Longitudinal Optical Coherence Tomography Angiography Findings in Malignant Hypertension Choroidopathy: A Case Report

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Abstract
This report presented the longitudinal optical coherence tomography angiography (OCTA) findings in a patient with malignant hypertension choroidopathy. An 87-year-old woman presented with acute unilateral central vision loss in the setting of hypertensive emergency. Spectral domain optical coherence tomography showed massive serous macular detachment. OCTA revealed extensive flow loss in the neuroretina and choriocapillaris. With blood pressure (BP) normalization and without any ocular intervention, visual acuity recovered on subsequent visits. Flow loss in the neuroretinal capillaries persisted, but significant improvement in the perfusion of the choriocapillaris was observed. This case demonstrates extensive choriocapillaris flow loss in the acute phase of malignant hypertension, and a temporal relationship between BP normalization, improvement of choriocapillaris perfusion, and decrease of subretinal fluid, providing additional insight into the pathophysiology of this life- and sight-threatening systemic condition.
Introduction

Systemic malignant hypertension can manifest as retinopathy and choroidopathy. Herein, we report a case of hypertensive choroidopathy and demonstrate the longitudinal optical coherence tomography angiography (OCTA) changes in the choriocapillaris.

Case Presentation

An 87-year-old woman with uncontrolled systemic hypertension and a remote history of central retinal vein occlusion in the right eye (RE) presented with acute painless vision loss in the left eye (LE). RE Snellen best corrected visual acuity (BCVA) was 20/200 (baseline), examination revealed diffuse intraretinal lipid, ultrawide-field fluorescein angiography (FA) showed diffuse leakage in the macula, and spectral domain optical coherence tomography showed chronic atrophic changes in the central macula (Fig. 1a–c). In the LE, BCVA decreased from a baseline of 20/20 to 20/400. In addition, a relative afferent pupillary defect, vascular tortuosity, intraretinal hemorrhages, and Elschnig spots were observed (Fig. 1g). Ultrawide-field FA showed an enlarged foveal avascular zone, capillary dropout, and multifocal leakage (Fig. 1h). Spectral domain optical coherence tomography in the LE showed serous macular detachment with a central subfield thickness of 1,285 microns (Fig. 1i). Systemic work-up revealed a blood pressure (BP) of 273/121. A diagnosis of grade 3 hypertensive retinopathy was made prompting hospitalization of the patient for management of hypertensive emergency [1]. After 1-month follow-up, her BP was 142/72 mm Hg, her BCVA improved to 20/50 without any ophthalmic intervention. Most abnormalities were seen at presentation in her LE on color fundus photo and FA resolved. In addition, subretinal fluid in the macula improved dramatically (Fig. 1 j–l) and resolved by month three.

The patient’s OCTA imaging showed extensive abnormalities as well. At the 1-week follow-up visit, BCVA improved from 20/400 to 20/80, but OCTA showed extensive flow loss in both the choriocapillaris (Fig. 2a) and neuroretina (Fig. 2c). At her 3-month follow-up, BCVA in the LE further recovered to 20/40 with significant improvement in the choriocapillaris perfusion (Fig. 2b), and stable flow deficits in the neuroretina (Fig. 2d). BP reading at the 3-month follow-up was 146/80.

Discussion/Conclusions

Ocular manifestations of systemic malignant hypertension include optic neuropathy, retinopathy, and the less frequently recognized choroidopathy. Hypertensive choroidopathy may present as exudative retinal detachments and Elschnig spots, which are well-demarcated retinal pigment epithelium (RPE) changes due to choriocapillaris nonperfusion [2, 3]. The pathophysiology of hypertensive choroidopathy is thought to stem from fibrinoid necrosis of choroidal arterioles and breakdown of the blood-retinal barrier from loss of tight junctions between the RPE cells, which leads to subretinal fluid (SRF) accumulation [2, 3]. While indocyanine green angiography may confirm the presence of obvious choroidal hypoperfusion, OCTA allows for noninvasive, detailed imaging of the retinal and choroidal microvasculature in the macula.

We present a case of hypertensive retinopathy and choroidopathy, where vascular perfusion at the choriocapillaris improved with BP normalization. In response to systemic hypertension, choroidal arterioles undergo initial constriction [4]. This likely explains the decrease in choriocapillaris perfusion in the acute phase that led to RPE dysfunction and SRF accumulation in our
patient. In a rhesus monkey model of malignant hypertension, choroidal arterioles and choriocapillaris undergo recanalization over time [2, 5], which may explain the significant improvement in choriocapillaris perfusion at our patient’s 3-month follow-up visit. Interestingly, similar improvements were not observed in the neuroretina; however, the flow loss in the retinal capillaries may have existed prior to the current episode of hypertensive emergency. While hypertensive retinopathy is typically a bilateral process, a dramatic increase in subretinal fluid was only observed in the LE in our patient. On presentation with hypertensive emergency, our patient’s RE did show pathologic hypertensive changes, such as vascular tortuosity and diffuse intraretinal lipid. We hypothesize that the lack of SRF in the RE was related to the chronic atrophic changes in the central macula due to a remote episode of central retinal vein occlusion. The chronically damaged, atrophic retinal and/or choroidal tissue might have responded differently to the acutely elevated systemic BP, as compared to the LE.

Hypertensive choroidopathy typically occurs in younger individuals as vascular constriction requires pliable vessels [3]. Rezkallah et al. [6] described the use of OCTA in a
young patient with malignant hypertension to assess choroidal flow loss, which in that particular case was primarily extramacular and likely explained the mild SRF in the macula. In contrast, our patient is older and presented with choroidal flow loss in the central macula and massive central SRF, both of which improved with systemic BP normalization. An important limitation of OCTA imaging is the presence of artifacts, such as shadowing artifacts. It is well recognized that the accumulation of subretinal or intraretinal fluid may impose shadowing artifacts on the choriocapillaris [7], resulting in the perception of decreased choriocapillaris flow. We believe the loss of choriocapillaris flow in our patient could not be accounted for by the presence of SRF alone. Specifically, there were numerous areas of choriocapillaris flow loss on OCTA that did not co-locate with overlying fluid or shadowing artifacts. Sample areas are highlighted in Figure 3.

In conclusion, our case demonstrates extensive choriocapillaris nonperfusion during the acute phase of malignant hypertension and the temporal relationship between normalization of systemic BP, improvement in choriocapillaris flow and decrease in subretinal fluid, thus providing additional insight into the pathophysiology of this life- and sight-threatening systemic condition.

**Fig. 2.** 3 × 3 mm OCTA of the LE. Diffuse patchy choriocapillaris flow loss was observed at week 1 (**a**) that improved significantly by month 3 (**b**). Corresponding images of neuroretina were shown and demonstrated diffuse loss of flow at week 1 (**c**) that remained relatively unchanged at month 3 (**d**).
Statement of Ethics

This retrospective review of individual patient data did not require ethical approval in accordance with local guidelines. Written informed consent was obtained from the patient for publication of the details of their medical case and any accompanying images.

Conflict of Interest Statement

Narine Viruni, Jo-Hsuan Wu, and T.Y. Alvin Liu have no competing interests to disclose. Sally S. Ong served on an advisory board for Alimera.

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Author Contributions

Narine Viruni interpreted the patient data and was a major contributor in writing the manuscript. Sally S. Ong and Jo-Hsuan Wu revised the manuscript and helped with visualization. T.Y. Alvin Liu provided and interpreted the patient data, revised the manuscript, and supervised the whole process. Narine Viruni, Sally S. Ong, Jo-Hsuan Wu, and T.Y. Alvin Liu read and approved the final manuscript.
Data Availability Statement

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study, and all data that support the findings of this study are included in this article. Further enquiries can be directed to the corresponding author.

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