

Management of peripheral polypoidal choroidal vasculopathy with intravitreal bevacizumab and indocyanine green angiography-guided laser photocoagulation

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A 69-year-old lady presented with complaints of decreased vision in left eye since one month. Best Corrected Visual Acuity (BCVA) was 6/18 in that eye. Fundus examination revealed non-central geographic atrophy and soft drusens at macula in both eyes. Temporal periphery of left eye revealed subretinal exudates with altered sub-RPE hemorrhage mimicking peripheral exudative hemorrhagic chorioretinopathy (PEHCR). Fundus Fluorescein Angiogram showed window defects at macula and blocked fluorescence at temporal periphery in left eye. However, Indocyanine green angiography (ICGA) revealed active peripheral choroidal polyps. The patient was successfully treated with intravitreal bevacizumab and ICGA-guided laser photocoagulation. 27 months after laser treatment, BCVA improved to 6/9. Rationale of consecutive anti-vascular endothelial growth factor (VEGF) treatment followed by more definitive laser photocoagulation is that anti-VEGF aids in resolution of subretinal fluid, thus making the polyp more amenable to focal laser photocoagulation which stabilizes the choroidal vasculature and prevents further leakage.

Key words: Indocyanine green angiogram, intravitreal bevacizumab, laser photocoagulation, peripheral hemorrhagic exudative chorioretinopathy, polypoidal choroidal vasculopathy

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Idiopathic polypoidal choroidal vasculopathy (PCV) was first described by Yannuzzi and colleagues as a vascular disorder originating from the choroidal circulation.^[1] However, the pathogenesis of PCV remains unclear. Recent genetic research has shown that PCV and age-related macular degeneration (AMD) share common genetic factors, which may suggest that although PCV and wet AMD have different clinical characteristics, they may share similar pathophysiologic aspects.^[2] PCV resembles neovascular AMD; however, the clinical course and visual outcomes of PCV are more stable, more favorable, and different than those of AMD.^[3] Peripheral exudative hemorrhagic chorioretinopathy (PEHCR) is typically characterized by advanced age at presentation (60–91 years with a mean age of 71 years), female preponderance (69%), frequent pigment epithelium detachment, temporal equatorial location, and a highly hemorrhagic and exudative presentation, sometimes extending to the macula.^[4] Bilateral involvement is seen in 24% cases. Lesions of PEHCR are seen in elderly people, especially with hypertension or atherosclerotic artery disease.

In our case report, we arrived at a provisional diagnosis of PEHCR based on the clinical picture and fundus fluorescein angiography (FFA). However, indocyanine green angiography (ICGA) provided us with valuable insight to refine the diagnosis and aided us in treating the peripheral polyps. This is the first report of peripheral PCV from an Indian subject (PubMed search) and further re-emphasizes the role of ICGA in diagnosis and treatment of PCV.

Case Report

A 69-year-old lady presented with diminution of vision in the left eye since a month. On examination, Best Corrected Visual Acuity (BCVA) in the right eye was 20/30 and in the left eye was 20/60. Anterior segment examination was unremarkable except for early nuclear sclerosis in both eyes. Intraocular pressures were 16 mmHg in both eyes. Fundus examination of both eyes revealed non-central geographic atrophy and soft drusens at the macula. Temporal periphery of the left eye revealed subretinal exudation with altered subretinal hemorrhage and subretinal pigment epithelium (RPE) hemorrhage [Fig. 1]. FFA revealed window defects in the macula corresponding to RPE atrophy, in both eyes. Blocked fluorescence was noted at the temporal periphery in left eye due to subretinal and sub-RPE hemorrhage. A provisional diagnosis of PEHCR

was arrived at. However, ICGA revealed the presence of multiple hyperfluorescent polypoidal lesions in the temporal periphery at the choroidal level, suggestive of PCV [Fig. 1]. A crossover run showed no such vascular abnormality in the fellow eye. After obtaining an informed consent and explaining about its “off-label” use, the patient underwent intravitreal bevacizumab (Avastin®, Genentech labs, San Diego, CA, USA) (1.25 mg in 0.1 ml) injection in her left eye. Five weeks hence, her BCVA improved to 20/40. Fundus examination revealed a significant reduction of subretinal hemorrhage and exudates in the left eye. ICGA was repeated and showed persistent leakage from peripheral choroidal polyps in the left eye. The patient underwent repeat intravitreal bevacizumab injection in the left eye. Six weeks later, her BCVA improved to 20/25. When reviewed 2 months later, her clinical condition showed significant improvement while ICGA showed persistent leakage from the peripheral polyps in the left eye [Fig. 2]. ICGA-guided laser photocoagulation was done to the peripheral polyps with a slit-lamp laser delivery system. Laser treatment was aimed at occluding the leaking, peripheral polyps. Two months later, her clinical condition further improved and ICGA showed no evidence of any active polyp in the left eye [Fig. 3]. She was then followed up quarterly; fundus examination was stable and BCVA maintained. At the last review, 27 months after laser treatment, her BCVA was 20/30 in both eyes [Fig. 4].

Discussion

Choroidal vascular lesions of PCV are preferentially found at the posterior pole of the retina,^[5] although the lesions could also be found in the peripheral retina in up to 63% eyes.^[3,6] Idiopathic PCV is a peculiar vascular abnormality of the inner choroid, composed of network of branching vessels terminating in aneurysm-like enlargements with episodic serosanguineous detachments of the retinal pigment epithelium and neurosensory retina. RPE detachments can be associated with choroidal polyps. When PCV lesions accompany PED, the lesions are usually located at the margins of PED.^[6] The network of vessels usually emanates from the peripapillary area or less commonly as an isolated macular lesion.^[5] The vascular abnormality is most clearly discernible using ICGA for enhanced choroidal imaging.^[2]

PEHCR is a characteristic peripheral degenerative disorder where chorioretinal lesions lead to small or large lesions of poorly defined accumulations of subretinal or sub-RPE hemorrhage, exudation, or both. Lesions are seen in temporal quadrant twice as commonly as in nasal quadrant. Frequently, it has a benign outcome, although it can be vision threatening because of hemorrhage or exudation. Basic pathology in PEHCR is formation of neovascular membrane. Anatomical differences may be responsible for the contrasting appearance of lesions in the macula and in the retinal periphery. PEHCR can be associated with disciform macular degeneration, which is a common macular disorder seen in elderly; rarely it can occur in the periphery. PEHCR is a relatively uncommon disorder of the peripheral retina. There is a paucity of such reports in the literature, although its exudative and hemorrhagic presentations account for 8% and 5%, respectively, of pseudo-melanomas of the posterior uvea.^[7] ICGA is an excellent diagnostic tool for visualizing and monitoring choroidal polyps. Bevacizumab has anti-angiogenic and anti-permeability effects that help in

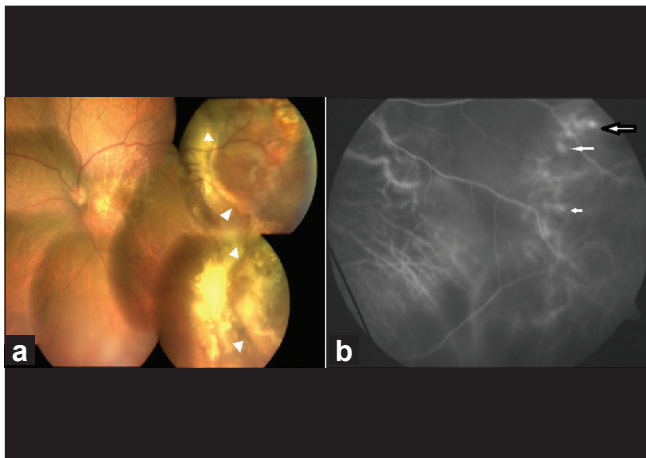


Figure 1: At presentation. (a) Color fundus photograph of the left eye reveals subretinal altered hemorrhages and mounds of sub-RPE hemorrhage (arrowheads) in the temporal periphery. (b) Indocyanine green angiography reveals multiple, peripheral, discrete, hyperfluorescent, polypoidal lesions (arrows) at the choroidal level, suggestive of idiopathic polypoidal choroidal vasculopathy

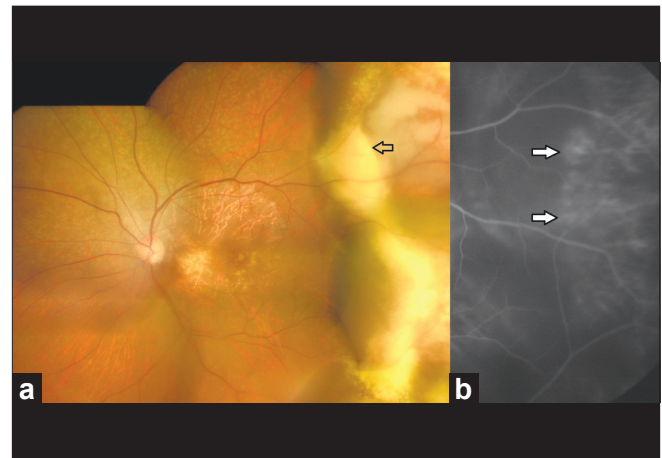


Figure 2: Six weeks after second bevacizumab injection. Color fundus montage (a) shows consolidation of hard exudates and reduction in the sub-RPE mounds of hemorrhages in the temporal periphery (arrow). Indocyanine green angiography (b) reveals persistent polyps (arrows)

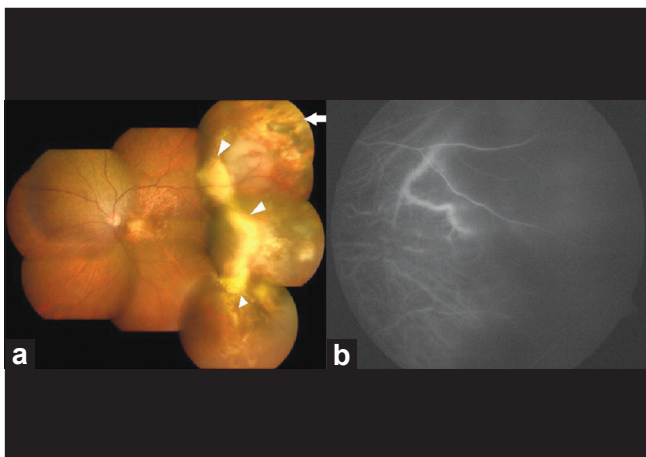


Figure 3: Two months after laser photocoagulation. (a) Color fundus photograph of the left eye reveals significantly reduced subretinal altered hemorrhages (arrowheads). Laser photocoagulation marks are seen in the area treated (arrow). (b) Indocyanine green angiography shows no leak from the area of treated polyps

resolution of subretinal exudation. Laser photocoagulation of the choroidal polyps leads to thrombosis and occlusion, with cessation of leakage.^[8]

Rationale of sequential anti-vascular endothelial growth factor (anti-VEGF) treatment followed by more definitive laser photocoagulation is that the anti-VEGF agent aids in resolution of subretinal fluid, thus making the polyp more amenable to focal laser photocoagulation. Moreover, laser photocoagulation helps in vaso-occlusion of the polyps and regression of vascular network associated with PCV without the inherent limitations of intravitreal anti-VEGF agents. Alternately, peripheral lesions can also be treated with trans-scleral cryopexy. However, cryopexy carries the disadvantage of inducing marked inflammation.

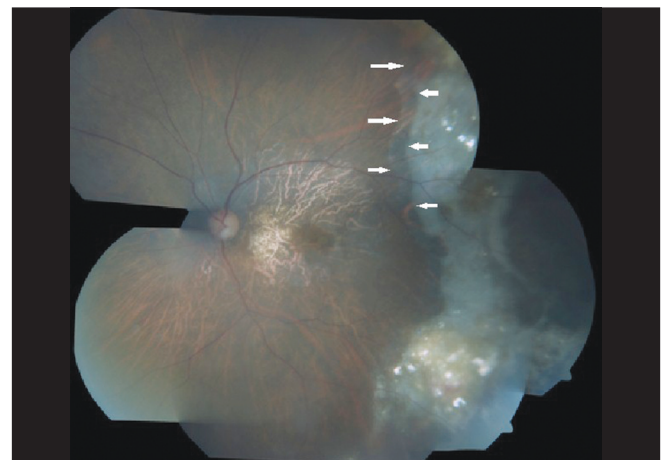


Figure 4: At 27 months follow-up, color fundus photograph of the left eye reveals "water-mark" area of RPE atrophy (right pointing arrows) and a subretinal scar (left pointing arrows)

To conclude, sequential intravitreal bevacizumab and ICGA-guided laser treatment of peripheral polyps seems to stabilize and improve the treatment outcomes in eyes with peripheral PCV as it stabilizes the choroidal vasculature and prevents further leakage.

References

1. Yannuzzi LA, Sorenson J, Spaide RF, Lipson B. Idiopathic polypoidal choroidal vasculopathy (IPCVC). *Retina* 1990;10:1-8.
2. Yannuzzi LA, Nogueira FB, Spaide RF, Guyer DR, Orlock DA, Colombero D, *et al.* Idiopathic polypoidal choroidal vasculopathy: a peripheral lesion. *Arch Ophthalmol* 1998;116:382-3.
3. Gomi F, Tano Y. Polypoidal choroidal vasculopathy and treatments. *Curr Opin Ophthalmol* 2008;19:208-12.
4. Mantel I, Uffer S, Zografos L. Peripheral exudative hemorrhagic chorioretinopathy: a clinical, angiographic, and histologic study. *Am J Ophthalmol* 2009;148:932-8.
5. Cackett P, Wong D, Yeo I. A classification system for polypoidal

- choroidal vasculopathy. *Retina* 2009;29:187-91.
6. Lafaut BA, Aisenbrey S, Van den Broecke C, Bartz-Schmidt KU, Heimann K. Polypoidal choroidal vasculopathy pattern in age-related macular degeneration: a clinico-pathologic correlation. *Retina* 2000;20:650-4.
 7. Shields JA, Mashayekhi A, Ra S, Shields CL. Pseudomelanomas of the posterior uveal tract: the 2006 Taylor R. Smith Lecture. *Retina* 2005;25:767-71.
 8. Anantharaman G, Ramkumar G, Gopalakrishnan M, Rajput A. Clinical features, management and visual outcome of polypoidal choroidal vasculopathy in Indian patients. *Indian J Ophthalmol* 2010;58:399-405.
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