choroidal melanoma, recognition of clinical features and appropriate use of imaging are needed in guiding management and diagnosis.

CASE REPORT

A 91-year-old Caucasian male inpatient presented for his annual eye exam. He reported stable vision and denied flashes, floaters, diplopia, eye pain, or recent trauma. His medical history included prediabetes, anemia, atrial fibrillation, chronic kidney disease, hypertension, hyperlipidemia, and a history of bladder cancer. His systemic medications included apixaban, lisinopril, metoprolol succinate, atorvastatin, tamsulosin, finasteride, memantine, mirtazapine, acetaminophen, tramadol, and simethicone. His blood pressure was 145/56 mmHg, which was measured by hospital staff. His social history was negative for alcohol, tobacco, and recreational drug use. Ocular history included pseudophakia, dry eye syndrome, and refractive error with presbyopia. There was no prior history of retinal disease.

Best corrected visual acuity was 20/25 in the right eye and 20/30+1 in the left eye, which was stable compared to previous visits. Pupils were equal and reactive without an afferent defect. Extraocular motility was full. Confrontation

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visual fields were full to finger counting. Intraocular pressure measured by Goldmann applanation was 13 mmHg in both eyes. Anterior segment exam showed mild lid debris and trace corneal guttata with superficial punctate staining in both eyes. The anterior chambers were deep and quiet. Posterior chamber intraocular lenses were well centered and clear. A dilated fundus exam was performed using slit lamp biomicroscopy and binocular indirect ophthalmoscopy. The vitreous showed mild syneresis, but no cells in both eyes. The optic nerves were well perfused with distinct margins (cup-to-disc ratio 0.20 in both eyes). Both maculae were flat and intact. Retinal vessels in the posterior pole appeared normal in both eyes. In the left eye, there was extensive multilayered hemorrhaging involving the superotemporal, temporal, and inferotemporal periphery. The hemorrhaging appeared intraretinal, subretinal, and sub-RPE, with small areas of dehemoglobinized blood. No retinal breaks were identified. B-scan ultrasound was not performed at the initial visit.

Given the peripheral location, multilayered appearance, and preserved central vision, PEHCR was considered the most likely diagnosis. However, because of overlap with choroidal melanoma, the patient was referred to a retina specialist for further evaluation with fluorescein angiography. Ultrawide field imaging was performed using the Optos, as seen in Figure 1, for the left eye with temporal steering.

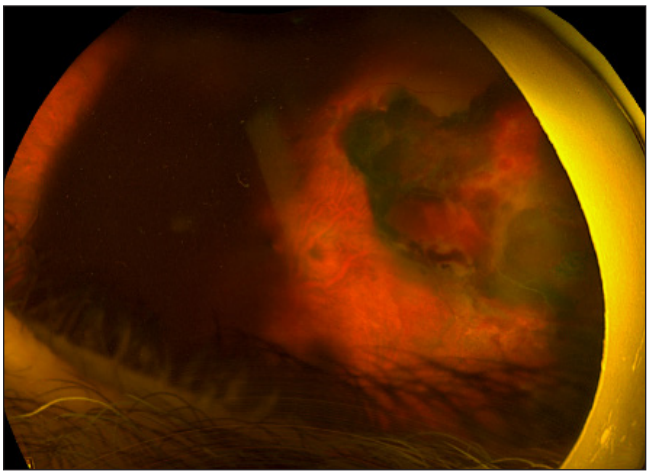

Figure 1: This Optos image of the left eye shows extensive intraretinal, subretinal pigment epithelium, and subretinal hemorrhages in the peripheral temporal retina. The black shadow is an artifact.

FOLLOW-UP VISIT #1

At his three-month follow-up visit, the patient reported no new symptoms. Visual acuity was 20/30 in the right and left eyes. Areas of dehemoglobinized hemorrhaging were observed in the left temporal periphery, appearing more yellow as the blood had broken down, with significant improvement compared to the initial presentation. No retinal breaks or lesions were noted. The patient attended his retinal referral appointment. However, documentation was unavailable due to the retina provider's sudden departure, and it is unknown whether fluorescein angiography or ultrasound was performed. Observation was continued with a follow-up scheduled in three months due to resolution. Ultrawide field imaging was performed using the Optos, as seen in Figure 2, for the left eye.

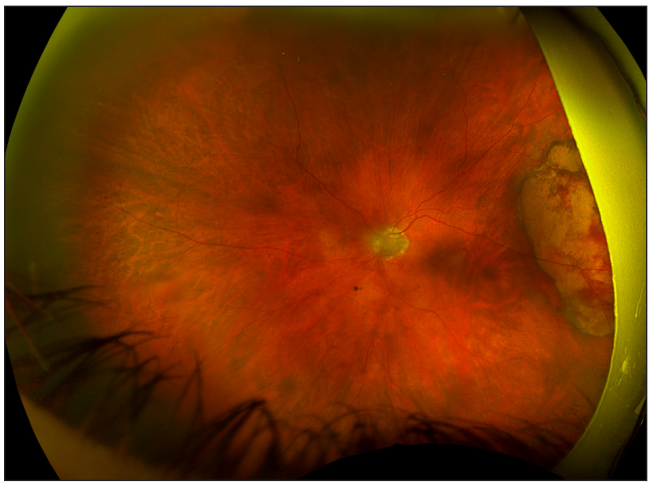

Figure 2: This Optos image of the left eye shows extensive dehemoglobinized hemorrhaging and resolving hemorrhages in the peripheral temporal retina.

FOLLOW-UP VISIT #2

At the next follow-up visit, visual acuity was 20/25+2 in the left eye. The hemorrhaging had further resolved, leaving residual peripheral fibrosis in the temporal retina. No macular involvement developed. Given stable vision and spontaneous resolution, follow-up was extended to six months. However, the patient did not return for the scheduled visit and was lost to follow-up. Ultrawide field imaging was performed using the Optos, as seen in Figure 3, for the left eye.

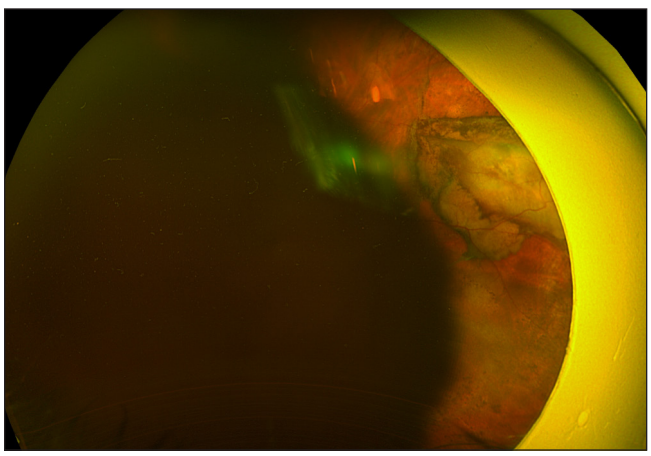

Figure 3: This Optos image of the left eye shows a large area of fibrosis with resolved hemorrhaging in the peripheral temporal retina. The black shadow is an artifact.

DISCUSSION

PEHCR is an uncommon degenerative condition affecting both the retina and choroid. First described in 1961 by Reese and Jones, it was initially characterized as subretinal pigment epithelial hematomas resembling lesions seen in age-related macular degeneration.[1] In 1980, Annesley introduced the term "PEHCR" after recognizing additional features, including subretinal fluid, intraretinal hemorrhages, exudates, vitreous hemorrhage, and other vascular changes in the overlying retina.[1-2] Today, PEHCR is often referred to as peripheral age-related retinal degeneration due to its association with subretinal or subretinal pigment epithelium hemorrhages, exudation, and fibrosis.[3] It has also been described as a variant of AMD due to its similar pathology and is often compared to polypoidal choroidal vasculopathy because of its highly similar hemorrhagic and exudative nature.[1]

The exact pathophysiology of PECHR remains unclear. While it has been described as a variant of AMD due to shared pathological features, there is no histological evidence directly linking this condition to AMD.[4] Unlike exudative AMD, most cases of peripheral exudative hemorrhagic chorioretinopathy occur without macular drusen and tend to resolve spontaneously rather than progress. Given its similarities to polypoidal choroidal vasculopathy, some studies suggest that patients with PEHCR undergo fluorescein angiography or indocyanine green angiography.[5] In cases where indocyanine green angiography is used, it helps determine whether these large hemorrhagic lesions may be caused by pigment epithelial detachments associated with polyp-like lesions, which could be the underlying cause of PEHCR.[5] However, the connection between this condition and polypoidal choroidal vasculopathy remains unclear.

PEHCR primarily affects elderly patients, typically between 70 and 80 years of age, with a slight female predominance (60 to 70%) and higher reported prevalence in Caucasian populations.[3-4] Systemic comorbidities such as hypertension and anticoagulant use have been frequently reported in case series. In a case series of 146 patients with PEHCR, 51% had hypertension, 14% had diabetes, and 44% were using anticoagulants.[3] However, these findings represent associations rather than proven risk factors.[3] Up to 60 percent of patients reported no symptoms at initial presentation with PEHCR.[7] When symptoms do occur, the most common symptoms include vision loss, an increase in floaters, or new flashes of light. Less commonly, patients may experience visual field changes, metamorphopsia, or pain. Two case series found that 48 to 51% of patients had a best-corrected visual acuity between 20/20 and 20/40.[3-4] However, about 20% had a best-corrected visual acuity of 20/200 or worse, often due to vitreous hemorrhage, subretinal fluid, or lipid exudation affecting the macula.[3-4]

The most common clinical signs are peripheral subretinal or subretinal pigment epithelium hemorrhages. These hemorrhages are usually found between the equator and the ora serrata, more often in the inferior-temporal quadrant than the superior-temporal quadrant. The temporal location has been reported in over 75% of cases.[3] This condition most often affects one eye, but bilateral involvement has been reported in 9 to 43% of cases.[6] Subretinal hemorrhages are the most frequently observed retinal finding, with one case study reporting them in more than 70% of cases.[7] Subretinal exudation is also common and can be helpful in distinguishing this condition from choroidal melanoma. Other clinical findings may include subretinal fibrosis, serous pigment epithelial detachments, and retinal pigment epithelium hyperplasia or atrophy.[3]

The differential diagnosis of peripheral exudative hemorrhagic chorioretinopathy includes multiple retinal and choroidal conditions with overlapping peripheral findings. Among these, choroidal melanoma remains the most common misdiagnosis, as PEHCR is the second most common cause of pseudomelanoma after choroidal nevus.[3] Several distinguishing features help differentiate PEHCR from choroidal melanoma. Patients with choroidal melanoma are often more symptomatic, as these lesions may be larger and associated with serous retinal detachment, leading to flashes, floaters, or peripheral visual field defects.[3] On examination, choroidal melanoma typically presents as a darker, variably pigmented choroidal mass and may demonstrate overlying lipofuscin and subretinal fluid. In contrast, peripheral exudative hemorrhagic chorioretinopathy is characterized by prominent subretinal or subretinal pigment epithelium hemorrhaging, which is a hallmark feature of the condition. Additionally, lesions in PEHCR tend to appear hemorrhagic and exudative rather than as a solid, elevated pigmented mass, further aiding differentiation. Retinal detachment may mimic PEHCR when peripheral elevation is present, but the underlying mechanisms and clinical appearances differ. Rhegmatogenous retinal detachment occurs due to a retinal break that permits vitreous fluid to accumulate beneath the retina, whereas tractional retinal detachment results from fibrovascular membranes exerting traction on the retina. Exudative retinal detachment arises from fluid accumulation due to lesions, inflammation, or vascular diseases that lead to a separation of the neurosensory retina from the retinal pigment epithelium. Unlike in cases of PEHCR, retinal detachment typically presents as a smooth, elevated, gray-appearing retina with folds and undulations and lacks the dense subretinal hemorrhage characteristic of PEHCR.[9] Retinal capillary hemangioma presents as a well-circumscribed red or pink vascular mass, often accompanied by a dilated feeding artery and draining vein.[10] Although exudation may occur and lead to subretinal fluid or tractional changes, hemorrhage is not a defining feature. In contrast, lesions in PEHCR are not associated with prominent feeder vessels and instead demonstrate irregular hemorrhagic and exudative changes in the peripheral retina. Vitreoretinal lymphoma typically presents with vitreous cellular infiltration and multifocal creamy or white

lesions that may be retinal or subretinal.[11] These lesions can occur anywhere in the retina and may increase in size over time or spontaneously resolve. When they resolve, they may leave retinal pigment epithelium atrophy and subretinal fibrosis, which can take on a "leopard spot" appearance due to collections of subretinal pigmented lesions that coalesce over time.[11] The presence of sheets or clumps of vitreous cells is a key distinguishing feature.[11] Hemorrhaging and exudation are less prominent and generally occur only in advanced stages. This contrasts with PEHCR, in which hemorrhaging is a main finding and vitreous inflammation is absent. Coats disease is characterized by retinal telangiectasia, aneurysmal vascular changes, and significant intraretinal and subretinal lipid exudation, most commonly in the inferotemporal periphery.[12] While exudative retinal detachment may occur in advanced stages, the hallmark findings are vascular abnormalities and lipid deposition rather than hemorrhaging. In PEHCR, there is no prominent telangiectasia or aneurysmal vasculature, and hemorrhage is more prominent than lipid exudation. Overall, while these conditions may share features such as peripheral lesions, exudation, or retinal elevation, PEHCR is distinguished by its peripheral location, characteristic subretinal or subretinal pigment epithelium hemorrhage, and the absence of intrinsic vascular tumors or vitreous inflammation.

Multimodal imaging plays an important role in diagnosing and managing PEHCR. A combination of ultra-wide field or wide field fundus photography with fundus autofluorescence, optical coherence tomography (OCT), fluorescein angiography, indocyanine green angiography, and B-scan ultrasound helps differentiate PEHCR from other conditions. OCT may reveal subretinal fluid, elevation, pigment epithelial detachments, and fibrosis, which can offer more detailed structural information.[2-3] Fluorescein angiography helps assess retinal circulation by detecting areas of pooling in serous pigmental epithelial detachments, early leakage from neovascularization, areas of ischemia, and blockages due to extensive hemorrhaging.[2-3] It can also highlight regions of hyper- or hypofluorescence that correspond to retinal pigment epithelium changes, such as atrophy. Indocyanine green angiography is mostly useful for evaluating abnormalities in the choroidal vascular network, as the dye penetrates the choroidal layer better with less diffusion, revealing a pathologic choroidal network that may not be visible in fluorescein angiography.[4,8] A key distinguishing feature of peripheral exudative hemorrhagic chorioretinopathy (PEHCR) on B-scan ultrasound is the presence of a retraction cleft, which represents a hyporeflective space separating the subretinal or subretinal pigment epithelial hemorrhagic clot from the underlying choroid.4 This finding is not seen in retinal detachment, in which the detached retina typically remains tethered at the optic nerve and ora serrata, producing a mobile V-shaped or funnel configuration rather than a focal cleft. In choroidal melanoma, lesions are usually solid, dome-shaped, and acoustically hollow, but they do not demonstrate a separable hemorrhagic clot plane as seen in PEHCR. In addition to the retraction cleft, PEHCR lesions often demonstrate mixed internal reflectivity. The hemorrhagic component may appear moderately to highly reflective depending on the age and organization of the blood.[4] This evolving reflectivity pattern can support the diagnosis and may also provide indirect information regarding lesion chronicity. B-scan ultrasound is particularly useful in PEHCR because it can evaluate lesions that are too peripheral or too elevated to be fully characterized with OCT or FA. OCT has limited utility in these cases, as peripheral pathology often falls outside the imaging field, and media opacity from hemorrhage can further limit visualization. Similarly, FA may be restricted by blockage from dense hemorrhage and does not reliably delineate the elevated subretinal or subretinal pigment epithelial components that define PEHCR. As a result, B-scan ultrasound is a very reliable imaging modality in cases where PEHCR is suspected but fundus visualization is limited or when other imaging techniques are inconclusive.[4]

Most cases of PEHCR are self-limiting, with lesions often undergoing spontaneous resolution without intervention. In a case series of 90 patients, spontaneous regression was observed in 89% of cases.[3] However, treatment may be necessary when complications arise, such as choroidal neovascularization or vitreous hemorrhage. Therapeutic options include intravitreal anti–vascular endothelial growth factor (anti-VEGF) injections, laser photocoagulation, cryotherapy, and pars plana vitrectomy.[1] Intravitreal anti-VEGF injections are the most common treatment, particularly in cases with macular involvement from subretinal fluid, hemorrhaging, or exudation. These agents inhibit VEGF, thereby reducing vascular permeability and leakage from abnormal choroidal vessels, promoting reabsorption of subretinal fluid and stabilization of hemorrhaging.[1] Laser photocoagulation delivers focal thermal energy to peripheral lesions, inducing coagulative necrosis and closure of abnormal vessels. This reduces ongoing leakage or bleeding and is typically reserved for well-localized lesions that are clearly visualized and located away from the macula.[1] Cryotherapy achieves vascular closure through controlled freezing, resulting in tissue necrosis and subsequent scarring that destroys abnormal vessels.[1] It is generally used for more peripheral or more extensive lesions that are not responsive to laser treatment. Pars plana vitrectomy is reserved for advanced cases, most commonly those with an unresolving vitreous hemorrhage.[1] Unlike other treatments that target the peripheral lesion directly, a vitrectomy removes vitreous hemorrhage to clear the visual axis. Overall, the prognosis for PEHCR is generally favorable due to its tendency toward spontaneous resolution. Visual outcomes depend largely on the extent of hemorrhage and whether the macula is involved. In general, anti-VEGF therapy is most often used for active or macula-threatening disease, while laser photocoagulation and cryotherapy are directed toward stabilization of peripheral lesions, and vitrectomy is reserved for vision-limiting complications.

LIMITATIONS

This case is limited by the absence of a B-scan ultrasound and peripheral OCT raster scans at the initial visit, as well as the lack of fluorescein angiography at the time of retina referral. Having these studies earlier would have likely helped clarify the diagnosis sooner and better characterize the lesion. A B-scan ultrasound could have provided valuable information regarding the lesion's internal reflectivity and helped exclude alternative etiologies such as a choroidal mass. Peripheral OCT raster imaging may have added additional structural detail, particularly in identifying subretinal fluid, the extent of retinal hemorrhaging, or subtle elevations in the far periphery that are often difficult to visualize during a fundus exam. Fluorescein angiography would also have been useful in assessing for areas of leakage or late staining patterns, potentially helping to differentiate PEHCR from choroidal neovascularization or inflammatory and vascular conditions. The absence of visual field testing represents another potential limitation, as it may have helped correlate the extent of peripheral involvement with functional impact and provided a useful baseline for monitoring, including potential effects on activities of daily living.

Despite these limitations, the overall clinical course showing gradual regression with residual fibrosis and retinal pigment epithelial changes is most consistent with PEHCR and supports the final diagnosis.

CONCLUSION

PEHCR is an uncommon chorioretinal condition that can be challenging to diagnose due to its resemblance to other ocular diseases, such as choroidal melanoma. A solid understanding of its epidemiology and prognosis can help clinicians recognize its distinct clinical and imaging features, thereby reducing the risk of misdiagnosis. Early recognition is important, as PEHCR is often self-limiting and may be managed conservatively in many cases, thereby avoiding unnecessary invasive procedures or overtreatment. At the same time, identifying complications such as macular involvement or a vitreous hemorrhage allows for timely intervention when needed to preserve visual function. Overall, this case emphasizes the diagnostic challenges posed by PEHCR and reinforces how understanding its characteristic features can help reduce misdiagnosis, guide appropriate management, and ultimately improve patient outcomes.

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