

Case Report

Six Cases of Polypoidal Choroidal Vasculopathy in Eyes with Choroidal Nevi

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Keywords

Choroidal nevus · Polypoidal choroidal vasculopathy · Multimodal imaging · Optical coherence tomography · Fluorescein angiography

Abstract

Introduction: The aim of this study was to report the cases of 6 patients with the coexistence of polypoidal choroidal vasculopathy (PCV) and choroidal nevi. **Case Presentation:** Six patients with the coexistence of PCV and choroidal nevi were thoroughly evaluated by slit-lamp biomicroscopy examination, color fundus photography, optical coherence tomography (OCT), OCT angiography (OCT-A), fluorescein angiography (FA), indocyanine green angiography (ICG-A), fundus blue autofluorescence (BAF), and ocular ultrasound (OU). The typical features of PCV and nevi were present in all patients, three of whom were treated with intravitreal anti-angiogenic agents. In each clinical case, the choroidal Haller's vessels adjacent to the nevus were visibly more dilated compared to normal. Color fundus photography, OCT, OCT-A, FA, ICG-A, BAF, and OU revealed similar findings across all cases. Observations revealed that choroidal nevi could instigate modifications in the outer retina, resulting in persistent alterations capable of triggering the formation of neovascularization. **Conclusion:** The occurrence of a PCV alongside nevus is an uncommon complication. Findings from all exams performed were consistent across all cases, highlighting the potential link between PCV and nevi.

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Introduction

Choroidal nevus, the most prevalent noncancerous eye tumor in adults, clinically manifests a solid choroidal lesion of variable thickness, ranging from golden-tan (if lacking pigment) to dark brown [1]. Nevi in the eyes might lead to additional changes in the retinal pigment epithelium (RPE), including pigment epithelial detachment (PED), drusen, atrophy, and subretinal fluid (SRF) [2]. In just 1% of cases, a nevus is accompanied by choroid neovascularization [1]. It's possible that the existence of a choroidal nevus might cause such alteration initiating the development of new blood vessels such as polypoidal choroidal vasculopathy (PCV). PCV, first described by Yannuzzi et al. [3] in 1990, is a subtype of choroid neovascularization characterized by the presence of aneurysmatic dilatations. It has a grape-like structure and affects generally macular, peripapillary, and in some cases peripheral retinal areas [4, 5]. The presence of neovascularization exhibiting PCV-like features has been documented in conditions such as pathologic myopia and tilted disk syndrome, optic nerve melanocytoma, circumscribed choroidal hemangioma, and sickle cell retinopathy [6–10].

Nevertheless, the occurrence of a PCV alongside nevus is an uncommon complication [1]. In 2009, Peiretti [11] made the groundbreaking discovery that choroidal nevi could indeed be complicated by PCV, marking the first documented case of such an occurrence.

Multimodal imaging such as optical coherence tomography (OCT), OCT angiography (OCT-A), fundus blue autofluorescence (BAF), fluorescein angiography (FA), indocyanine angiography (ICG-A) and ocular ultrasounds can be used to study PCV and choroidal nevi [12–16]. In cases where PCV's treatment is required, the options include intravitreal (IV) anti-vascular endothelial growth factor (anti-VEGF) injections, photodynamic therapy, or focal argon laser [17–21].

Case Reports

Case 1

An 82-year-old Caucasian woman presented to the emergency room reporting visual impairment in left eye (LE). Her BCVA was 20/20 in right eye (RE), 20/25 in LE. Fundus examination (FE) in her LE disclosed the presence of a round pigmented lesion of about 2.5 disc diameter (DD), located superior to the optic disc with overlying drusen and small hemorrhages (Fig. 1a). BAF was irregular at the lesion site (Fig. 1b). OCT revealed the presence of a sharp-peaked multilobular PED over the lesion, associated with SRF and IRF (Fig. 1c). Choroidal thickness at the fovea was 240 μm and 280 μm proximal to the nevus. Moreover, OCT-A demonstrated a polypoidal lesion with branching vascular network (BVN) (Fig. 1e) At B-scan, the choroidal lesion was characterized by a dome-shaped morphology, high echogenicity, medium internal reflectivity, and rare internal vacuoles (Fig. 1d). FA highlighted the presence of multiple irregular bright spots distributed in a patchy configuration superiorly to the optic disc. ICG-A revealed the presence of multiple rounded bright spots compatible with PCV (Fig. 1e). In order to prevent vitreous or retinal bleedings, since exudation has progressively increased, 3 injections of bevacizumab and 5 injections of aflibercept were performed in the LE. The patient was followed up for 18 months. At the last visit, the BCVA in LE was 20/25; fluid was completely reabsorbed with contraction of the multilobular PED dimensions (Fig. 1h).

Case 2

A 64-year-old Caucasian woman was referred to the retina service after a recent diagnosis of a pigmented lesion in her LE. FE in her LE highlight a slightly elevated 2 DD pigmented lesion was noted along the inferotemporal vascular arcade, with overlying drusen and slight lipofuscin mobilization as demonstrated by BAF (Fig. 2a, b). OCT revealed the presence of a multinodular PED with sub-RPE ring-like lesions, associated with perilesional SRF (Fig. 2c).

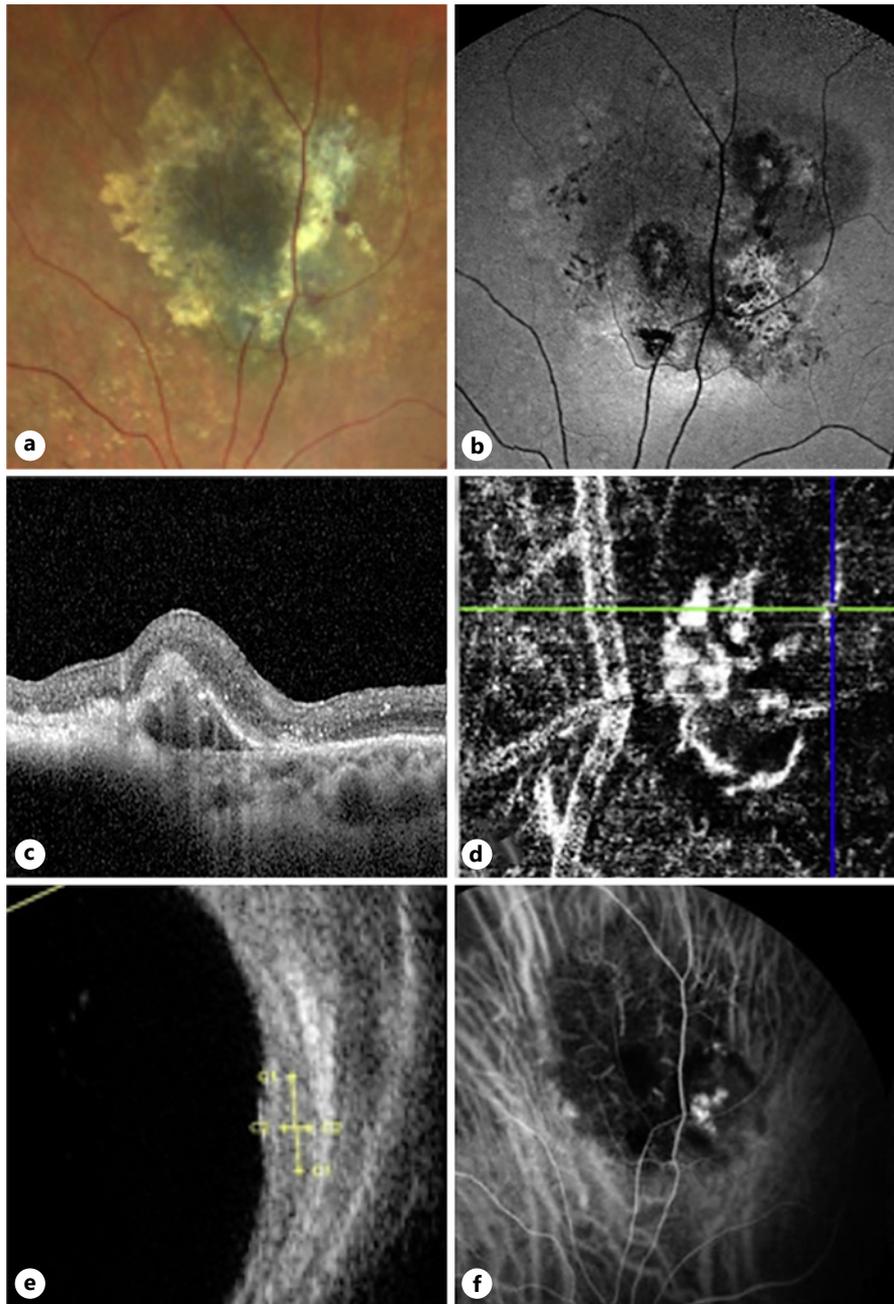


Fig. 1. **a** Fundus examination revealed the presence of a round pigmented lesion of about 2.5 disc diameter (DD). **b** Irregular BAF at lesion site. **c** OCT revealed the presence of a sharp-peaked multilobular PED over the lesion, associated with SRF and IRF. **d** OCT-A demonstrated a polypoidal lesion. **e** At B-scan, the choroidal lesion was characterized by a dome-shaped morphology, high echogenicity. **f** ICG-A revealed the presence of multiple rounded bright spots compatible with PCV.

OCT-A revealed the presence of multiple polyps (Fig. 2d). At B-scan, the choroidal lesion appeared dome-shaped, hyperechogenic with medium internal reflectivity, no vacuoles; any sign of choroidal excavation and no choroidal shadowing, was detected (Fig. 2e). FA demonstrated pooling of dye consistent with PED. ICG-A highlighted the presence of hypercyanescence lesion within the choroidal nevus (Fig. 2f). Patient refused IV treatment.

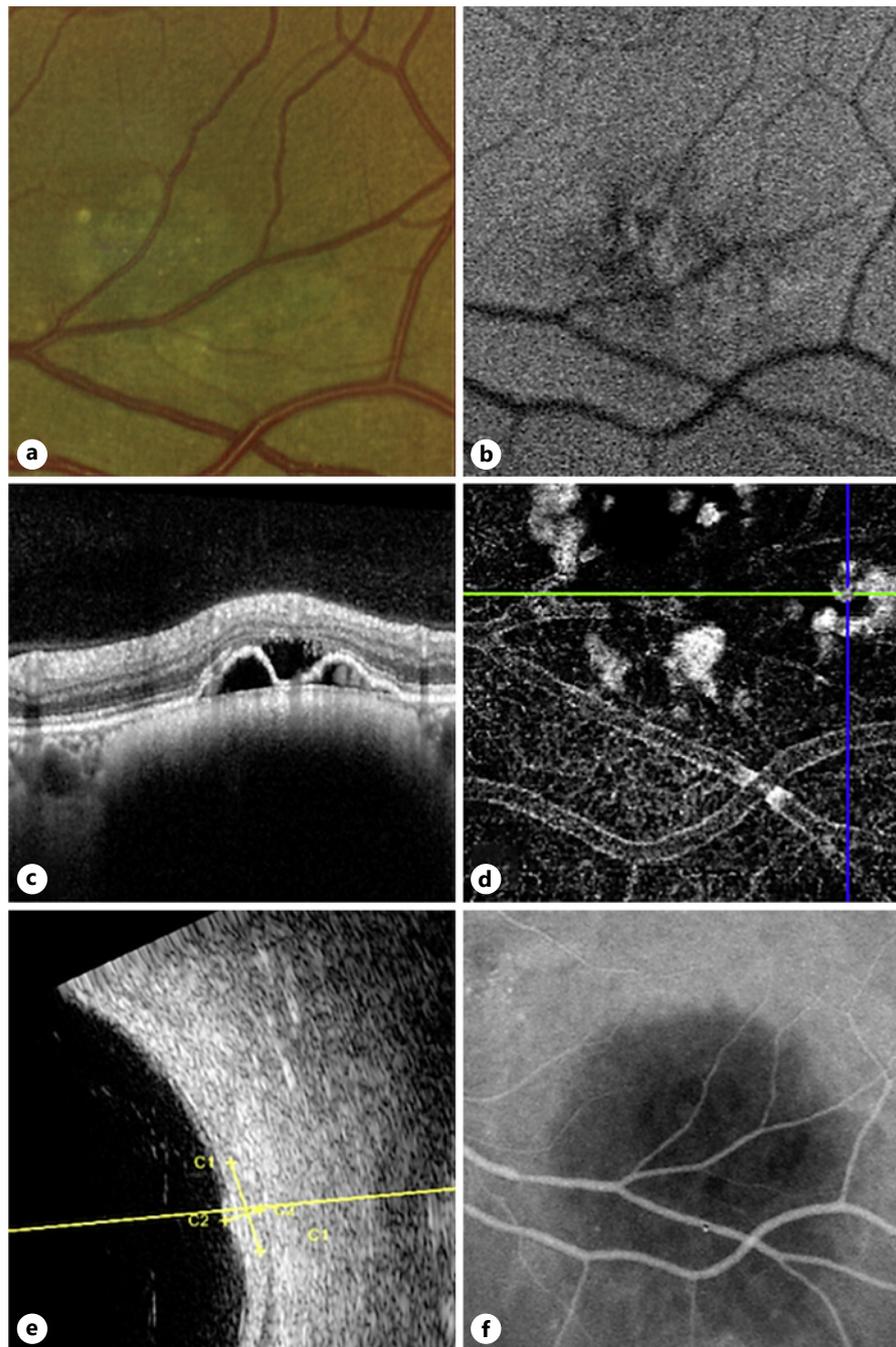


Fig. 2. **a** Fundus examination revealed elevated 2 DD pigmented lesion along the infero-temporal vascular arcade. **b** Accumulation of lipofuscin as demonstrated by BAF. **c** OCT scan revealed the presence of a multi-lobular PED with sub-RPE ring-like lesions, associated with perilesional SRF. **d** OCT-A revealed the presence of multiple polypoidal lesions. **e** At B-scan, the choroidal lesion appeared dome-shaped, with high echogenicity, medium internal reflectivity, no vacuoles. **f** ICG-A highlighted the presence of bright spots within the choroidal nevus.

Case 3

A 77-year-old woman presented to the emergency room complaining of progressive visual loss and metamorphopsia in her RE that started 1 month before. FE disclosed the presence of a foveal gray neovascular lesion associated with hemorrhages and subretinal

exudation; in addition, a flat subretinal, pigmented juxtapapillary lesion of about 3.5 DD, with overlying drusen and a round red spot at its inferonasal margin (Fig. 3a). The juxtapapillary lesion appeared irregularly hyper autofluorescence at BAF (Fig. 3b). The OCT revealed multiple double layered PEDs, a subRPE round lesion was disclosed at its inferonasal margin (Fig. 3c). OCT-A disclosed the presence of a BVN (Fig. 3d). Ultrasound was also performed (Fig. 3e). FA revealed foveal early hyperfluorescence corresponding to the neovascular lesion, associated with hypofluorescence due to sub/intraretinal hemorrhage, with late-phase dye leakage. ICGA highlighted the presence of multiple hypercyanescence small and well-delimited lesions within the choroidal nevus border that persisted throughout the entire duration of the examination (Fig. 3f). IV injections of bevacizumab were prescribed.

Case 4

A 77-year-old Caucasian woman was referred to the retina service after being diagnosed with proliferative diabetic retinopathy (PDR). FE in RE showed an irregular hyperreflective lesion, located superotemporally to the optic disc and sized approximately 1–1.5 DD (Fig. 4a). The lesion appeared hyperreflective on IR and irregularly hyper autofluorescence (Fig. 4b). OCT highlighted a multiple sharp-peaked PED associated with minimum IRF (Fig. 4c). OCT-A demonstrated a polypoidal lesion with terminal aneurysmal dilatations with an associated BVN (Fig. 4d). Ultrasound examination confirmed the diagnosis of a choroidal nevus (Fig. 4e). FA highlighted the presence of a PDR with irregular early hyperfluorescence corresponding to RPE alterations overlying the nevus. The ICG-A showed multiple hypercyanescence lesions within the choroidal lesion (Fig. 4f). IV injections of anti-VEGF and laser photocoagulation in OU were recommended for the PDR.

Case 5

A 64-year-old Caucasian man was referred to the retina service after the finding of a pigmented lesion in his LE. FE revealed a flat 1.5 DD pigmented lesion inferotemporal to the macula with overlying drusen and neuro retinal detachment (Fig. 5a, b). OCT showed the presence of multinodular PED with sub-RPE ring-like lesions, associated with perilesional SRF (Fig. 5c). OCT-A revealed a vast neovascular network associated with multiple polyps (Fig. 5d). Ultrasound examination confirmed the diagnosis of a choroidal nevus (Fig. 5e). FA highlighted a pin-point hyperfluorescence in the region of the neovascularization. ICGA confirmed the presence of polypoidal lesions within the choroidal nevus (Fig. 5f). The patient is undergoing IV therapy with bevacizumab.

Case 6

A 79-year-old Caucasian patient was referred to the retina service for the evaluation of a choroidal lesion. She was diagnosed with an asymptomatic dome shaped pigmented lesion in the parapapillary nasal region of her LE. The patient refuses performing FAG and ICGA exams, so we do not have fundus photography. OCT centered at the nevus revealed the presence of a peaked PED containing thin-walled polyp and double layer sign; no IRF/SRF was reported (Fig. 6a). CT at the fovea (250 μ m) was lower than near the nevus (269 μ m). OCT-A revealed the presence of a BVN characterized by terminal aneurysmal dilatations consistent with PCV (Fig. 6b). B-scan revealed a focal hyperechogenic choroidal thickening with no sign of choroidal erosion or exudation (Fig. 6c). The patient refused any addition examination. In this case, the diagnosis of PCV associated with choroidal nevus was based on the OCT characteristics, following the nomenclature published by Cheung et al. [22].

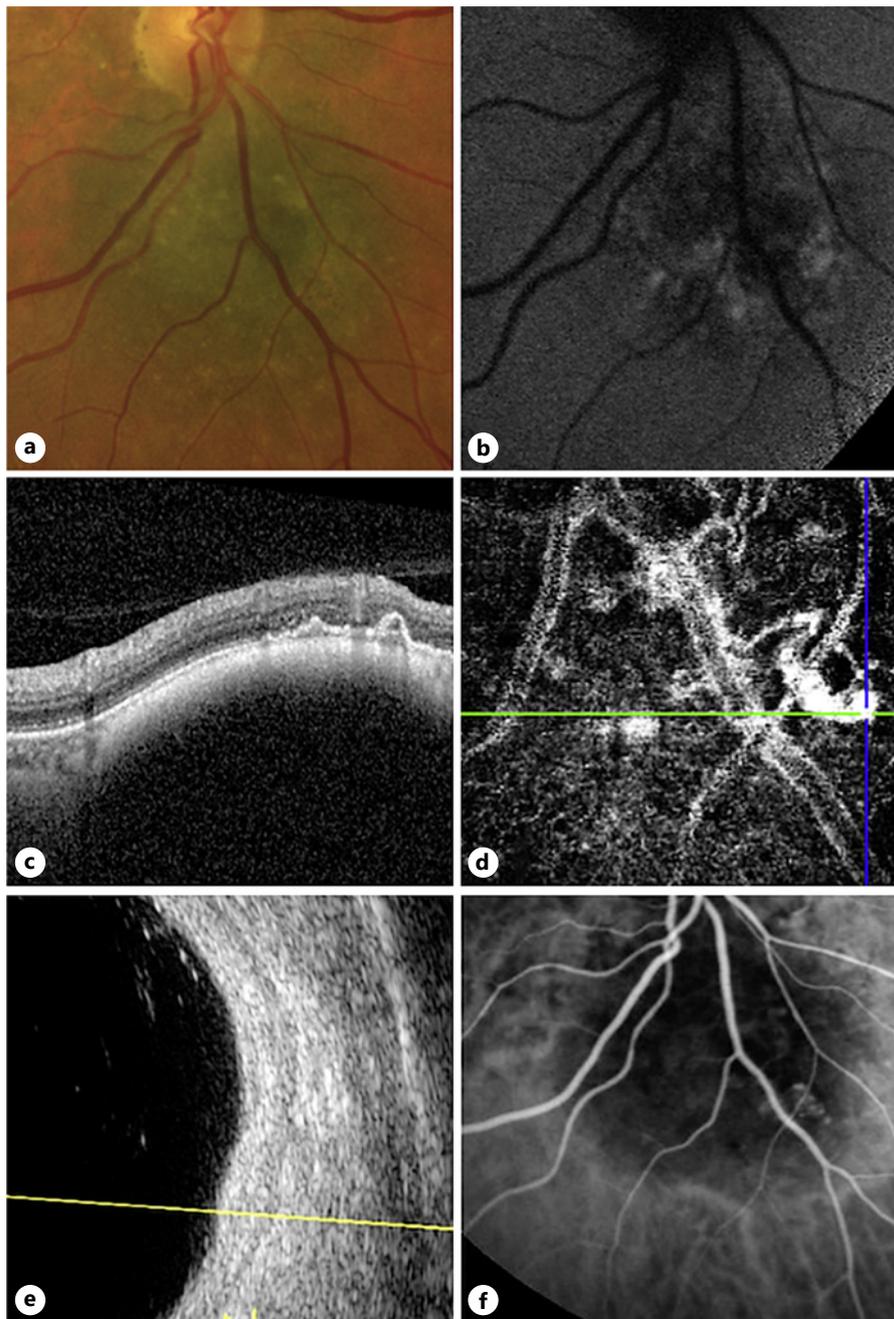


Fig. 3. **a** Fundus examination revealed flat subretinal pigmented juxtapapillary lesion of about 3.5 DD, with overlying drusen and a round red spot at its inferonasal margin. **b** Irregular BAF over the juxtapapillary lesion. **c** The OCT revealed multiple double layered PEDs. **d** OCT-A disclosed the presence of a BVN. **e** At B-scan, the choroidal lesion appeared dome-shaped, with high echogenicity. **f** ICGA highlighted the presence of multiple small and well-delimited bright lesions at the choroidal nevus border that persisted throughout the entire duration of the examination.

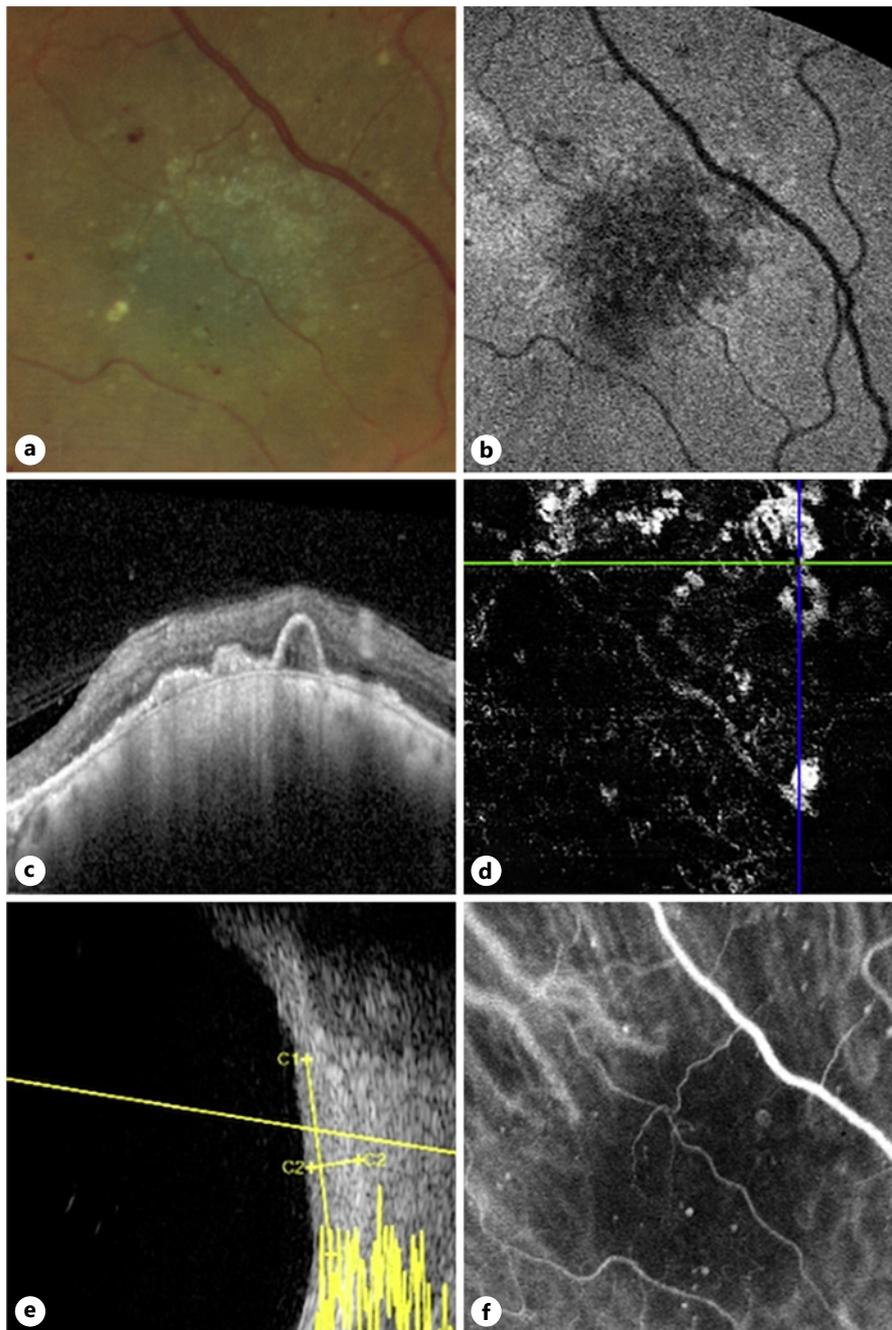


Fig. 4. **a** Fundus examination revealed a pigmented choroidal lesion, sized approximately 1–1.5 DD, supero-temporally to the optic disc. **b** irregular autofluorescence on BAF. **c** OCT highlighted a multiple sharp-peaked PED associated with minimum IRF. **d** OCT-A demonstrated a polypoidal lesion with terminal aneurysmal dilatations. **e** At B-scan, the choroidal lesion appeared dome-shaped, with high echogenicity. **f** The ICG-A showed multiple bright spots within the choroidal lesion.

Discussion

Choroidal nevi prevalence ranges between 4.6% and 7.9% while the presence of an associated contemporary PCV has been infrequently documented. However, PCV might be found in a higher proportion of nevi if specialized examinations were undertaken more

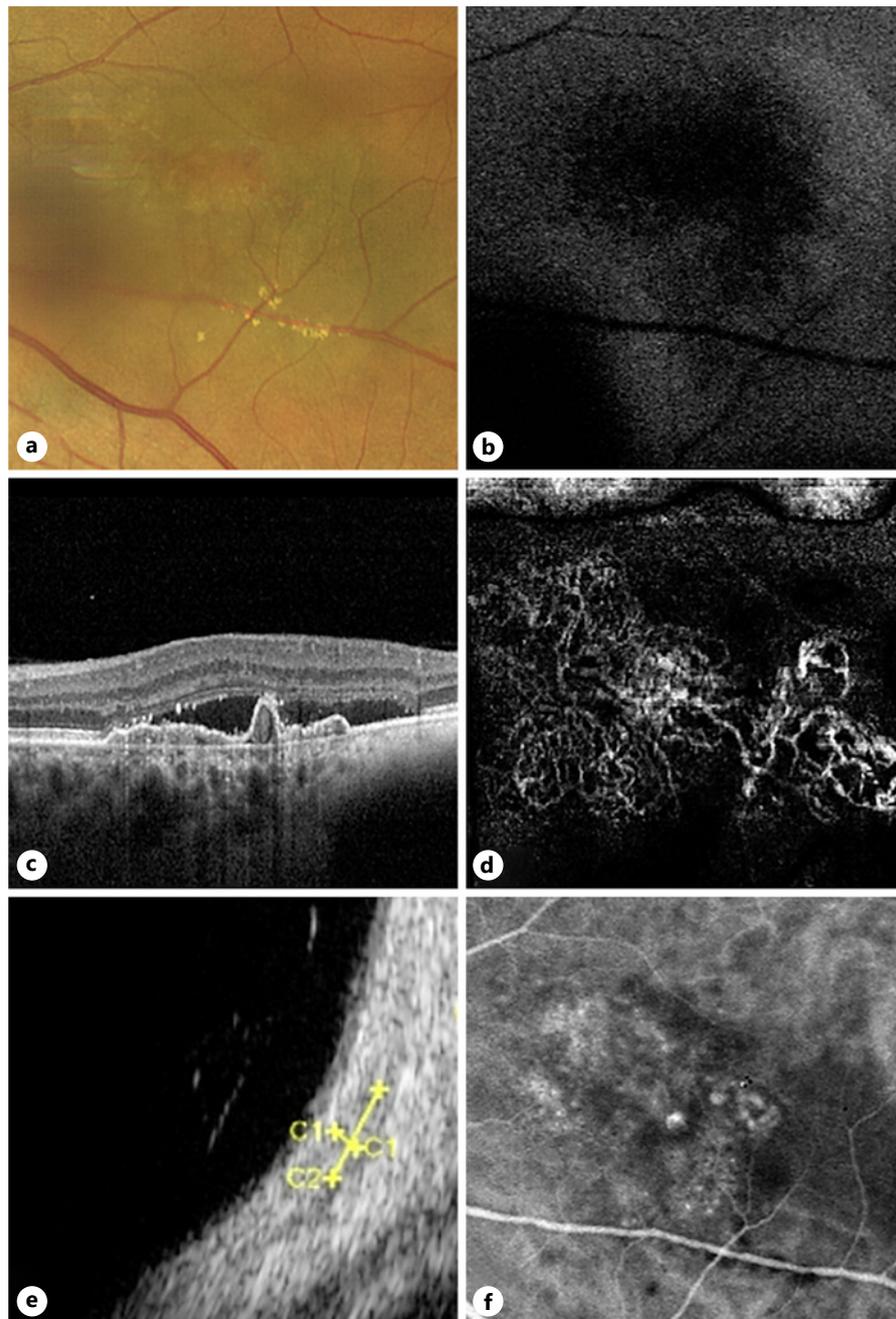


Fig. 5. **a** Fundus examination revealed a pigmented choroidal lesion, sized approximately 1.5 DD, inferotemporal to the macula. **b** Irregular autofluorescence on BAF. **c** OCT highlighted a multinodular PED associated with SRF. **d** OCT-A demonstrated a vast neovascular network. **e** At B-scan the lesion appeared with high echogenicity. **f** The ICG-A showed multiple bright spots within the choroidal lesion.

frequently [1, 23–30]. Recently, pachychoroid has emerged as a potential rationale for PCV formation, it is defined by the presence of dilated choroidal vessels that can lead to compression of the choriocapillaris and increased vascular permeability due to the vessels enlargement [27, 31–33].

Lee et al. [34] proposed that PCV formation has been linked to increased vascular permeability resulting from the expansion of Haller vessels. This could also happen in the case

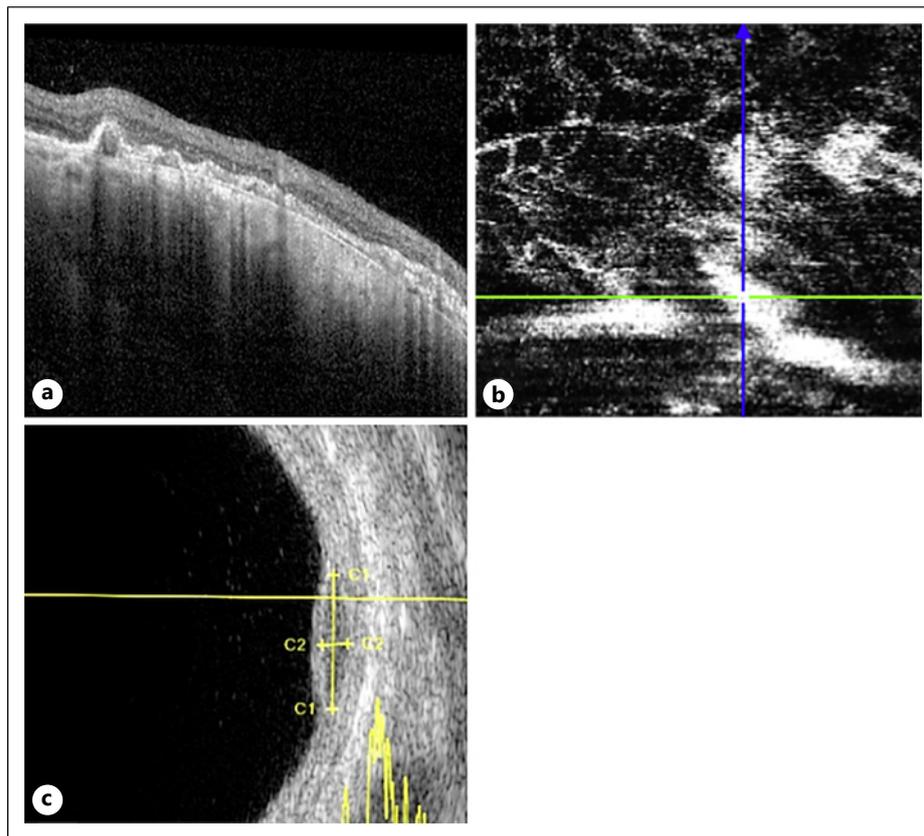


Fig. 6. **a** OCT scan over the lesion revealed the presence of a peaked PED containing thin-walled polypoidal lesions and double layer sign. **b** OCT-A over the nevus revealed the presence of a BVN characterized by terminal aneurysmal dilatations consistent with PCV. **c** At B-scan the choroidal lesion appeared dome-shaped, with high echogenicity.

of choroidal nevi that can cause chronic compression of the choroidal vasculature, leading to an alteration of choroidal hemodynamics with a dilatation of Haller's and a thinning of Sattler's vessels [35, 36]. The alterations mentioned could promote the formation of type 1 MNV, ultimately resulting in the emergence of PCV adjacent to the melanocytic lesion [37]. Various authors have highlighted the association between PCV and choroidal nevi, supporting the relevance of this correlation [5, 38, 39].

Gomel et al. [5], in a multicenter study involving 14 patients (with a mean follow-up of 9 years) with choroidal nevi associated with PCV, proposed that the compression caused by nevi on the choroidal vasculature can lead to choriocapillaris loss and, consequently, the formation of polyps. In a paper published in 2018, Anzidei [38] highlighted that unusual choroidal blood vessel patterns appear to cause alterations in the RPE resulted in Bruch's membrane disruption, leading to the formation of neo-vascular membranes.

Contrary to the pathogenic mechanism proposed by the previous authors, Wong and collaborators [39] highlighted that the RPE can probably experience chronic degenerative or inflammatory changes, due to the presence of a choroidal nevus, leading to the neo-vascularization formation. In cases without exudation, lesions were observed, while in the presence of exudation, reported treatments included: photodynamic therapy, single IV anti-VEGF injections, or a combination of both; a single case reported by Querques et al. success with focal laser photocoagulation [11, 18–21].

In all our clinical cases, it is evident that the choroidal Haller's vessels adjacent to the nevus are more dilated compared to normal whereas Sattler's vessels appear thinner than usual. Choroidal nevi are known to be associated with chronic compression of the choroidal vasculature [35]. Although increased vascular permeability in enlarged Haller vessels has been proposed as a contributing factor to the formation of PCV, we suggest that ischemia caused by altered choroidal blood flow, rather than hyperpermeability of the Haller layer, plays a more significant role in the development of polypoidal lesions. However, not all choroidal nevi lead to the formation of PCV, indicating that there are likely unknown mechanisms at play, which warrant further investigation to fully understand the factors involved.

The fluorangiographic features in all our cases, both FAG and ICGA, are characteristic and diagnostic for PCV. ICGA revealed the presence of PCV as multiple rounded hypercyanescence lesions within the choroidal nevus, persisting throughout the examination. During the OCT examination, PEDs appeared sharp-peaked and multilobular, sometimes associated with SRF and/or IRF. Additionally, B-scan ultrasound showed the choroidal lesion as dome-shaped, hyperechogenic with medium internal reflectivity, without signs of choroidal excavation or shadowing. Three out of our six patients underwent IV injections with anti-VEGF agents resulting in a significant reduction and/or resolution of the IRF and SRF.

The use of multimodal imaging has allowed the early identification and follow-up of PCV and choroidal nevi [2,24]. Our observations revealed that choroidal nevi could instigate modifications in the outer retina, resulting in persistent alterations capable of triggering the formation of neovascularization.

The occurrence of a PCV alongside nevus is an uncommon complication. Findings from all exams were consistent across all cases, highlighting the potential link between PCV and choroidal nevus.

Statement of Ethics

This retrospective review of patients' data did not require ethical approval in accordance with local guidelines. Written informed consent was obtained from all patients for publication of the details of their medical case and any accompanying images. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material at <https://doi.org/10.1159/000543643>.

Conflict of Interest Statement

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding Sources

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Author Contributions

Andrea Gaggino: conceptualization, investigation, and methodology. Leandro Infrerra: methodology, project administration, and writing. Serena Milan: investigation and writing. Marianna Presotto: writing and formal analysis. Daniele Tognetto: investigation and supervision.

Data Availability Statement

The data are not publicly available due to privacy concerns but can be obtained upon request from the University of Trieste by contacting the corresponding author. Further inquiries can be directed to the corresponding author.

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