

Multimodal imaging of hypertensive chorioretinopathy by swept-source optical coherence tomography and optical coherence tomography angiography

Case report

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Abstract

Rationale: To investigate malignant hypertension ocular lesions with swept source optical coherence tomography (SS-OCT) and optical coherence tomography angiography (OCT-A).

Patient concerns: Visual loss due to malignant hypertension.

Diagnoses: Hypertensive chorioretinopathy.

Interventions: Patients were thoroughly examined on presentation and 30 days after their first visit, with swept-source optical coherence tomography and optical coherence tomography angiography.

Outcomes: Lesions were totally absorbed during the follow-up time. Additionally, they presented fibrin deposits, as multiple solid hyper-reflective structures overlying retinal pigment epithelium, on both—SS-OCT and OCT-A. The last were still detected even larger in size at the last visit of the patients.

Lessons: These novel imaging examinations allow the ophthalmologist to detect in detail the several clinical manifestations of malignant hypertension on the fundus, and draw useful conclusions about their peculiar pathogenesis.

Abbreviations: OCT = optical coherence tomography, OCT-A = optical coherence tomography angiography, PED = pigment epithelium detachment, RPE = retinal pigment epithelium, SRF = subretinal fluid, SS-OCT = swept-source optical coherence tomography.

Keywords: chorioretinopathy, Elsching spots, malignant hypertension, optical coherence tomography angiography, swept-source OCT

1. Introduction

Malignant (accelerated) arterial hypertension is a life-threatening condition resulting from acute elevation of systemic blood pressure (BP) which may lead to severe complications involving the heart, brain, kidneys, eyes, and other organs. Acute hypertension can induce a number of fundus changes, including retinopathy, choroidopathy, and neuropathy, resulting in severe visual deterioration.^[1–13] Sudden vision loss can be the first and only symptom in these patients.^[14,15]

Herein, we describe 2 patients, who attended to the ophthalmic emergency department of our clinic with severe bilateral visual deterioration as the first symptom of acute hypertension. They were thoroughly examined in our department, including swept-source optical coherence tomography (SS-OCT) and optical coherence angiography (OCT-A) examinations, at their first visit and 1 month later. To the best of our knowledge, this is the first report of a presentation of SS-OCT and OCT-A findings in patients with accelerated hypertensive chorioretinopathy.

2. Methods

Both patients involved were provided a written informed consent in accordance with the tenets of the Declaration of Helsinki to having their medical data used for research purposes. The study was approved by the institutional review board of “Georgios Gennimatas” Hospital.

Both patients underwent a thorough ophthalmological examination on presentation, which included Snellen visual acuity assessment, fundoscopy, autofluorescence (SW-FAF), SS-OCT, and OCT-A. All the above tests were repeated 30 days after baseline examination.

For image acquisition, we used the DRI OCT Triton system (Topcon Co, Tokyo, Japan) able to acquire 100,000 A-scans per second with a 2.6 μm axial and 20 μm lateral resolution. The system utilizes the incorporated SMARTTrack functionality to

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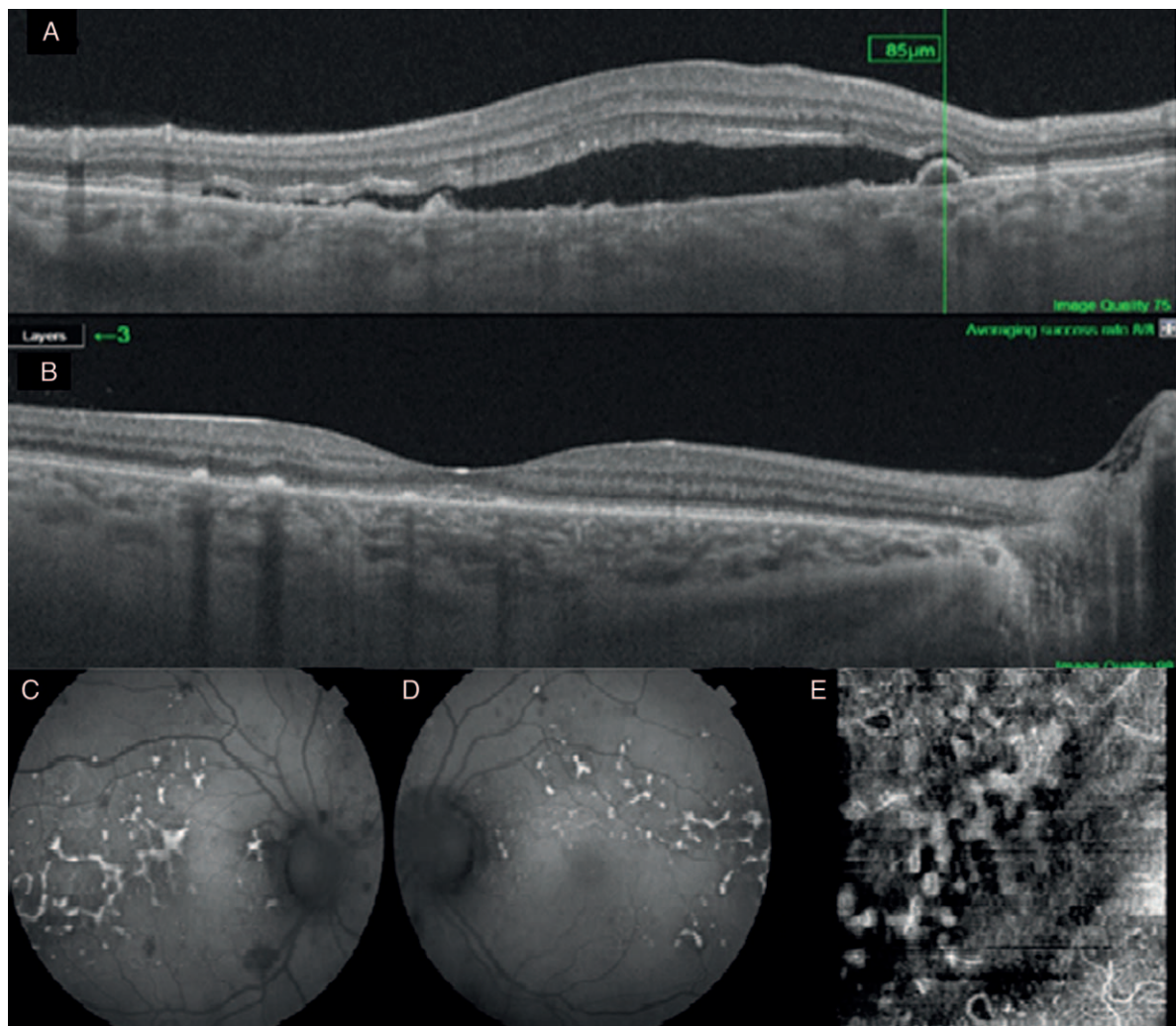


Figure 1. Case 1: (A) First examination. SS-OCT of the right eye showing SRF, PEDs, and multiple hyperreflective solid structures overlying the RPE at the macula. (B–E) One month later. SS-OCT (A) of the right eye showing complete resolution of SRF and PEDs. The hyperreflective solid structures are still present. (C, D) SW-FAF of both eyes. Multiple hyper-autofluorescent lesions at the macula and the posterior pole. (E) OCT-A of the right eye showing multiple hyperreflective irregular shaped lesions at the choriocapillaris layer. PED=pigment epithelium detachment, RPE=retinal pigment epithelium, SRF=subretinal fluid, SS-OCT=swept-source optical coherence tomography, SW-FAF=Snellen visual acuity assessment, funduscopy, autofluorescence.

compensate microsaccades. A 3-dimensional (3D) wide acquisition protocol, a 3D optic nerve acquisition protocol, and a 3D macula acquisition protocol were used. Furthermore, OCT angiograms were acquired at 3×3 and 6×6 mm zones centered on the macula, the optic nerve, and on fundus areas with Elschnig spots.

Blood pressure was recorded using a suitably sized cuff attached to an automated digital sphygmomanometer (UA-767; A&D Company, Ltd, Tokyo, Japan) after the patient had been seated for 5 minutes.

2.1. Case 1

A 46-year-old woman with no known history of hypertension or other systemic or ocular disease presented in the emergency department with sudden visual loss in both eyes. Best corrected Snellen visual acuity was 6/36 in the right eye and 6/48 in the left. Flame-shaped hemorrhages perpendicular to the optic disc margin and punctate yellow Elschnig spots scattered in the posterior pole were seen by funduscopy in both eyes. SS-OCT

revealed bilateral macular serous retinal detachment (subretinal fluid [SRF]), multiple pigment epithelium detachments (PEDs), and multiple hyperreflective solid structures overlying the retinal pigment epithelium (RPE) of both eyes (Fig. 1A). A small amount of intraretinal fluid was present in the right eye. The solid structures overlying the RPE were also noted as whitish, roundish hyperreflective lesions by OCT-A.

As her BP was 230/140 mm Hg, she was immediately referred to the cardiology department of our hospital. She was treated with Trinitroglycerin solution 25 mg IV (diluted in 500 mL dextrose 5% solution, 80 mL/h) and Furosemide injection solution 20 mg IV (1 injection twice daily). Subsequently, the treatment was modified by the administration of Nebivolol hydrochloride tablet 5 mg (one-half tablet daily) and Amlodipine besylate capsule 5 mg (one capsule daily). Her systemic laboratory testing revealed mild anemia and hypocalcaemia, and also serum creatinine at 4 mg/dL, serum urea at 107 mg/dL, and 24-hour urinary proteins of 5.6 g. Based on these data, she started repeated dialysis sessions 8 days later.

We examined the patient 1 month after her first visit in our department. She was being received an oral treatment with

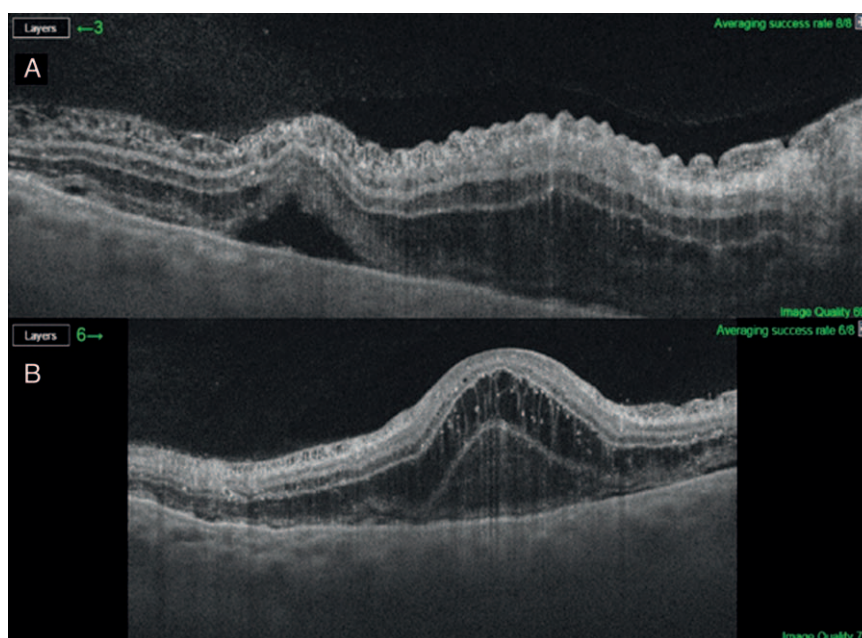


Figure 2. (A, B) Case 2 at first examination. SS-OCT of the right eye showing macular SRF, multiple solid hyperreflective structures overlying the RPE, and wrinkling of the inner retinal layers with splitting of the ganglion cell and nerve fiber layer. RPE=retinal pigment epithelium, SRF=subretinal fluid, SS-OCT=swept-source optical coherence tomography.

Nebivolol hydrochloride tablet 5 mg (one-half tablet daily) and Nifedipine tablet 30mg (1 tablet twice daily). Her BP was ordinarily at 130/80mm Hg. Her best corrected visual acuity restored to 6/6 right and 6/9 left. Complete hemorrhage enhancement was revealed on fundus examination. SS-OCT showed macular SRF and PED absorption, but the hyperreflective solid structures seen at her first visit were still present (Fig. 1B). The lesions were also present by the SW-FAF (Fig. 1C, D) and the OCT-A examinations (Fig. 1E).

2.2. Case 2

A 35-year-old woman with no known history of hypertension or other systemic or ocular disease presented in the emergency department with sudden visual loss in both eyes. Best corrected Snellen visual acuity was 6/48 in the right eye and 6/36 in the left. Fundus examination revealed optic disc edema, flame-shaped hemorrhages, cotton-wool spots, and punctate yellow Elschnig spots scattered in the posterior pole of both eyes. Meticulous examination of the retinal periphery revealed an inferior exudative retinal detachment. SS-OCT showed a macular SRF, wrinkling of the inner retinal layers with splitting of the ganglion cell and nerve fiber layer, and also multiple solid hyperreflective structures overlying the RPE in both eyes (Fig. 2A, B).

The patient's BP was 210/135 mmHg and she was referred to the cardiology department of our hospital, where she was started a treatment with 1 Nifedipine tablet 30mg. Her systemic laboratory testing, including serum creatinine and urea, were within normal limits. The patient left the hospital on her own decision. One month later she presented to our department for examination. She was being treated with Amlodipine besylate capsule 10mg (1 capsule daily) and Amilovide hydrochloride combined with hydrochlorothiazide tablet (Moduretic tablet 5 + 50) (1 tablet daily). Her BP was 120/70 mm Hg. Her best corrected visual acuity had been improved to 6/12 in the right eye and 6/9 in the left. Fundus examination showed complete

hemorrhages and inferior retinal detachment absorption. Elschnig spots and optic nerve edema were still present and a macula star had begun to develop. SS-OCT showed a restoration of the retinal architecture with complete resolution of the macula SRF and a slight SRF perpendicular to the optic disc (Fig. 3A, B). However, the solid hyperreflective structures overlying the RPE seen on her first examination were still present and even larger in size, on SS-OCT (Fig. 3A, B), OCT-A (Fig. 3C), and SW-FAF (Fig. 3D). OCT-A did not seem to reveal any ischemic focal areas in the choriocapillaris layer corresponding to the existing Elschnig spots (Fig. 4A, B).

3. Discussion

Hypertensive chorioretinopathy is a well-known manifestation of malignant (accelerated) hypertension. Flame-shaped hemorrhages, cotton-wool spots, SRFs, PEDs, Elschnig spots, and optic disc edema are known lesions of this condition.^[1-20] All the above lesions were presented in both eyes of our patients at their first visit. OCT images of malignant hypertensive choroidopathy, which visualized SRFs, PEDs, and Elschnig spots, have already been published.^[14-20] To the best of our knowledge, our report is the first one that presents the SS-OCT and OCT-A findings in patients with accelerated hypertensive chorioretinopathy.

In our cases, using swept-source technology, we were able to visualize the existing SRFs and PEDs, the detailed changes in the RPE layer and Bruch membrane, and also to detect multiple solid hyperreflective structures overlying the RPE. The last ones were also detected by en face OCT-A imaging as hyperfluorescent lesions and by SW-FAF imaging as auto-hyperfluorescent areas. Based on all the above characteristics, we can presume that these peculiar lesions are fibrin deposits overlying the RPE.

In the past, solid hyper-reflective structures on the surface of the RPE were also detected by spectral domain OCT in patients with hypertensive choroidopathy due to eclampsia.^[17-19] Further, electron microscopically, Kishi et al^[2] detected fibrinous

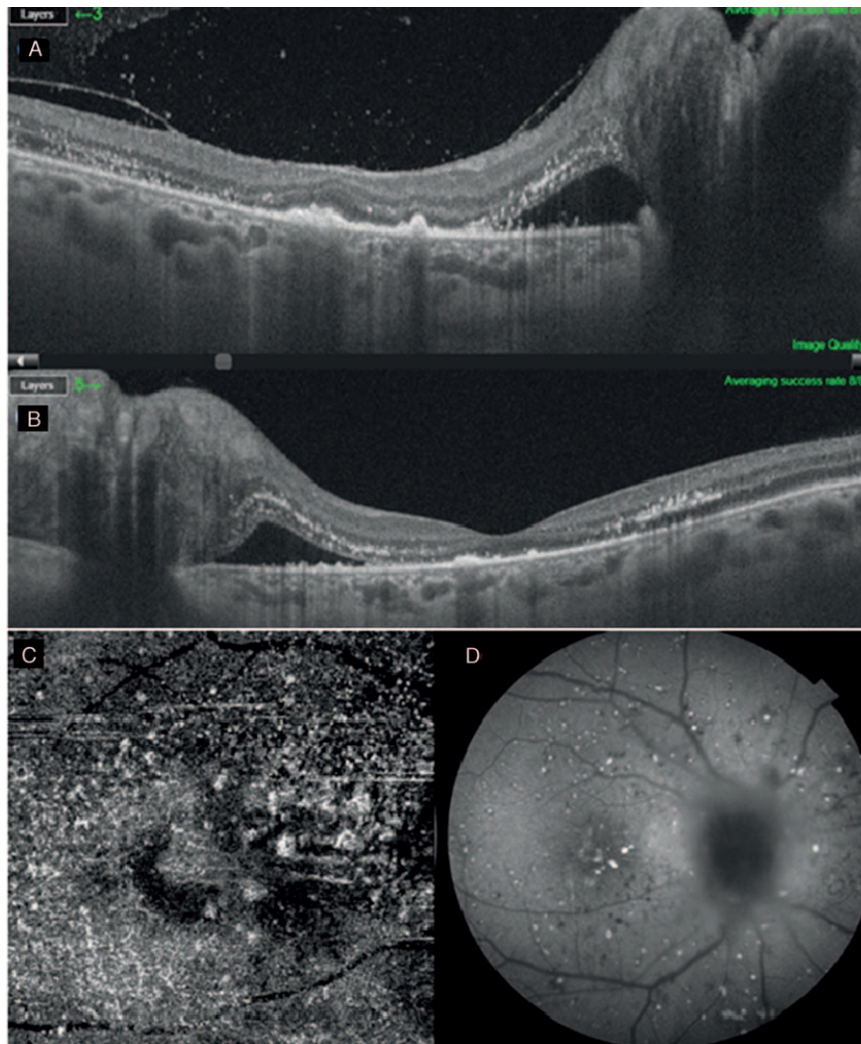


Figure 3. Case 2 one month after first examination. (A, B) SS-OCT of both eyes. Complete resolution of the macula SRF with restoration of the retinal architecture. A slight parapillary SRF still remains and the solid hyperreflective structures overlying the RPE are even larger in size. (C) OCT-A of the right eye showing multiple hyperreflective irregular-shaped lesions at the choriocapillaris layer. (D) SS-FAF of the right eye showing multiple hyper-autofluorescent lesions at the macula and the posterior pole. OCT-A=optical coherence angiography, RPE=retinal pigment epithelium, SRF=subretinal fluid, SS-OCT=swept-source optical coherence tomography.

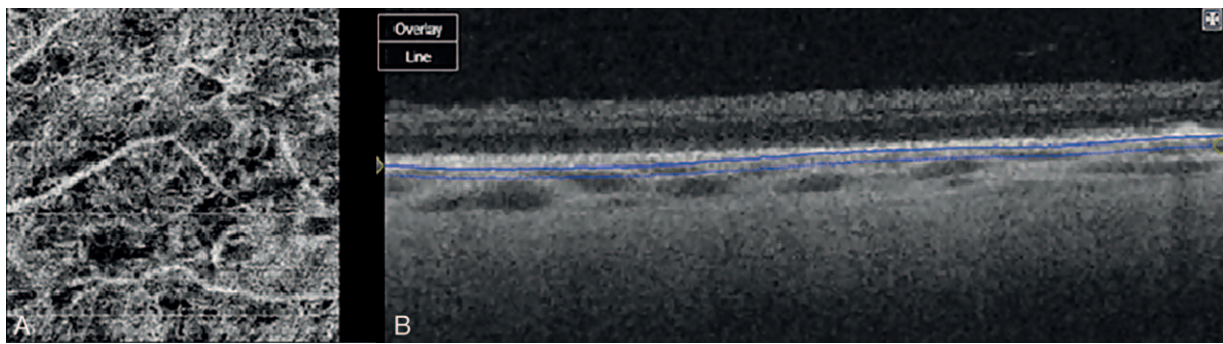


Figure 4. Right eye of case 2 one month after first examination. (A) OCT-A image does not seem to reveal any ischemic areas in the choriocapillaris layer corresponding to the Elschnig spots. (B) SS-OCT at the same area showing the thickness of the optical section of OCT-A. OCT-A=optical coherence angiography, SS-OCT=swept-source optical coherence tomography.

deposits at Bruch membrane in their experimental monkeys with hypertensive choroidopathy during its acute ischemic phase. Finally, in their experimental model of rhesus monkeys with accelerated hypertension, de Venecia et al^[16] histopathologically found out fibrinous exudation into the subretinal space. Our observations by SS-OCT, OCT-A, and SW-FAF in our patients are in agreement with all these previously presented findings and provide support to the contention that the solid structures overlying the RPE represent fibrin deposits.

According to the prevailing scientific view, Elschnig spots are presented during the acute phase of malignant hypertension due to focal necrosis of choroidal arterioles with resultant non-perfusion of the superimposed choriocapillaris that causes ischemic damage to the overlying RPE.^[1,16–17,20] However, in our cases, OCT-A did not reveal any ischemic focal areas at the choroidal circulation corresponding to the existing Elschnig spots. We presume that the resolution of the current OCT-A systems does not allow the imaging of such minute details of the choroidal circulation at the present time.

After antihypertensive treatment a gradual reduction in the size and number of hypertensive chorioretinopathy lesions was observed in both patients. At the end of the first month, almost all the initial lesions had been subsided, apart from the Elschnig spots, the optic disk edema that nevertheless was much better, and the solid fibrin accumulations overlying the RPE which were showed an increasing at their size.

4. Conclusions

In conclusion, correlating with the previous histopathologic or spectral-domain OCT studies, we present here for the first time the findings of malignant hypertensive chorioretinopathy and its short course after BP regulation, using SS-OCT technology and OCT-A. These novel examinations will probably help us to detect in detail the various manifestations of malignant hypertension on the fundus, and also to understand better the dark sides of their pathophysiology.

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