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The choroidal structure changes in a case with hypertensive choroidopathy

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ABSTRACT

Purpose: The aim of this study is to report clinical course and provide novel ophthalmic findings by spectral-domain optical coherence tomography (SD-OCT) in a patient with hypertensive choroidopathy secondary to hypertensive disorders of pregnancy (HDP).

Observations: A 27-year-old woman, who was diagnosed HDP complicated with disseminated intravascular coagulation (DIC), noticed abnormality of color vision and metamorphopsia in her right eye, a half day after an emergency cesarean delivery. Fundus examinations showed developing serous retinal detachment (SRD) from superior hemisphere to the posterior pole in her right eye. Then, fluorescein angiography (FA) showed some granular leakages from the areas above the optic disc in her right eye and around the optic disc in her left eye. Indocyanine green angiography (IA) also showed choroidal hypoperfusion during the early-phase and choroidal hyperpermeability during the mid-phase in the same areas of leakages in FA. SD-OCT also showed posterior SRD in her right eye and peripapillary flat SRD in her left eye, and enhanced depth imaging OCT (EDI-OCT) revealed increased choroidal thickness. SRD gradually disappeared and her symptoms improved during observation with appropriate treatment for hypertension and resolution of DIC. Moreover, increased choroidal thickness in right eye improved in the only areas showing abnormal angiography findings, but subfoveal choroidal thickness in both eyes did not change over the clinical course.

Conclusions: Our case shows that hypertensive choroidopathy with developing SRD and visual disorder in the patient with HDP, can be improved by appropriate treatment for general state. Moreover, SD-OCT shows a new finding that increased choroidal thickness improved in the abnormal angiography areas earlier than SRD was completely disappeared. OCT may be a useful module to evaluate the changes in the choroidal structures for diagnosis and follow-up in a patient with hypertensive choroidopathy.

1. Introduction

Choroid is an overgrown vascular layer of the eye and the most vascular tissue of the human body. It is well known that the choroid circulation provides blood to outer one third of the retina, including photoreceptors and pigment epithelium.[1] In 1990s, development of indocyanine green angiography (IA) gave visualization of the choroidal circulation.[2] Recently, development of spectral-domain optical coherence tomography (SD-OCT) with enhanced depth imaging (EDI-OCT) technique and swept source OCT (SS-OCT) technologies have enable us to evaluate the structure of choroid.[3] With using these technologies, it has been revealed that changes of choroid play important role in pathophysiology of various retinal diseases such as Vogt–Koyanagi–Harada (VKH) disease and pachychoroid spectrum diseases including central serous chorioretinopathy.[4] Choroidal ischemia associated with obstruction of choroidal arterioles and non-perfusion of choriocapillaris could cause necrosis of overlying retinal pigment epithelium and then breakdown of the outer blood-retinal barrier, and might subsequently lead development of serous retinal detachments.[5,6] Therefore, choroidal circulatory insufficiency could cause visual disturbance in patients who had severe hypertension and other systematic abnormality including hypertensive disorders of pregnancy (HDP), renal failure, systemic lupus erythematosus, disseminated intravascular coagulation (DIC), and thrombotic thrombocytopenic purpura, previously reported.[7,8] We report a patient case with HDP who developed serous retinal detachment (SRD) after delivery and had focal choroidal changes in SD-OCT over the clinical course.

2. Case report

A 27-year-old woman at 33 weeks of pregnancy with proteinuria and elevation of blood pressure was diagnosed as HDP, though she had

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no history of hypertension or other diseases before pregnancy. Despite of treatment with intravenous nicardipine and induction of labor, her blood pressure was uncontrolled. Moreover, she developed coagulopathy and her blood test indicated DIC (PLT: 24000/μl, PT-INR: 1.44, fibrinogen: 65 mg/dl, FDP: 15.8μg/ml). She received emergency cesarean section as she had premature separation of normally implanted placenta at 36 weeks of pregnancy. Before delivery, her maximum blood pressure was 172/112 mmHg.

About 12 hours after delivery, she noticed abnormality of color vision and metamorphopsia. At 5 days after the delivery, she visited at the department of ophthalmology in Kobe university hospital. Then, she treated for hypertension by orally amlodipine 5mg.

Her best-corrected visual acuity (BCVA) in decimal points was 1.2 in each eye and intraocular pressures were 13 mmHg in each eye at initial examination. At this moment, the patient denied any symptoms such as meningismus, tinnitus, alopecia, a patch of white hair and vitiligo in her skin except her vision change.

Regarding to anterior segment examination, she had only subconjunctival hemorrhage in her right eye. There were no inflammatory findings in the anterior chamber and posterior segment in both eyes. On the other hand, fundus-ophthalmoscope showed SRD in the posterior pole of her right eye (Fig. 1). and no active lesion of her left eye (Fig. 2).

We performed fluorescein and indocyanine green angiographies (FA and IA) in her both eyes using a Spectralis HRA2 device (Heidelberg Engineering, Heidelberg, Germany) at 8 days after delivery.

Because the patient was in breast feeding, FA and IA were performed after informed consent was obtained adequately, and patients were advised not to breast feed for 1 week after angiography.

FA showed granular leakages from the areas above the optic disc in her right eye and around the optic disc in her left eye during the early to mid-phase. IA showed focal filling delay of the choroid during the early-phase, which subsequently take place of mottled hyperfluorescent lesions during the mid-phase in the same area of leakage in fluorescein angiography (Fig. 1).

SD-OCT images (Heidelberg Engineering, Heidelberg, Germany) also revealed macular SRD in right eye and peripapillary flat SRD in left eye (Figs. 2 and 3).

EDI-OCT showed subfoveal choroidal thickening in both eyes.

Moreover, RPE irregularity and shallow SRD with increased choroidal thickness were shown above the optic disc in her right eye, corresponding to the area of abnormal angiography (Fig. 4).

Finally, we diagnosed her as hypertensive choroidopathy secondary to HDP from these findings.

Since her blood pressure was still high (150/100 mmHg), we ordered intension of antihypertensive therapy to obstetrician. Orally amlodipine 10mg and nifedipine 20mg made her blood pressure low effectively. Her coagulopathy resolved spontaneously after delivery.

SRD in right eye gradually disappeared during observation over 8 weeks with appropriate treatment for hypertension and resolution of DIC. Peripapillary flat SRD in left eye was also improved. At 8 weeks after first visit, the patient stated that the abnormality of color vision in her both eyes was completely improved and the metamorphosis in her right eye was also almost recovered, who eventually kept her BCVA in decimal points at 1.2 in the right eye and 1.0 in the left eye. Moreover, increased choroidal thickness decreased at the same time as RPE irregularity with RPE elevation and SRD improved in the areas showing abnormal angiography findings in right eye, but subfoveal choroidal thickness in both eyes did not change in clinical course (Figs. 3 and 4).

3. Discussion

Our case report provides the details imaging of hypertensive choroidopathy with a 27-year-old woman who developed SRD in her both eyes after delivery.

The differential diagnosis is important in diagnosis of hypertensive choroidopathy. In particular, central serous chorioretinopathy (CSC) and VKH disease should be ruled out, because the management is completely different between hypertensive choroidopathy and them. The treatment of VKH is high-dose systemic corticosteroids and those of CSC are photocoagulation or careful observation, but main treatment of hypertensive choroidopathy is blood pressure management.

VKH has characteristic findings including multiple leak points in FA and dark dots in IA and CSC has those including hotspots on FA and choroidal hyperpermeability in IA retinal pigment epithelium folds as a diagnostic finding of VKH disease.[9–11]

Eventually, since the patient had no evidence of inflammation in her eyes and no general symptom associated with VKH disease, VKH disease was denied in accordance with the diagnostic criteria.[12]

The patient did not have neither any findings which can indicate CSC or VKH except SRD and choroidal thickening in her examination nor any ocular inflammation signs, thus we diagnosed her with hypertensive choroidopathy due to HDP.

Regarding to anatomical characteristics in ocular circulation, there are the differences of end-artery vessel characteristics between retinal circulation and choroidal circulation. While retinal circulation characterized capillary vessel by the presence of inner retinal-blood barrier,

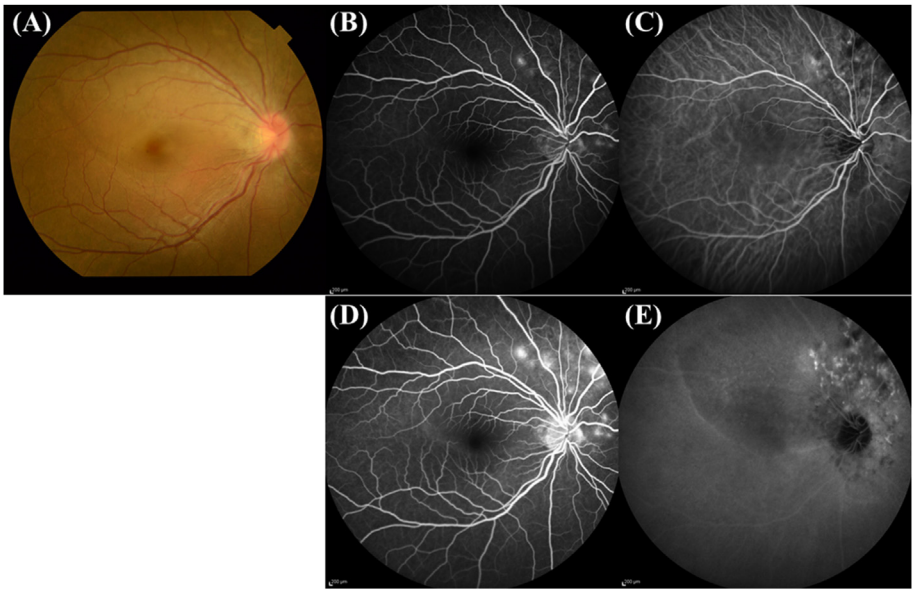

Fig. 1. Color fundus image and angiography findings in the right eye.
Fundus image shows serous retinal detachment in the posterior pole. (A) Fluorescein angiography shows multiple hyperfluorescent lesions as granular leakage points above the optic disc during the early to mid-phase. (B, C) Indocyanine green angiography shows focal filling delay during the early-phase, (D) which subsequently take place of mottled hyperfluorescent lesions during the mid-phase. (E). (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

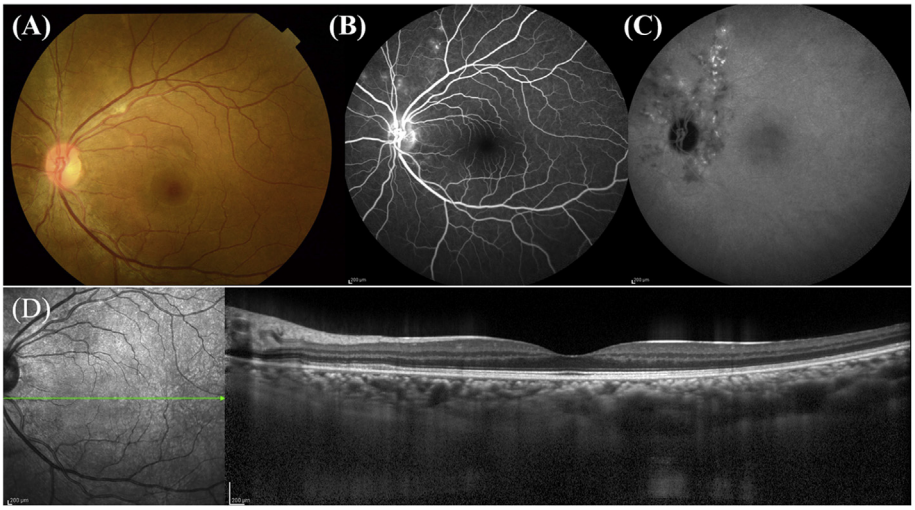

Fig. 2. The multimodal imaging findings in the left eye.
Fundus image shows no apparent evidence of active lesion. (A) Angiography shows similar findings to the right eye. In the mid-phase, fluorescein angiography shows some hyper fluorescent points superior to the optic disc along with arcade vessel, and indocyanine green angiography shows mottled hyperfluorescent lesions around optic disc over abnormal findings in fluorescein angiography. (B, C) Macula optical coherence tomography scan through the fovea shows peripapillary flat SRD and thicken subfoveal choroid at 501μm. (D). (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

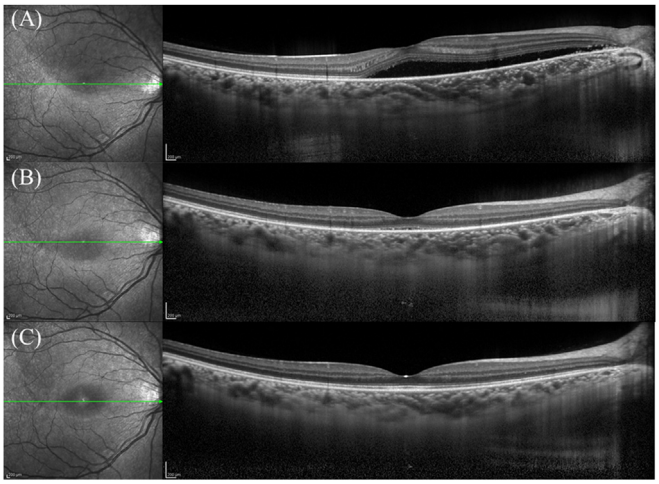

Fig. 3. Macula optical coherence tomography (OCT) scan through the fovea in the right eye.
At the initial visit, OCT image shows SRD including macula and increased choroidal thickness. (A) At 3 weeks later, OCT image shows slight residual SRD. The choroidal thickness did not change. (B) At 8 weeks later, OCT image shows completely disappearance of the SRD. The choroidal thickness is still increasing. (C) Subfoveal choridal thickness were 444μm, 421μm and 431μm at the initial visit, at 3 weeks later and at 8 weeks later, respectively (A, B, C).

choroidal circulation consists of capillary vessel floor arisen from lobular-shaped domain as choriocapillaris.[1] Choroidal arterial tracks divide into multiple branches and then reaches a vast bed of choriocapillaris. Then, the flow decelerates into choriocapillaris, and thereby the thrombus formation can lead to occur, especially in hypercoagulation condition.[13]

Patients with HDP could have retinochoroidal lesions, including arteriolar constriction, retinal hemorrhages, cotton wool spots, retinal edema, lipid exudates, and SRD due to the disturbance of retinal and choroidal circulation, previously reported.[14]

The mechanisms of HDP-induced retinochoroidal lesions could be classified into three types: retinal circulation disturbance type (retinal type), choroidal circulation disturbance type (choroidal type) which is characterized by SRD, and combined type. It was hypothesized that the presence of coagulopathy in patients with lower fibrinogen, decreasing platelet and longer prothrombin time (PT) could cause the choroidal type disorder.[15,16]

Therefore, in this study, it was possible that HDP with abnormality in general coagulate state such as DIC cause choroidal circulation

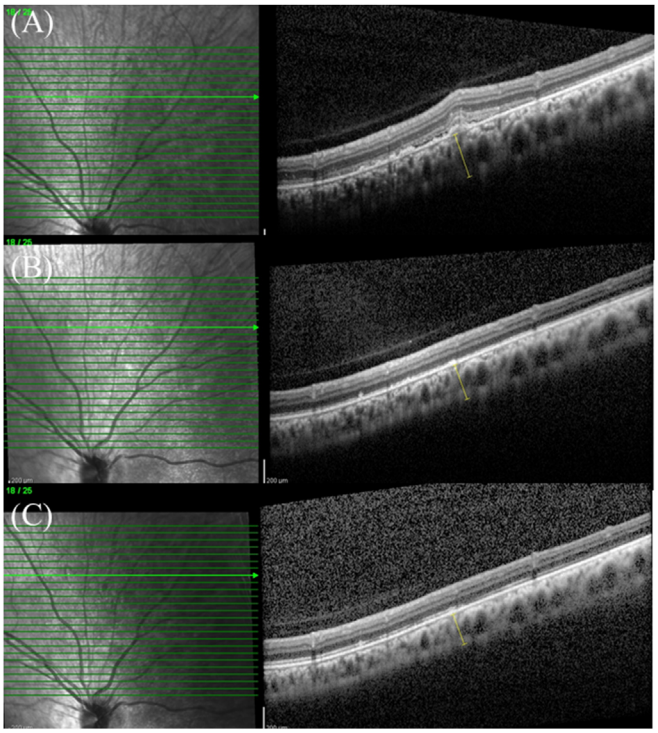

Fig. 4. Above optic disc optical coherence tomography (OCT) scan through the hyper fluorescent lesions in fluorescein angiography in right eye
RPE elevation and irregularity with increased choroidal thickness(A) gradually improved at 3 weeks and 8 weeks after initial visit (B, C). RPE deposit and partial EZ disruption were detected at 3 weeks.(B) Integrity of RPE and EZ almost improved at 8 weeks after initial visit (C). Choroidal thickness showed by yellow bar at the fixed point were 472μm, 394μm and 345μm at the initial visit, at 3 weeks later and at 8 weeks later, respectively (A, B, C). (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.).

disturbance due to occlusion of choriocapillaris,[13] and then hypertensive choroidopathy were occurred in her both eyes.

In this case, we detected abnormality of choroidal circulation and morphology in hypertensive choroidopathy by using FA/IA and EDI-OCT. The findings of focal filling delay during the early-phase in IA might indicate choroidal ischemia, mottled hyperfluorescence lesions during the mid-phase might do choroidal hyperpermeability and some granular leakage points in FA might do a defect in the blood-retinal barrier.

These findings meet three conditions of Marmor's hypothesis to form serous detachment: (1) a source of fluid pressure, (2) a defect in the blood-retinal barrier (entry site), and (3) disorder of fluid transport area around leakage points in FA.[17]

In this case, SD-OCT showed increased subfoveal choroidal thickness in both eyes and dynamic change of choroidal thickness only in the areas corresponding to the abnormal angiography areas in FA/IA as general state was improved.

Moreover, increased choroidal thickness in the abnormal angiography areas improved earlier than SRD completely disappeared in OCT.

In this aspect of choroidal thickness, Yeung SC et al.[18] mentioned a hypothesis about the mechanism of choroidal thickness change over clinical course. They also reported that choroidal ischemia occurs when choroidal vessel autoregulation cannot withstand acute hypertension and causes choroidal capillary fibrinoid necrosis and retinal pigment epithelium necrosis. Subsequently, it is believed as one of the possibilities that the result of altered choroidal permeability can cause interstitial fluid accumulation, and thus increased choroidal thickness. Actually, choroidal vessel layer, especially Haller's layer, seemed dilated in this case, but the involvement of choroidal vessel layer in hypertensive choroidopathy will be considerable future task.

RPE irregularity with RPE elevation shown in Fig. 4 would be a characteristic finding in this case. The similar abnormal OCT findings of RPE elevation was reported in other cases of hypertensive choroidopathy.[19,20] These findings were improved after treatment. It was mentioned that focal occlusion of choriocapillaris leads to necrosis of RPE in hypertensive choroidopathy.[7] This OCT finding of RPE elevation might show secondary damage of RPE due to focal occlusion of choriocapillaris.

Moreover, Rezkallah A et al. reported temporally multifocal choriocapillaris non-perfusion in acute phase of hypertensive choroidopathy and retinopathy using multimodal imaging with wide-field SS-OCT angiography, who concluded that choriocapillaris non-perfusion altered the RPE and could weaken its pump function which caused retinal serous detachment.[20] Though their case had very similar aspect of pathological background to this case, the apparent difference between both cases is shown in the presence of focal choroidal change without subfoveal choroidal change, which indicate focal choroidal circulation disturbance in this case.

Therefore, this case would give new evidences that the focal disturbance of choroidal circulation could occur in hypertensive choroidopathy and affect on focal choroidal thickness, and choroidal circulatory insufficiency could improve prior to improvement of RPE function to absorb SRD.

4. Conclusion

In conclusion, our case shows that hypertensive choroidopathy with developing SRD and visual disorder in the patient with HDP, can be improved by appropriate treatment for general state. Moreover, SD-OCT shows a novel finding that increased choroidal thickness can improve in abnormal angiography areas earlier than SRD completely disappeared. OCT may be a useful module to evaluate the choroidal structures for diagnosis and follow-up in patients with hypertensive choroidopathy.

Patient consent

Consent to publish the case report was obtained. Moreover, this report does not contain any personal information that could lead to the identification of the patient.

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Authorship

All authors attest that they meet the current ICMJE criteria for authorship.

Declaration of competing interest

None of the authors have financial disclosures or conflicts of interest relating to this topic.

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