Peripheral Exudative Hemorrhagic Chorioretinopathy with Clinical Presentation Similar to Choroidal Melanoma: Case Report

Coriorretinopatia Exsudativa Hemorrágica Periférica com Apresentação Semelhante a Melanoma Coroideu: Caso Clínico

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ABSTRACT

Peripheral exudative hemorrhagic chorioretinopathy (PEHCR) is a rare degenerative retinal disorder characterized by peripheral hemorrhagic and exudative lesions that frequently mimic choroidal melanoma and other intraocular tumors. We report a case of a 78-year-old male with initial suspicion of choroidal melanoma, ultimately diagnosed as PEHCR following multimodal imaging. This report highlights the diagnostic challenges posed by PEHCR, the fundamental role of advanced imaging techniques, and the importance of a conservative management approach in most cases. A brief review of the literature is provided to contextualize the clinical, imaging, and therapeutic features of this underrecognized entity.

KEYWORDS: Choroid Diseases; Multimodal Imaging; Retinal Diseases; Retinal Hemorrhage.

RESUMO

A coriorretinopatia exsudativa hemorrágica periférica é uma doença degenerativa rara da retina, caracterizada por lesões hemorrágicas e exsudativas periféricas que frequentemente simulam o melanoma da coroide e outros tumores intraoculares. Relatamos um caso de um homem de 78 anos, com suspeita inicial de melanoma da coroide, posteriormente diagnosticado como retinopatia coriorretiniana hemorrágica exsudativa periférica após estudo de imagem multimodal. Este relato destaca os desafios diagnósticos colocados pela retinopatia coriorretiniana hemorrágica exsudativa periférica, o papel fundamental das técnicas avançadas de imagem e a importância de uma abordagem conservadora na maioria dos casos. É apresentada uma breve revisão da literatura para contextualizar as características clínicas, imagiológicas e terapêuticas desta entidade pouco reconhecida.

PALAVRAS-CHAVE: Doenças da Coroide; Doenças da Retina; Imagiologia Multimodal; Neoplasias da Retina.

INTRODUCTION

Peripheral exudative hemorrhagic chorioretinopathy (PEHCR) is a rare degenerative retinal disorder typically affecting elderly patients, characterized by peripheral hemorrhagic or exudative lesions located beneath the retina or the retinal pigment epithelium (RPE).[1] It is a well-recognized mimicker of choroidal melanoma, as overlapping clinical and imaging characteristics often make differentiation challenging.[2]

PEHCR was initially described by Reese and Jones in 1962 as a localized collection of blood beneath the RPE, which they termed a sub-retinal pigment epithelial hematoma.[3] In 1980, Annesley *et al* introduced the term peripheral exudative hemorrhagic chorioretinopathy (PEHCR) to describe this entity.[4] Despite its rarity, PEHCR is recognized as the second most common cause of pseudomelanoma, accounting for approximately 13% of cases, following choroidal nevus (49%).[5] Its dark and elevated appearance in the peripheral retina often leads to misdiagnosis as choroidal melanoma, but it may also mimic other conditions such as retinal detachment, posterior vitreous degeneration, retinal artery macroaneurysm, congenital hypertrophy of the retinal pigment epithelium, circumscribed choroidal hemangioma, age-related macular degeneration (AMD), and even primary vitreoretinal lymphoma.[5]

Clinical and epidemiological data suggest an association with advanced age, systemic hypertension, and the use of anticoagulant or antiplatelet agents, and the condition appears to be more common in women and Caucasian patients.[6] Because of shared pathological and imaging features, PEHCR has been described as a peripheral variant of AMD.[5] Additionally, it has increasingly been considered part of the polypoidal choroidal vasculopathy (PCV) spectrum, owing to its shared highly hemorrhagic and exudative characteristics and the presence of polypoidal lesions observed in some cases on indocyanine green angiography (ICGA).[5] Lesions are frequently asymptomatic due to their peripheral location; however, patients may present with visual loss secondary to hemorrhagic or exudative complications extending to the macula, or with nonspecific symptoms such as floaters, metamorphopsia, or scotoma.[5]

In this case report, we describe a patient with PEHCR initially referred with presumed choroidal melanoma, thoroughly documented through multimodal imaging. We emphasize the key clinical and imaging features that help distinguish PEHCR from intraocular malignancy and discuss current evidence regarding its pathophysiology, natural history, and management.

CASE REPORT

A 78-year-old male was referred for urgent ophthalmology evaluation due to suspicion of choroidal melanoma in the left eye. The patient reported progressive visual loss and floaters in the same eye over one month. Medical history included type 2 diabetes mellitus, ischemic heart disease and right-sided colon adenocarcinoma treated with right hemicolectomy 5 years earlier, with no evidence of recurrence to date.

At initial presentation in the emergency department, the patient underwent a comprehensive systemic workup, including an extended blood panel with liver function tests, cranial computed tomography with angiographic sequences, and thoracic and abdominal computed tomography, in order to exclude systemic pathology and subclinical metastatic disease, particularly hepatic involvement, given the initial suspicion of choroidal melanoma.

On initial examination, best-corrected visual acuity (BCVA, Snellen chart) was 20/25 in the right eye and 20/50 in the left eye. Intraocular pressure was within normal limits in both eyes. Slit-lamp biomicroscopy revealed bilateral corticonuclear cataracts and pseudoexfoliation, with no other abnormalities in the anterior segment. Fundoscopy of the right eye showed a posterior pole with macular drusen and mild pigmentary changes compatible with early age-related macular degeneration, without hemorrhage or exudation (Fig. 1A). In the left eye, fundoscopy identified two elevated hemorrhagic lesions located temporal to the macula, surrounded by exudation, a mottled area anterior to the larger lesion, and central atrophic and pigmentary changes, without the presence of orange pigment (Fig. 1B). Optical coherence tomography of the left eye revealed a dome-shaped retinal elevation with subretinal fluid, compatible with a hemorrhagic pigment epithelial detachment (Fig. 2).

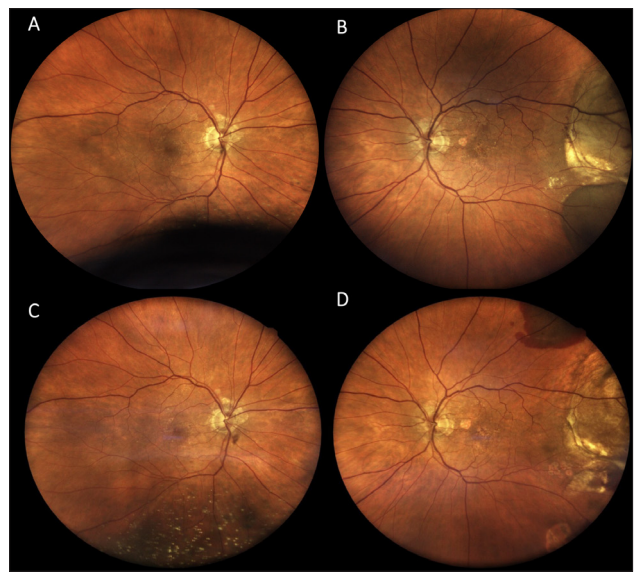


Figure 1. Color fundus photography obtained at initial presentation, showing the right eye (A) with macular drusen and mild retinal pigment epithelium irregularities compatible with early age-related macular changes, without macular edema, hemorrhage or exudation, and the left eye (B) with macular drusen and pigmentary changes coexisting with two elevated peripheral hemorrhagic lesions temporal to the macula, associated with surrounding exudation and macular atrophic changes consistent with peripheral exudative hemorrhagic chorioretinopathy. The right eye (C) remained unchanged, while the left eye (D) demonstrated disease progression with new peripheral hemorrhages six months after aflibercept treatment.

B-scan ultrasonography of the left eye demonstrated a temporal choroidal mass with heterogeneous echogenicity, associated with inferior exudative retinal detachment and a

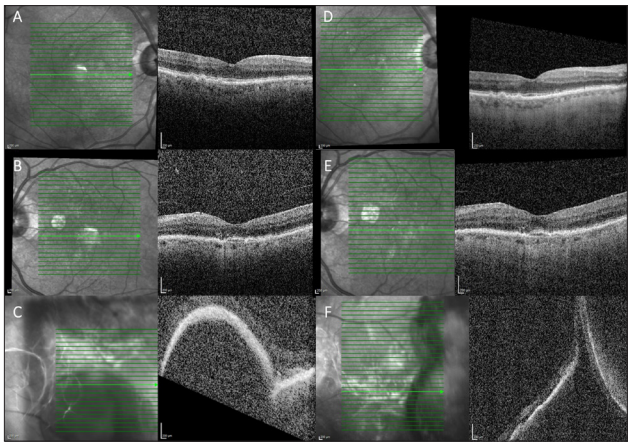


Figure 2. Optical coherence tomography at baseline (A-C) and six months after aflibercept treatment (D-F). Right eye macula (A, D) demonstrates drusen and retinal pigment epithelium irregularities consistent with early age-related macular changes, remaining stable throughout follow-up. Left eye macula (B, E) shows intraretinal and subretinal fluid with outer retinal layer disruption at baseline, with persistent macular fluid after treatment. Peripheral hemorrhagic lesion (C, F) reveals subretinal hyperreflective material and associated fluid at baseline, demonstrating progression with increased lesion elevation and subretinal exudation after treatment.

prechoroidal cleft, without choroidal excavation or intrinsic vascularity (Fig. 3). Orbital and cranial computed tomography revealed no significant abnormalities. Based on these findings, the patient was referred for further investigation.

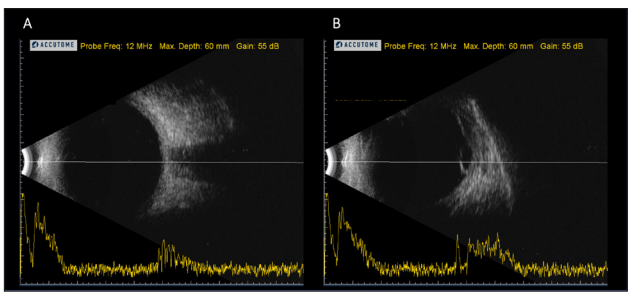


Figure 3. B-scan ultrasonography of the left eye at presentation. (A) Transverse scan showing no abnormalities with normal choroidal profile. (B) Longitudinal scan revealing an elevated, heterogeneous choroidal lesion with irregular contour, without choroidal excavation or intrinsic vascularity. These findings are consistent with peripheral exudative hemorrhagic chorioretinopathy and distinguish it from choroidal melanoma, which typically demonstrates choroidal excavation and intrinsic vascularity.

At a subsequent ophthalmology consultation, fluorescein angiography was performed, showing window defects and peripheral temporal exudation with blockage corresponding to areas of hemorrhage, without double circulation or other features suggestive of choroidal melanoma (Fig. 4). In the macular region, fluorescein angiography demonstrated irregular hyperfluorescence with late leakage and staining corresponding to central exudative and atrophic changes, consistent with active macular involvement (Fig. 4).

Pelvic computed tomography and orbital magnetic resonance imaging (MRI) were subsequently requested for further evaluation, with pelvic computed tomography (CT)

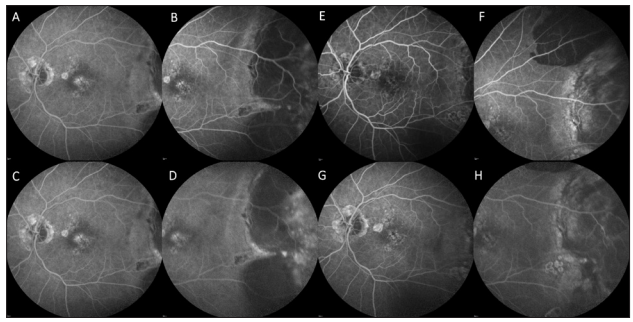


Figure 4. Fluorescein angiography of the left eye at initial presentation in early (A, B) and late (C, D) phases, demonstrating peripheral hypofluorescence due to blocking effect of hemorrhage, window defects from RPE alterations, surrounding late leakage from exudative changes, and irregular macular hyperfluorescence with late leakage consistent with active involvement, without double circulation or other features suggestive of choroidal melanoma. Six months after aflibercept treatment (E-H), early (E, F) and late phases (G, H) demonstrate disease progression with increased peripheral leakage, new areas of hypofluorescence from hemorrhage, and persistent macular hyperfluorescence with late staining.

performed to complete abdominopelvic metastatic screening and orbital MRI obtained to further characterize the intraocular lesion and to rule out extraocular extension.

At a follow-up appointment one week later, the results of the previously requested tests were reviewed. Liver function tests and pelvic computed tomography were normal. Orbital MRI showed a temporal, lens-shaped intraocular thickening in the left eye, showing T1 hyperintensity and T2 hypointensity, without nodular features, gadolinium enhancement, or diffusion restriction. There was no extraocular extension, and the possibility of subretinal exudate could not be excluded. The optic nerves and muscle-aponeurotic cones were preserved.

Based on these findings, the diagnosis of PEHCR was established, and an initial conservative approach with regular follow-up at approximately one-month intervals was adopted. At a subsequent consultation, treatment was initiated due to the persistence of exudative and hemorrhagic activity. This included laser photocoagulation targeting the peripheral lesion and three intravitreal injections of aflibercept, to be administered at monthly intervals.

At three-month follow-up after treatment completion, BCVA in the left eye showed improvement to 20/40, and clinical examination revealed reduced exudation with increasing fibrosis of the peripheral lesions and decreased subretinal fluid on optical coherence tomography.

However, at six-month follow-up, BCVA in the left eye remained stable at 20/25, although multimodal imaging demonstrated disease progression despite anti-VEGF therapy. Color fundus photography revealed new peripheral hemorrhages in the left eye, while the right eye remained stable (Fig. 1). Optical coherence tomography showed persistent intraretinal and subretinal fluid in the left macula with increased elevation of the peripheral hemorrhagic lesion, while the right eye maintained unchanged drusen without macular edema (Fig. 2). Fluorescein angiography demonstrated increased peripheral leakage with new areas of hypofluorescence from

hemorrhage and persistent macular hyperfluorescence with late staining, indicating continued disease activity (Fig. 4).

Given the disease reactivation with persistent exudative activity despite initial anti-VEGF therapy with aflibercept, treatment was switched to intravitreal faricimab. A regimen of three-monthly injections was planned, with subsequent reassessment using optical coherence tomography, fluorescein angiography, and B-scan ultrasonography to evaluate treatment response and guide further management.

DISCUSSION

PEHCR is an uncommon degenerative chorioretinal disorder that predominantly affects elderly individuals, with a mean age at diagnosis ranging from 77 to 83 years.[5,6] The disease is more prevalent among women and Caucasians,6 and has been associated with systemic hypertension, anticoagulant or antiplatelet therapy, and coexistent AMD,[7] although the exact relationship between PEHCR and AMD remains uncertain. In our case, the patient's advanced age and cardiovascular comorbidities were consistent with the demographic and systemic characteristics commonly reported in PEHCR, and both eyes showed macular drusen and mild retinal pigment epithelium irregularities compatible with early age-related macular changes.

The clinical presentation of PEHCR is highly variable, ranging from asymptomatic, incidentally detected lesions, to severe visual loss secondary to hemorrhage, exudation or vitreous involvement.[5,6,8] Lesions are most often located in the temporal quadrants, particularly in the inferotemporal region, and may be associated with serous or hemorrhagic retinal detachment.[5,6] In our patient, the presence of multiple temporal hemorrhagic and exudative lesions with associated macular changes was consistent with the classic phenotype of PEHCR.

Multimodal imaging is essential to differentiate PEHCR from choroidal melanoma and other intraocular tumors. B-scan ultrasonography typically demonstrates a dome-shaped, heterogeneous lesion without choroidal excavation or intrinsic vascularity, contrasting with the excavated, acoustically hollow and vascularized pattern often seen in melanoma.[5,7] Optical coherence tomography is valuable for assessing macular involvement and monitoring subretinal fluid and exudation, supporting both diagnosis and longitudinal management of the disease.[5,7] Fluorescein angiography and indocyanine green angiography may reveal peripheral blockage from hemorrhage, late leakage, and, in some cases, polypoidal lesions suggestive of PCV.[2,5]

The differential diagnosis of PEHCR includes choroidal melanoma, choroidal metastasis, choroidal hemangioma, vasoproliferative tumor, and other causes of peripheral exudative or hemorrhagic lesions.[5,6,8] Accurate distinction between PEHCR and choroidal melanoma is essential, as the latter carries a significant risk of systemic metastasis if left untreated, necessitating prompt intervention. In contrast, PEHCR generally follows a more benign course and may regress spontaneously or evolve into fibrotic scars. Large series have shown that a meaningful proportion of lesions initially presumed to be melanoma were subsequently identified as PEHCR, highlighting the need for careful clinical and imaging evaluation to avoid overtreatment.[2,5,8] In our case, the absence of choroidal excavation and intrinsic vascularity on ultrasonography, together with angiographic findings not supportive of melanoma, were decisive in establishing the diagnosis of PEHCR.

Management of PEHCR is usually individualized according to symptomatology, lesion activity and macular involvement. Asymptomatic or stable lesions are often managed conservatively with observation, as most regress spontaneously or evolve into fibrotic scars, with visual acuity remaining stable or improving over time, avoiding the potential risks associated with unnecessary interventions.[2,5,8] However, in eyes with persistent or recurrent hemorrhage, significant exudation involving the macula, or vitreous hemorrhage with visual impairment, active treatment may be warranted. In such cases, therapeutic options include intravitreal anti-VEGF injections, laser photocoagulation, and, in selected situations, pars plana vitrectomy.[5,7,9,10] Intravitreal anti VEGF is the most frequently reported treatment in recent series, with bevacizumab and ranibizumab predominantly used in earlier reports and, more recently, aflibercept also being employed. Case series have described a median of 3–4 injections to achieve resolution of macular subretinal fluid, with additional pro re nata injections in eyes with recurrent exudative activity.

In the present case, an initially conservative approach was adopted, however, persistent exudative and hemorrhagic activity with macular involvement prompted escalation to peripheral laser photocoagulation combined with intravitreal aflibercept, consistent with previously reported treatment approaches for symptomatic PEHCR. Published reports have documented anatomical improvement and variable visual recovery after intravitreal bevacizumab or ranibizumab, typically after 2–5 injections, as well as small series of aflibercept-treated eyes showing regression of peripheral lesions and macular exudation after 2–3 injections.[5,9,11] In our patient, the partial anatomical improvement was observed at three-month follow-up on fundus examination and optical coherence tomography, with increasing fibrosis, reduced exudation and decreased subretinal fluid.

However, at six-month follow-up, multimodal imaging demonstrated disease recurrence despite initial response to aflibercept therapy. Color fundus photography revealed new peripheral hemorrhages, optical coherence tomography showed persistent subretinal fluid with increased elevation of the peripheral lesion, and fluorescein angiography demonstrated increased peripheral leakage. This evolution illustrates that response to anti-VEGF therapy in PEHCR may be incomplete or transient, and that initial anatomical improvement does not preclude subsequent disease reactivation.

Given this incomplete response to aflibercept, a switch to intravitreal faricimab was initiated. Faricimab is a bispecific monoclonal antibody targeting both VEGF-A and angiopoietin-2, with potential advantages in vascular instability and exudative disease through modulation of the angiopoietin/Tie-2 pathway.[12] Although evidence for faricimab in PEHCR is currently lacking,[13] indirect support may be drawn from studies in polypoidal choroidal vasculopathy, a related exudative condition characterized by

peripheral polypoidal lesions and exudation, in which recent data have shown anatomical improvement and polyp closure in a substantial proportion of treated eyes.[14]

PEHCR is an underrecognized but clinically relevant entity that should be considered in elderly patients presenting with peripheral hemorrhagic or exudative retinal lesions that may simulate intraocular tumors, particularly choroidal melanoma. Its clinical and imaging resemblance to choroidal melanoma represents a substantial diagnostic challenge and may lead to unnecessary invasive interventions, including radiotherapy or even enucleation, if not correctly identified. This case illustrates the central role of multimodal imaging in distinguishing PEHCR from malignant pathology, and highlights the variable therapeutic response that may be observed in symptomatic PEHCR, with initial anatomical improvement followed by disease recurrence. It also demonstrates that switching to an anti-VEGF agent with a different mechanism of action may be considered in selected refractory cases, although further studies are needed to establish the role of newer bispecific agents such as faricimab in the management of PEHCR. As evidence accumulates regarding the relationship between PEHCR, polypoidal choroidal vasculopathy, and the pachychoroid disease spectrum, prospective studies are needed to define standardized diagnostic criteria and evidence-based therapeutic strategies.

CONTRIBUTORSHIP STATEMENT / DECLARAÇÃO DE CONTRIBUIÇÃO

AMSampaio, AMSilva, JCoelho, JCosta and LM: Made substantial contributions to the work, participated in the drafting and/or critical revision of the manuscript, approved the final version for publication, and agree to be accountable for all aspects of the work.

AMSampaio, AMSilva, JCoelho, JCosta e LM: Contribuíram substancialmente para o trabalho, participaram na redação e/ou revisão crítica do manuscrito, aprovaram a versão final para publicação e concordam em assumir a responsabilidade por todos os aspetos do trabalho.

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