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Case report

Bilateral serous retinal detachment associated with subretinal fibrin-like material in a case of pregnancy-induced hypertension



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ARTICLE INFO	A B S T R A C T
Keywords: Angiography Choroidal hypoperfusion Pregnancy-induced hypertension Serous retinal detachment Subretinal fibrin-like material	<i>Purpose</i> : To describe a case of bilateral serous retinal detachment (SRD) associated with subretinal fibrin-like material (SRFM) in pregnancy-induced hypertension (PIH). <i>Observations</i> : Angiography of a 31-year-old primigravida with PIH who developed acute bilateral SRD with SRFM after caesarean section showed choroidal hypoperfusion and dye leakage. Optical coherence tomography revealed irregularity of the ellipsoid zone and retinal pigment epithelium. The patient's visual acuity was hand motion in the both eyes at the initial examination. After 30 days, SRD and SRFM spontaneously disappeared Moreover, the EZ returned and the visual acuity significantly improved to 20/25 in the right and 20/20 in the left eye. <i>Conclusions and importance</i> : Although there are reports on central serous chorioretinopathy or Vogt–Koyanagi–Harada disease, there have been no studies on SRFM complications in patients with PIH. In the current case, we speculated that SRFM was associated with acute intense choroidal ischemia and inflammation secondary to this ischemic condition. Distinguishing whether inflammation necessitating treatment is involved in SRFM may be difficult. Short observation intervals and frequent examinations are important to ensure that treatment timings are not missed.

1. Introduction

Pregnancy-induced hypertension (PIH) is classified as a hypertensive disorder that occurs between 20 weeks of the gestation period and 12 weeks after delivery. PIH is characterized by elevated blood pressure (\geq 140 mmHg systolic or 90 mmHg diastolic on at least two occasions that are 6 hours apart), is accompanied by proteinuria (\geq 300 mg/24 hours), and has a prevalence of 3–10% in all pregnancies.¹ Previous reports have shown that bilateral serous retinal detachment (SRD) is one of the causes of visual loss in PIH, with a prevalence varying from 0.1% to 32.4%.¹ However, there have been no studies that have investigated the association of bilateral SRD with subretinal fibrin-like material (SRFM). In our current study, we report on a case of PIH associated with both SRD and SRFM complications.

2. Case report

A 31-year-old Japanese primigravida female was hospitalized at 37 weeks of pregnancy because of PIH. Up to this point, she had no medical history, including a history of hypertension. At the time of hospitalization, her blood pressure was 141/88 and 138/105 mmHg at

two different measurement points, in addition to having proteinuria of 2+. Since delivery did not occur after an attempt to induce labor, an emergency caesarean section was performed. The total amount of bleeding at the end of the operation was approximately 2200 mL. The patient complained of a visual disorder that occurred immediately after the operation. However, there were no abnormal findings on brain imaging (computed tomography and magnetic resonance imaging). On the next day, she was referred to our emergency outpatient clinic and underwent ophthalmologic evaluations by the doctor on call. Examinations revealed bilateral retinal detachment with macular involvement, and visual acuity was hand motion in both eyes. Since there were no retinal tears or holes, she was scheduled for an angiographic examination 2 days after the first examination, due to an intervening weekend and because of her clinical condition.

Three days after onset, her visual acuity had spontaneously improved to 20/50 in the right and 20/33 in the left eye. Anterior segment examinations were normal. Fundus examination showed bilateral SRD with macular involvement and SRFM, with no observed abnormalities of the retinal vessel (Fig. 1A and B). Optical coherence tomography (OCT) demonstrated bilateral subretinal fluid (SRF), SRFM, and a thickened choroid at the macula (choroidal thickness of 427 µm in the

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Fig. 1. Fundus photographs and optical coherence tomography (OCT) scans at 3 days after onset. (A, B) Fundus photographs exhibiting bilateral serous retinal detachment (SRD) and subretinal fibrin-like material (SRFM) (white arrowhead). (C, D) OCT scans showing bilateral SRD, thickened choroid, and SRFM (asterisk).



Fig. 2. Fluorescein angiography at 3 days after the onset. (A, B) Early phase images showing delayed choroidal perfusion. (C, D) In the same area, leakage foci are observed in the late phase.

right and 423 µm in the left eye) (Fig. 1C and D). Fluorescein angiography (FA) and indocyanine green angiography (IA) were performed on the same day. FA showed patchy delayed choroidal perfusion in the early phase and foci of fluorescein leakage from the same area in the late phase (Fig. 2A–D). There was no abnormality in retinal circulation (starting at 15 s). IA showed choroidal hypoperfusion from the early to the late phase and late spot staining in the dark area (Fig. 3A–D). Her blood pressure was 122/87 mmHg at the time of angiography, and it did not meet hypertension criteria thereafter. Breastfeeding was postponed until 4 days after the FA and IA.

At 10 days after the onset, SRF, SRFM, and the thickened choroid improved without any treatment (choroidal thickness of 316 μ m in the right and 331 μ m in the left eye) (Fig. 4A and B). However, OCT revealed irregularity of the ellipsoid zone (EZ) and the retinal pigment epithelium (RPE) layer (Fig. 4C and D). There were no changes in her visual acuity.

At 30 days after the onset, SRF, thickened choroid, and SRFM spontaneously disappeared. Moreover, EZ returned and the patient's



Fig. 3. Indocyanine green angiography at 3 days after the onset. (A, B) Early phase images showing delayed choroidal perfusion. (C, D) In the late phase, there is punctuate staining in the dark area.

visual acuity improved to 20/25 in the right and 20/20 in the left eye, although a slightly irregular RPE layer remained (Fig. 4E and F).

3. Discussion

The current report presents a case of PIH with SRD and SRFM complications. Although the exact pathophysiology for PIH has not been completely elucidated, it has been demonstrated that PIH is associated with increased systemic vascular resistance and a generalized vasospasm that occurs secondary to abnormal placentation. Since the factors reflecting this condition are thought to cause retinal and choroidal circulation dysfunction, angiography evaluations have been



Fig. 4. Fundus photographs and optical coherence tomography (OCT) scans in the follow-up period. (A, B) Bilateral serous retinal detachment (SRD) and subretinal fibrin-like material (SRFM) had decreased significantly at 10 days after the onset. (C, D) OCT scans show decreased SRD and reduced choroidal thickness and SRFM at 10 days after the onset. However, irregularity of the ellipsoid zone (EZ) and retinal pigment epithelium (RPE) layer persisted (white arrow). (E, F) In OCT scans obtained 30 days after onset, SRF, thickened choroid, and SRFM had disappeared without treatment. While the irregularity of the EZ had reverted, that of the RPE layer persisted (white arrow).

reported as below. FA results have shown that there is delayed choroidal perfusion and dye leakage in the sub-RPE and subretinal spaces in the late phase. IA results have shown that there is early choroidal nonperfusion and late staining of choroidal vessel walls.¹ These findings were similar to those of the current case, which suggests that dysfunction of the choroidal circulation was primarily involved in the current pathology.

In severe cases, it has been reported that ocular findings associated with hypertensive retinopathy may occur, including retinal hemorrhages, exudates, and cotton wool spots.^{1,2} Our case did not demonstrate any of these findings or dysfunction of retinal circulation; our case had transient hypertension only in the perinatal period and the degree of hypertension was mild. Schobel et al. reported that PIH reflects a state of sympathetic overactivity, which reverts to normal after delivery.3 In addition, Zhao et al. demonstrated that the calcium-activated K channel KCa2.3, which has previously been implicated in aldosterone/mineralocorticoid receptor-induced choroidal vasodilation, was primarily expressed in the endothelium of choroidal vessels, but not in those of the inner retina, while excessive occupancy of choroidal mineralocorticoid receptors by glucocorticoids induced choroidal vessel enlargement in a rodent study.⁴ Consequently, only dysfunction of the choroidal circulation, without disorder of the retinal circulation, may be involved in the early stage of mild PIH. The current case also demonstrated choroidal thickening, which improved after delivery.

SRD, which occurs due to a breakdown of the RPE layer secondary to choroidal ischemia, is known to be causally associated with visual loss in PIH.⁵ The current case had bilateral SRD with SRFM, without any abnormalities of the retinal vessels. In addition, these findings and visual disturbance spontaneously improved during the 4 weeks after the delivery. In previous cases, Saito et al. reported that most of the SRD was resolved within 3 weeks, with 72% actually resolved within 1 week; no patients had permanent loss of vision.⁶ This may have led to good visual recovery during the early and mild stages of PIH, without retinal circulation dysfunction and reversal outer layer disorders because of an acute clinical course.

SRFM has been observed with central serous chorioretinopathy

(CSC) and posterior uveitis, such as in Vogt-Koyanagi-Harada disease (VKH).⁷ In CSC, it is possible that intense choroidal hyperpermeability and breaches in the RPE could allow a large molecule, such as fibrin, to enter from the sub-RPE into the subretinal spaces.⁸ Bindu et al. reported finding some CSC cases that had SRFM overlying the RPE defects on OCT and which corresponded to the area of leakage in FA.⁸ On the other hand, in VKH, these areas could represent inflammatory debris or macrophages engulfing shed outer segments. In fact, Xi et al. reported that these areas were seen at a high frequency during evaluations.⁷ In our current case, although the angiography showed the presence of choroidal hypoperfusion, there were no RPE defects found on OCT evaluations or any findings of inflammation during the slit lamp examination, such as the presence of both anterior chamber and anterior vitreous cells, flares, or vitreous opacifications. In addition, the SRFM disappeared without treatment shortly after delivery. We speculated that the increased blood viscosity related to pregnancy, the decreased plasma volume due to cesarean delivery, and choroidal vasoconstriction in PIH were all involved in causing the intense choroidal hypoperfusion that led to RPE damages and choroidal hyperpermeability. Although we were not aware of any involvement of inflammation in our current case, inflammation has been reported to affect the formation and the progression of retinal vein occlusion.9 Even though there was little evidence, is it possible that SRFM in our case was associated with acute intense choroidal ischemia and some type of inflammation secondary to ischemic conditions.

The management of this condition fundamentally involves treatment of the underlying disease. However, unlike in the current case, if the condition does not improve spontaneously, it is necessary to review the possibility of other pathophysiologies and to consider additional examinations and interventions.

4. Conclusions

We here reported on a case of PIH with SRFM that might have been associated with choroidal ischemia and inflammation caused by ischemic conditions. Even when there is severe SRFM, observation may be first choice for treatment, as in the current case. However, it may be difficult to distinguish whether the inflammation requires treatment based on observations only. Therefore, we concluded that short intervals and frequent examinations are important to ensure that treatment timings are not missed.

To the best of our knowledge, there have been no reports about the association of SRD with SRFM and PIH.

Patient consent

Consent to publish the case report was not obtained. 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

The following authors have no financial disclosures: SK, KM, NH,

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References

- Schultz KL, Birnbaum AD, Goldstein DA. Ocular disease in pregnancy. Curr Opin Ophthalmol. 2005;16:308–314.
- Neudorfer M, Spierer O, Goder M, et al. The prevalence of retinal and optical coherence tomography findings in preeclamptic women. *Retina.* 2014;34:1376–1383.
 Schobel HP, Fischer T, Heuszer K, Geiger H, Schmieder RE. Preeclampsia – a state of
- sympathetic overactivity. *N Engl J Med*. 1996;335:1480–1485.
 Seki H. The role of the renin-angiotensin system in the pathogenesis of preeclampsia -
- new insights into the renin-angiotensin system in the pathogenesis of precedampsia new insights into the renin-angiotensin system in preeclampsia. *Med Hypotheses*. 2014;82:362–367.
- Vigil-De Gracia P, Ortega-Paz L. Retinal detachment in association with pre-eclampsia, eclampsia, and HELLP syndrome. Int J Gynaecol Obstet. 2011;114:223–225.
- Saito Y, Tano Y. Retinal pigment epithelial lesions associated with choroidal ischemia in preeclampsia. *Retina*. 1998;18:103–108.
- Liu XY, Peng XY, Wang S, et al. Features of optical coherence tomography for the diagnosis of vogt-koyanagi-harada disease. *Retina*. 2016;36:2116–2123.
- Rajesh B, Kaur A, Giridhar A, Gopalakrishnan M. Vacuole" sign adjacent to retinal pigment epithelial defects on spectral domain optical coherence tomography in central serous chorioretinopathy associated with subretinal fibrin. *Retina*. 2017;37:316–324.
- Shahid H, Hossain P, Amoaku WM. The management of retinal vein occlusion: is interventional ophthalmology the way forward? *Br J Ophthalmol.* 2006;90:627–639.