

Case Report

Fovea Sparing Branch Retinal Artery Occlusions Imaged with Optical Coherence Tomography Angiography: Two Case Reports

Benjamin R. Lin Philip J. Rosenfeld Harry W. Flynn Jr.

Department of Ophthalmology, Bascom Palmer Eye Institute, Miami, FL, USA

Keywords

Optical coherence tomography · Optical coherence tomography angiography · Branch retinal artery occlusion · Case report

Abstract

Introduction: Central retinal artery occlusions and branch retinal artery occlusions (BRAOs) are ophthalmic emergencies that require workups for systemic risk factors. In the acute setting, BRAOs present with retinal whitening in a sectoral pattern on exam as well as hyperreflectivity and thickening of the inner retinal layers on optical coherence tomography (OCT). In the subacute to chronic phase, the retinal whitening dissipates, which may confound the diagnosis of remote arterial occlusions if there is no clearly visible plaque. **Case Presentations:** A 66-year-old male presented with 20/25 visual acuity (VA) and an inferior visual field defect in the right eye, and a 69-year-old male presented with 20/60 VA and a superior visual field defect in the left eye. Exams of both patients showed ischemic retinal whitening with visible Hollenhorst plaques in the affected eyes. OCT demonstrated inner retinal edema. At follow-up, wide-field OCT angiography (OCTA) showed persistent capillary dropout following the same initial vascular distribution but sparing the fovea and papillomacular bundle. VAs at the most recent follow-up visits were 20/30 and 20/20, respectively. **Conclusion:** These cases demonstrate the utility of wide-field OCTA in characterizing areas of capillary nonperfusion that can persist for years after the initial ischemic event. Additionally, patients with macula-involving BRAOs can have good VA outcomes if the fovea is spared.

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Correspondence to:
Harry W. Flynn, hflynn@med.miami.edu

Introduction

Retinal artery occlusions (RAOs) are ophthalmic emergencies that require workup for systemic risk factors in order to avoid secondary adverse vascular events. While RAOs are most commonly due to embolic events, numerous non-embolic etiologies include infections, inflammatory conditions, coagulopathies, malignancy, and trauma [1–6]. Central retinal artery occlusions (CRAOs) often are associated with poor visual outcomes, but branch retinal artery occlusions (BRAOs) are more likely to present with good central vision due to macular and/or foveal sparing [7]. Even with preserved central vision, patients with BRAOs often present with visual field deficits, which may include central scotomas, altitudinal defects, or other partial peripheral visual field defects [8].

A BRAO is usually associated with ischemia, which results in edema and retinal whitening in the distribution of the BRAO [9]. In BRAOs, this retinal whitening presents as a sectoral pattern, which follows a vascular distribution. Optical coherence tomography (OCT) typically demonstrates hyperreflectivity and thickening of the inner and middle retinal layers indicative of edema secondary to ischemia. Within 4–6 weeks after the inciting event, the retinal whitening dissipates on exam and OCT begins to demonstrate retinal atrophy, which most prominently affects the inner retinal layers [9, 10]. While fluorescein angiogram (FA) can be used to characterize the areas of retinal vascular nonperfusion, optical coherence tomography angiography (OCTA) can also readily demonstrate areas of vascular dropout and is less invasive with fewer risks. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material (for all online suppl. material, see <https://doi.org/10.1159/000543742>).

Case Reports

Case 1

A 66-year-old male presented with a partial inferior visual field defect in the right eye. The initial visual acuity (VA) was 20/25 and intraocular pressure (IOP) was 12. The patient previously had a macula-involving retinal detachment repaired 4 years prior. Exam revealed an attached retina with a zone of retinal whitening involving the superior macula in the distribution of a branch from the superotemporal arcade, which contained a visible Hollenhorst plaque (Fig. 1a). OCT revealed edema of the inner retinal layers corresponding to the area of retinal whitening (Fig. 1b, c). Humphrey visual field 10-2 of the right eye revealed an inferior paracentral scotoma (Fig. 1g). At the most recent follow-up 10 years after the initial BRAO, VA was 20/30 with correction. The retinal whitening was resolved on exam, but OCT was notable for atrophy of the inner retinal layers in the areas corresponding to prior ischemia (Fig. 1d–f). OCTA captured with the DREAM VG-200 OCT platform (Intalight; San Jose, CA, USA) revealed persistent capillary dropout in the same vascular distribution (Fig. 1h).

Case 2

A 69-year-old male presented with sudden-onset painless superior visual field loss in his left eye. Their VA at presentation was 20/60 and IOP was 15. Exam revealed an area of retinal whitening involving the inferior macula and following a vascular distribution. There was a visible Hollenhorst plaque at the proximal edge of the whitening (Fig. 2a). OCT demonstrated diffuse inner retinal edema in the inferior retina (Fig. 2b, c). At the most recent follow-up 18 months after the initial BRAO, VA had improved to 20/20. On exam, the retinal whitening was resolved, and the proximal portion of the inciting vessel appeared to have a large plaque

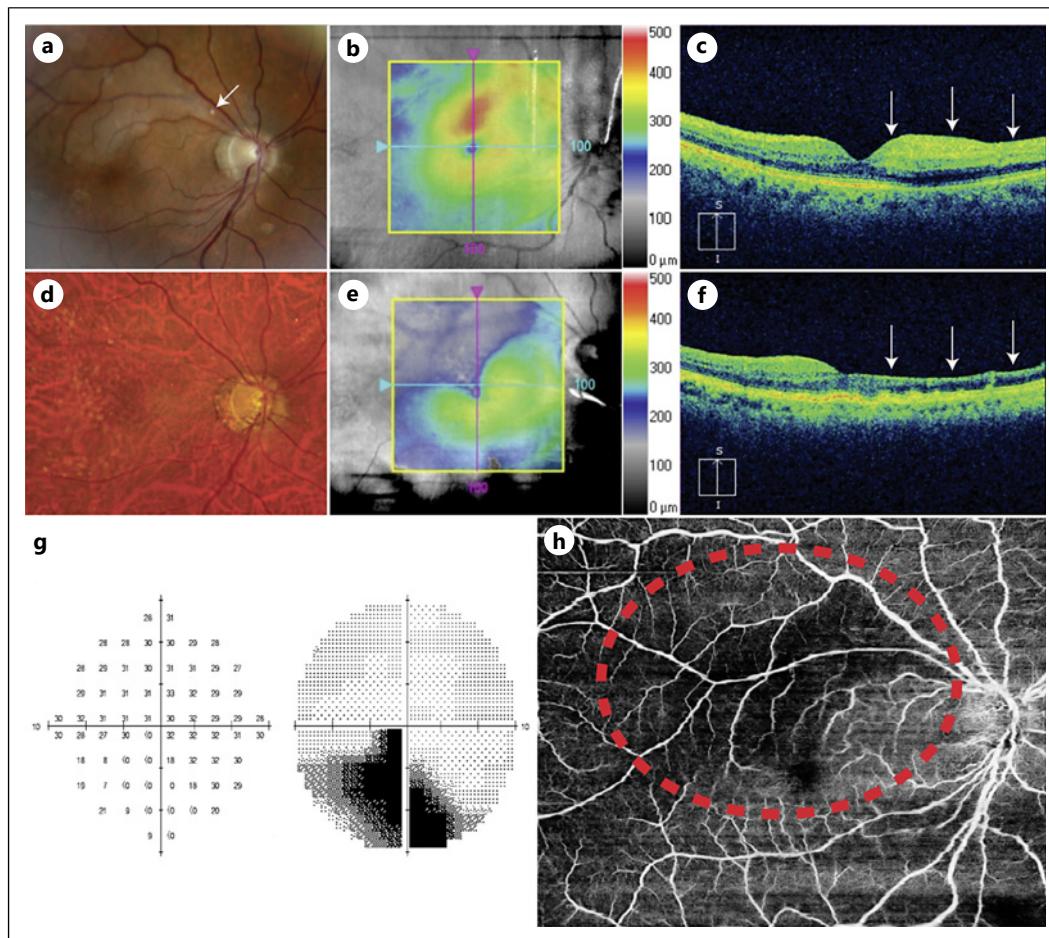


Fig. 1. Multimodal imaging for case 1. **a** Initial exam demonstrates a focal area of retinal whitening in a vascular distribution in the superior macula with a visible Hollenhorst plaque (white arrow). **b** *En face* retinal thickness map overlay demonstrates thickening in the superior macula. **c** OCT vertical B-scan demonstrates marked inner and middle retinal hyperreflectivity and edema corresponding to the area of retinal whitening (white arrows). **d** At the 10-year follow-up visit, the retinal edema noted on exam has resolved. **e** *En face* retinal thickness map overlay demonstrates thinning in the superior macula. **f** OCT vertical B-scan demonstrates diffuse inner retinal atrophy in areas of prior retinal edema (white arrows). **g** Visual field at the time of presentation demonstrates a partial inferior paracentral scotoma. **h** *En face* OCT angiography is notable for an ongoing area of nonperfusion corresponding to the prior area of retinal ischemia (red dotted circle).

(Fig. 2d). OCT demonstrated inner retinal atrophy associated with the areas of prior retinal edema (Fig. 2e, f). Wide-field OCTA on the DREAM VG-200 OCT platform revealed persistent capillary dropout in the same distribution (Fig. 2g).

Conclusions

In the setting of an RAO, initial VA is correlated with final VA [7, 11]. Patients with BRAOs are much more likely to present with preserved VA compared with patients with CRAO due to the frequent foveal sparing in BRAOs [7]. Ischemic damage to the papillomacular bundle has also been demonstrated to be a poor prognostic factor for vision in patients with BRAOs [12].

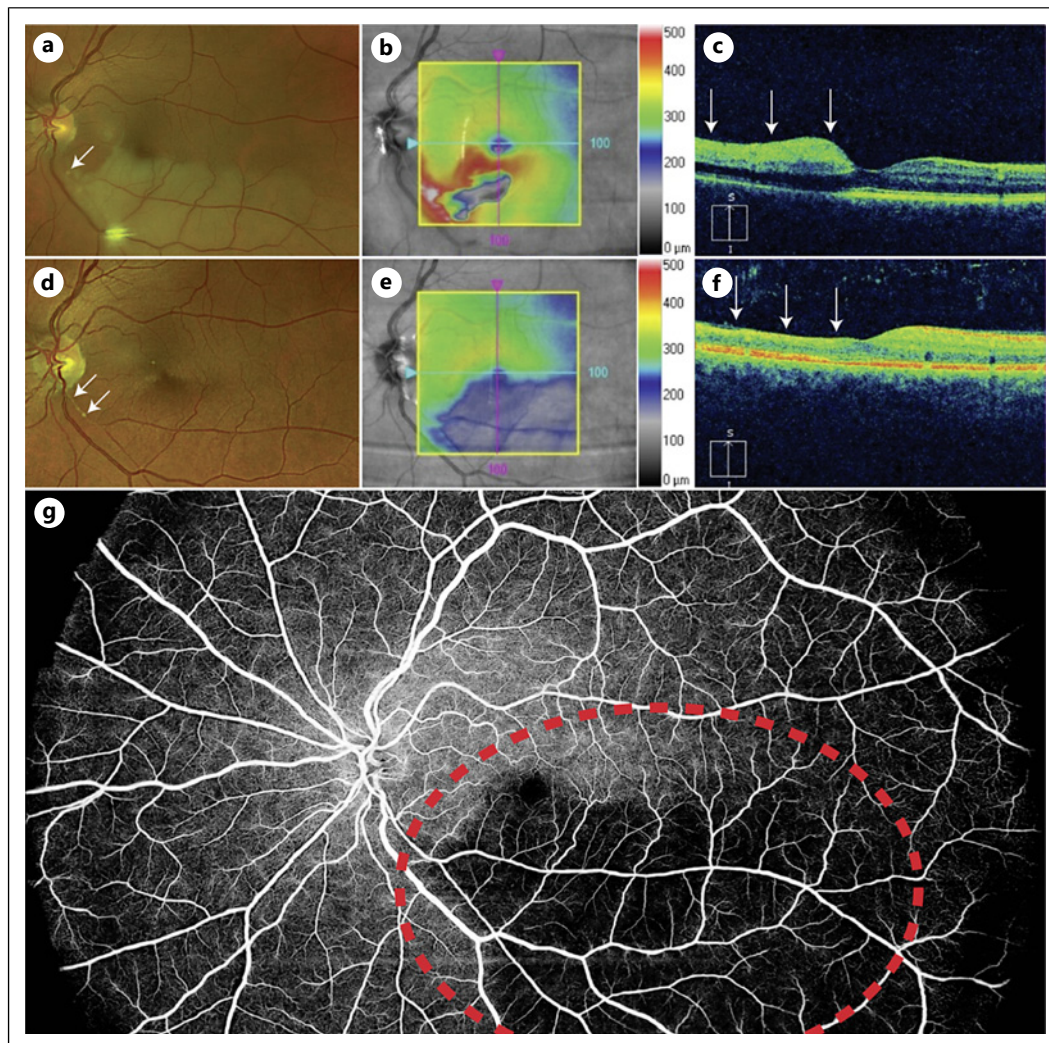


Fig. 2. Multimodal imaging for case 2. **a** Initial exam demonstrates a focal area of retinal whitening in a vascular distribution in the inferior macula with a visible Hollenhorst plaque (white arrow). **b** *En face* retinal thickness map overlay demonstrates thickening in the inferior macula. **c** OCT vertical B-scan demonstrates marked inner and middle retinal hyperreflectivity and edema corresponding to the area of retinal whitening (white arrows). **d** At 1-year follow-up, the retinal edema noted on exam has resolved with a persistent large plaque within the proximal vessel (white arrows). **e** *En face* retinal thickness map overlay demonstrates thinning in the inferior macula. **f** OCT vertical B-scan demonstrates diffuse inner retinal atrophy in areas of prior retinal edema. **g** *En face* OCT angiography is notable for an ongoing area of nonperfusion corresponding to the prior area of retinal ischemia (red dotted circle).

In both patients presented herein, the acute retinal edema and whitening along with the subsequent atrophy extended up to but did not involve the fovea. Additionally, both areas of ischemia spared the papillomacular bundle. These clinical characteristics allowed both patients to maintain relatively good central VA with long-term follow-up.

In both patients, the larger veins running through the affected areas of retina remained perfused despite the diffuse small capillary dropout in those areas. These veins are likely fed by the unaffected retina distal to the BRAO. A prior study by Venkatesh et al. [13] utilizing FA suggests that these veins may also be filled by either capillaries from the unaffected retina

crossing the horizontal raphe or by arteriovenous anastomoses connecting the retinal arteries in the unaffected segments to the retinal veins in the affected segments.

In both patients, visible Hollenhorst plaques were present at the proximal edge of the ischemic area. Even with a clear inciting plaque, there is no level 1 evidence supporting any specific treatment for RAOs. Interventions that have previously been investigated include decreasing IOP to improve ocular perfusion, digital massage to propagate the clot distally to reduce the ischemic area, vasodilation to increase blood flow, and hyperbaric oxygen to increase blood oxygen saturation [14, 15]. Intra-arterial and intravenous thrombolytics have also been studied, but effectiveness is limited by the short time of intervention required between onset of symptoms and intervention as well as complications such as intraocular or intracranial hemorrhage, transient ischemic attacks, and strokes [16–18].

Although there is no established treatment for RAOs, it is important to complete a systemic workup. In a study by Lauda et al. [19] investigating a series of consecutive patients with CRAOs, BRAOs, and amaurosis fugax, 23% of patients had concomitant acute brain infarcts on additional workup. Furthermore, the majority (89.8%) of those patients were asymptomatic except for their vision loss. An underlying etiology of the stroke was identified in 41.8% of individuals – the most common of which were large artery atherosclerosis and cardioembolism. Another study by Hayreh et al. [20] investigating non-arteritic CRAOs also demonstrated high incidences of internal carotid plaques and abnormal echocardiograms suggesting embolic sources in this population. Both patients presented herein also reflect the importance of a systemic workup in the settings of RAOs. Patient 1 was found to have atrial fibrillation and started on apixaban and patient 2 was found to have bilateral carotid artery plaques, left greater than right.

While OCT demonstrates the retinal edema and subsequent atrophy that occurs as a result of capillary ischemia, both FA and OCTA can be used to directly visualize the vasculature to demonstrate areas of nonperfusion [21]. The primary benefit of OCTA over FA is the noninvasive nature of OCTA without the need for fluorescein dye. The most common side effects from FA include nausea, syncope, and a vasovagal response [22]. Rarely, true allergic reactions occur and can lead to itching, hives, edema, anaphylaxis, and even death [23]. These 2 patients described demonstrate the utility of noninvasive OCTA. The areas of chronic capillary dropout can be seen sparing the fovea. While early OCTA imaging studies were limited by field of view and resolution, newer imaging platforms such as the one used herein rival wide-field FA studies but with a lower risk profile. OCTA should be considered as a first-line imaging modality when seeking to evaluate areas of vascular nonperfusion in patients with RAOs. Finally, these 2 patients also demonstrate that vascular dropout in the areas of prior RAOs can persist for years after the initial vascular insult.

Statement of Ethics

Ethical approval is not required for this study in accordance with local or national guidelines. Written informed consent was obtained from the patients for publication of the details of their medical case and any accompanying images.

Conflict of Interest Statement

Dr. Philip Rosenfeld received research support and is a consultant for Carl Zeiss Meditec. However, no author received any direct financial support for the research, authorship, and/or publication of this article.

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Author Contributions

Benjamin R. Lin, MD, and Harry W. Flynn Jr, MD: conceptualization, data curation, formal analysis, investigation, methodology, writing – original draft, and writing – review and editing. Philip J. Rosenfeld, MD, PhD: conceptualization, data curation, formal analysis, investigation, methodology, and writing – review and editing.

Data Availability Statement

All data generated or analyzed during this study are included in this article and its online supplementary material. Further inquiries can be directed to the corresponding author.

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