

Serous Retinal Detachment in Patients Under Systemic Corticosteroid Treatment

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Purpose: To understand the pathophysiology of central serous chorioretinopathy or bullous retinal detachment in patients under systemic corticosteroid treatment. Little is understood about the mechanism of the development of serous retinal detachment.

Methods: Three patients who had developed central serous chorioretinopathy or bullous retinal detachment during systemic corticosteroid administration were examined by fluorescein angiography and indocyanine green angiography.

Results: Indocyanine green angiography revealed dilated choroidal veins, delayed choroidal filling, intrachoroidal hyperfluorescence, and patchy hypofluorescence at or near the sites of dye leakage examined by fluorescein angiography.

Conclusions: The primary change caused by central serous chorioretinopathy is thought to occur in the choroid, followed by the breakdown of the outer blood-retinal barrier in the pigment epithelium, resulting in the development of serous retinal detachment. **Jpn J Ophthalmol** 2001;45:640–647 © 2001 Japanese Ophthalmological Society

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Introduction

It is known that central serous chorioretinopathy (CSC) sometimes occurs in patients under systemic corticosteroid treatment.^{1–3} A patient in whom CSC developed or worsened following an increase in the amount of corticosteroid, and ameliorated following its decrease was reported.¹ However, little is understood about the mechanism of the development of serous retinal detachment in these patients. On the other hand, the pathogenesis of idiopathic CSC or bullous retinal detachment without systemic corticosteroid treatment has been elucidated with the advent of indocyanine green angiography (IA).^{4–6} Iida et al^{4,5} reported that IA demonstrated intrachoroidal hyperfluorescence (abnormal choroidal tissue staining), delayed choroidal filling, and dilatation of chor-

oidal vessels in patients with idiopathic CSC or bullous retinal detachment. Furthermore, they described that hyperpermeability of the choroidal vessels played an important role in the development of serous retinal detachment.

However, to our knowledge, there has been only one report describing IA findings of CSC in patients under systemic corticosteroid treatment.⁷ We performed fluorescein angiography (FA) and IA on 3 patients with CSC or bullous retinal detachment under systemic corticosteroid treatment.

Case Reports

Case 1

A 34-year-old man had been administered prednisolone at a dosage of 15 mg daily because of systemic lupus erythematosus since 1992. He was admitted to the 2nd Department of Internal Medicine in Kochi Medical School in August 1993. Because his condition had worsened, prednisolone dosage was increased to 60 mg daily, and he also underwent ste-

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roid-pulsed therapy (methyl prednisolone, 1000 mg, for 3 days). He noticed blurred vision in both eyes 1 day after the therapy ended, and was referred to our clinic (Department of Ophthalmology, Kochi Medical School). Fundus examination revealed serous retinal detachment in both eyes, which resolved following retinal photocoagulation. In July 1997, he again noted blurred vision in his left eye. His best-corrected visual acuity was 1.5 in the right eye, and 0.5 in the left. Intraocular pressure was 17 mm Hg in the right eye, and 16 mm Hg in the left, as measured by applanation tonometer. Fundus examination of the left eye demonstrated serous retinal detachment of 2-disc diameters including the macular region. In the right fundus, there was a photocoagulation scar temporal to the macula.

FA examination of the left eye demonstrated two points of dye leakage (Figure 1A) On IA, a scanning laser ophthalmoscope (Rodentstock, Germany) was used. At the sites of eye leakage seen by FA, dilatation of underlying choroidal veins was confirmed by IA (Figure 1B). On the late-phase IA, dye leakage into subretinal space and patchy hypofluorescence around the leakage site were seen. FA examination of the right eye demonstrated a leakage point superotemporal to the macula. Around the site of the eye leakage seen by FA and at the photocoagulation scar, underlying choroidal vein dilatation was confirmed by IA. On the late-phase IA, dye leakage into subretinal space was seen. Krypton laser photocoagulation was performed on the sites of eye leakage seen by FA in the left eye, which resulted in resolution of the serous retinal detachment. The best-corrected visual acuity in the left eye recovered to 1.0.

Case 2

A 44-year-old man with idiopathic thrombocytopenic purpura noticed reduced visual acuity in his left eye, and was referred to our clinic (Department of Ophthalmology, Kochi Medical School) on January 31, 1995. He had been given oral prednisolone at a dosage of 10–15 mg daily for 16 years. His best-corrected visual acuity was 1.2 in the right eye, and 0.7 in the left. Intraocular pressure was 16 mm Hg in the right eye, and 17 mm Hg in the left, as measured by applanation tonometer. The anterior segment, lens, and vitreous were normal in both eyes. Fundus examination of the left eye demonstrated serous retinal detachment of 1.5-disc diameter including the macula. FA examination of the left eye revealed a dye leakage point superior to the macula (Figure 2A). On early-phase IA, there was delayed choroidal filling in the posterior pole (Figure 2B) and partial dilata-

tion of choroidal veins at the dye leakage sites seen by FA (Figure 2C). On late-phase IA, patchy hypofluorescence was seen in the posterior pole. Fundus examination of the right eye disclosed serous retinal detachment temporal to the macula. On FA, there were two dye leakage points temporal to the macula. Indocyanine green angiographic examination disclosed partial dilatation of choroidal veins underlying the dye leakage sites seen by FA. Krypton laser photocoagulation was performed on the site of dye leakage seen by FA in the left eye, and resulted in resolution of serous retinal detachment. Best-corrected visual acuity in the left eye improved to 1.0

Case 3

A 49-year-old woman with autoimmune hepatitis and diminished visual acuity in her left eye was examined on January 14, 1997. She had been given oral prednisolone at a dosage of 15 mg daily for 3 months. She had well-controlled hypertension. Her best-corrected visual acuity was 1.2 in the right eye, and 0.1 in the left. Intraocular pressure was 17 mm Hg in the right eye, and 18 mm Hg in the left. No abnormality was found in the anterior segment, lens, or vitreous in either eye. Fundus examination of the left eye demonstrated bullous retinal detachment inferiorly and a retinal pigment epithelial tear temporal to the macula. Fluorescein angiographic examination of the left eye revealed a marked eye leakage through a retinal pigment epithelial tear (Figure 3A). There was a nonperfusion area in the inferior equatorial area of the fundus. On IA, choroidal veins in the posterior pole were dilated, and in the late phase intrachoroidal hyperfluorescence was seen (Figures 3B, C). Fundus examination of the right eye demonstrated serous retinal detachment inferior to the macula. On FA, there was a dye leakage point inferior to the macula, and patchy hypofluorescence mixed with granular hyperfluorescence was seen temporal to the macula. Indocyanine green angiographic examination of the right eye disclosed intrachoroidal hyperfluorescence in the posterior pole. Krypton laser photocoagulation was performed around the retinal pigment epithelial tear in the left eye and on the site of dye leakage seen by FA in the right eye. Complete resolution of the bullous retinal detachment in the left eye was achieved 3 months later. Her best-corrected visual acuity in the left eye improved to 0.5.

Discussion

In these three cases, indocyanine green angiographic findings involving dilatation of choroidal

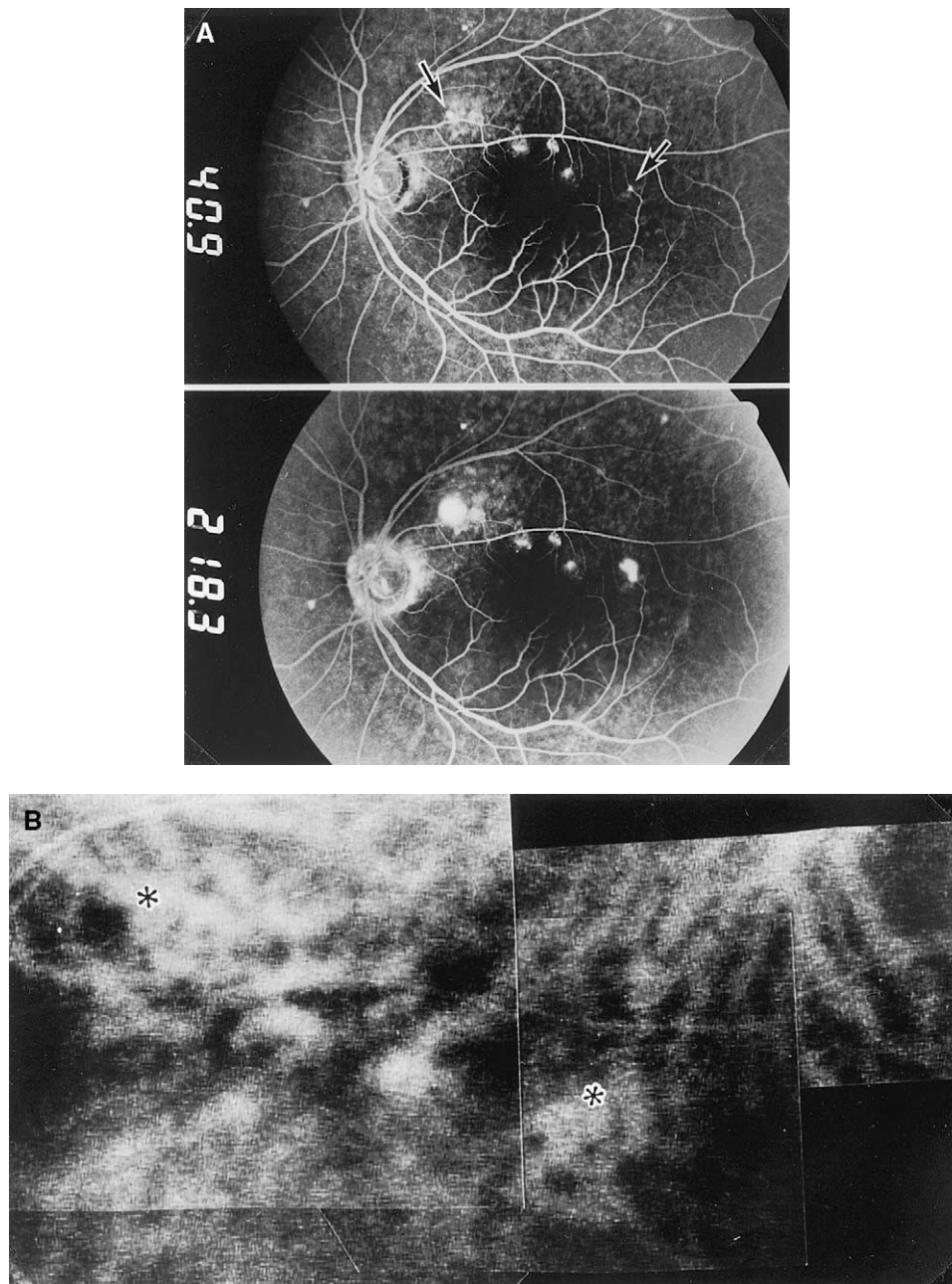


Figure 1. Case 1: 34-year-old man with serous retinal detachment. (A) Fluorescein angiography of left eye showing two leakage points (arrows). (B) Indocyanine green angiography showing dilatation of choroidal vein underlying dye leakage sites shown by fluorescein angiography (asterisks) (48 seconds and 58 seconds after dye injection).

veins, delayed choroidal filling, patchy hypofluorescence, and intrachoroidal hyperfluorescence were obtained at or near the sites of dye leakage seen by FA. These indocyanine green angiographic findings are similar to those seen in idiopathic CSC or bullous retinal detachment without systemic corticosteroid treatment except that in our cases there

was bilateral involvement. Iida et al^{4,5} reported that IA findings involving intrachoroidal hyperfluorescence (abnormal choroidal tissue staining), delayed choroidal filling, and dilatation of the choroidal vein were characteristic in patients with idiopathic CSC or bullous retinal detachment without systemic corticosteroid treatment. Among these findings, they also

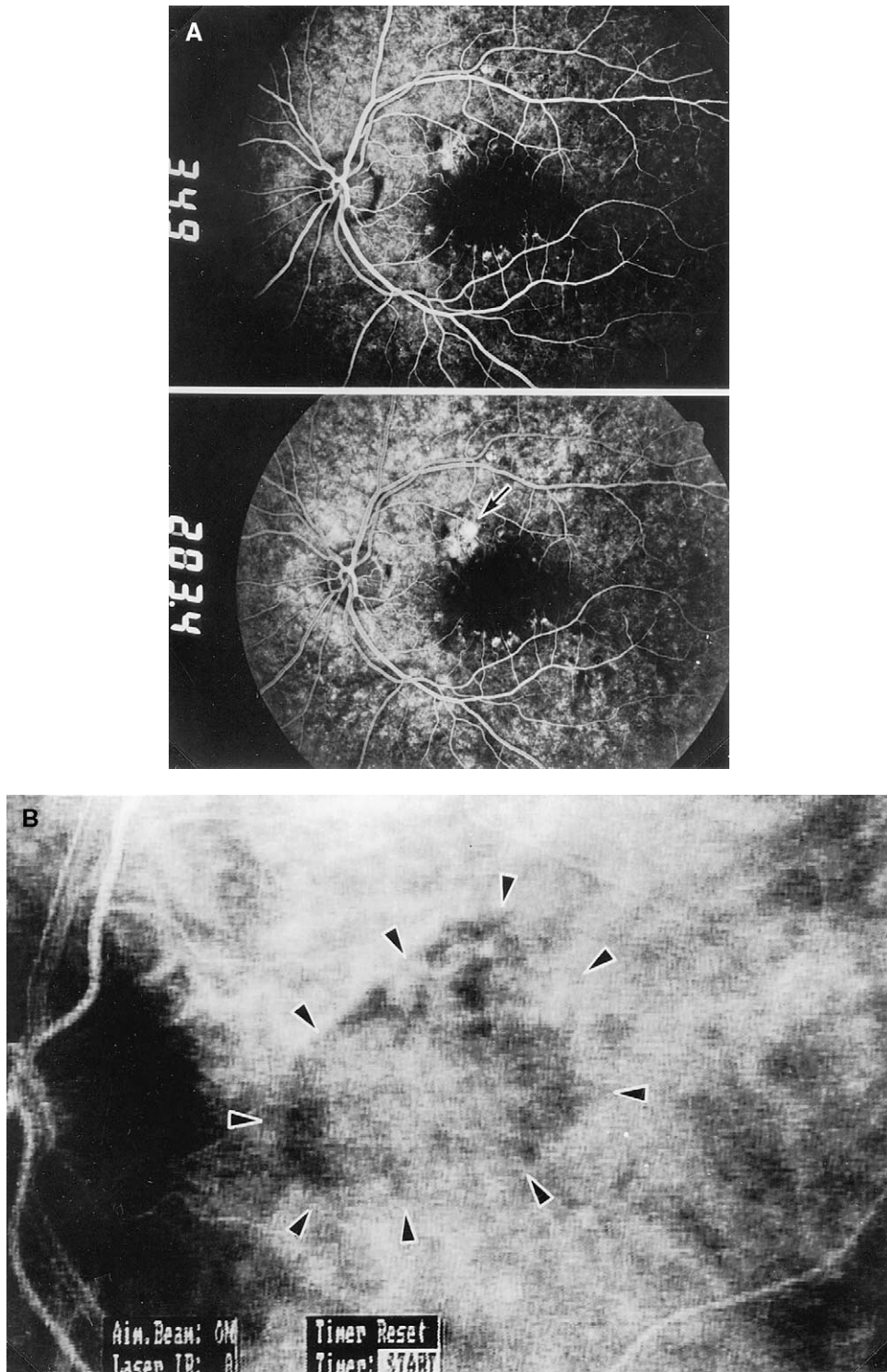


Figure 2. Case 2: 44-year-old man with serous retinal detachment. (A) Fluorescein angiography of left eye showing dye leakage point (arrow) superior to macula. (B) Early-phase indocyanine green angiography of left eye showing delayed choroidal filling (arrowheads) in posterior pole. (C) Indocyanine green angiography of left eye showing partial dilatation of choroidal vein underlying dye leakage site seen by fluorescein angiography (asterisk).

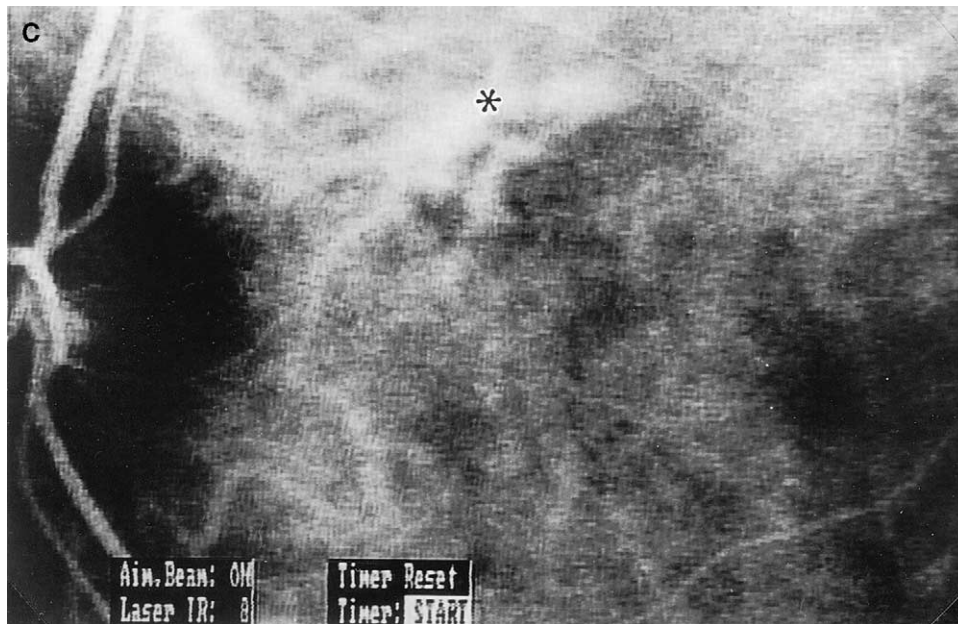


Figure 2. Continued

found intrachoroidal hyperfluorescence to be most frequently seen. However, our cases with systemic corticosteroid treatment presented dilatation of the choroidal vein on IA in all cases (6 eyes), and only one case (2 eyes) presented intrachoroidal hyperfluorescence. There is some difference between our cases and the cases reported by Iida et al regarding the choroidal features shown by IA. Machida et al⁷ performed IA on 5 patients (7 eyes) with CSC under systemic corticosteroid treatment, and described that the finding of intrachoroidal hyperfluorescence was seen in 7 of 8 eyes and that choroidal vein dilatation was observed in only 1 case (2 eyes). These results are different from ours. However, Machida's cases included 2 with unilateral involvement. Thus, there is some doubt whether all their cases were corticosteroid-induced CSC. Further investigation would be required to determine the answer to this question.

Based on IA findings, choroidal congestion appeared to be present in our 3 cases. It is possible that cases 1 and 2, with CSC, had partial choroidal congestion, and case 3, with bullous retinal detachment, had widespread choroidal congestion throughout the posterior pole. Were choroidal congestion severe, indocyanine green dye would permeate choroidal vessels, resulting in presenting intrachoroidal hyperfluorescence on IA. IA findings indicating choroidal circulatory disturbance, such as delayed choroidal filling or patchy hypofluorescence, are thought to be

secondary changes caused by preexisting choroidal congestion.

The mechanism of the development of CSC or bullous retinal detachment in patients with systemic corticosteroid treatment is now under investigation. Since the high urine level of catecholamine in patients under systemic corticosteroid administration is well known, one possibility which has been proposed is that a raised blood level of catecholamine increases the permeability of choroidal vessels.^{8,9} Kishimoto et al¹⁰ reported that corticosteroid inhibited the process of repairing retinal pigment epithelium under experimental conditions. Therefore, the outer blood-retinal barrier in the retinal pigment epithelium in patients under systemic corticosteroid treatment might easily be disrupted, leading to the development of serous retinal detachment, if some choroidal vascular abnormality preexists. As discussed earlier, IA findings in the present cases appeared to indicate focal or widespread choroidal congestion. IA findings of the choroids similar to those observed in the present cases have been reported in patients with idiopathic CSC or bullous retinal detachment without systemic corticosteroid treatment. It is dubious whether the choroidal changes seen in the present cases would be caused by corticosteroid administration. Iida et al^{4,5} stated that the unaffected eye had IA features similar to those seen in the affected eye in patients with unilateral idiopathic CSC. Our research suggests that se-

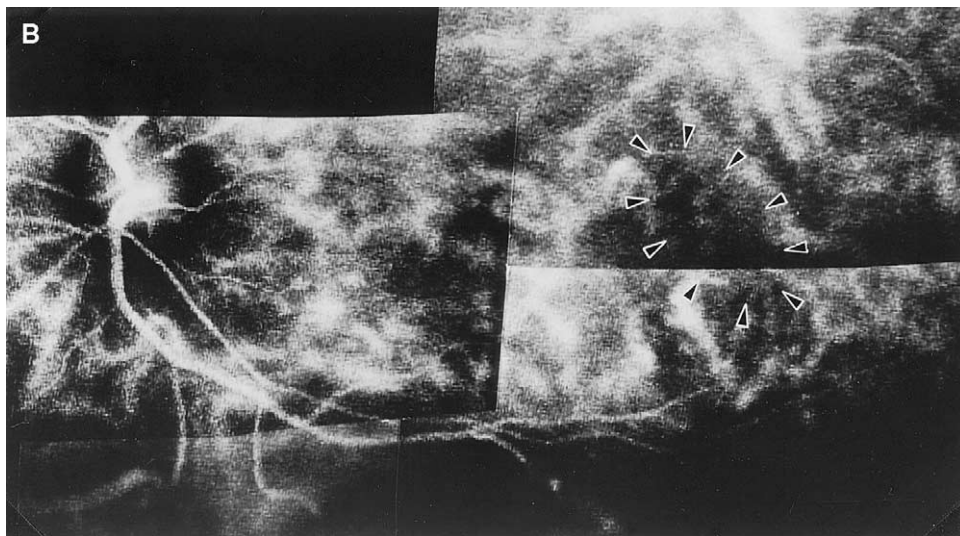
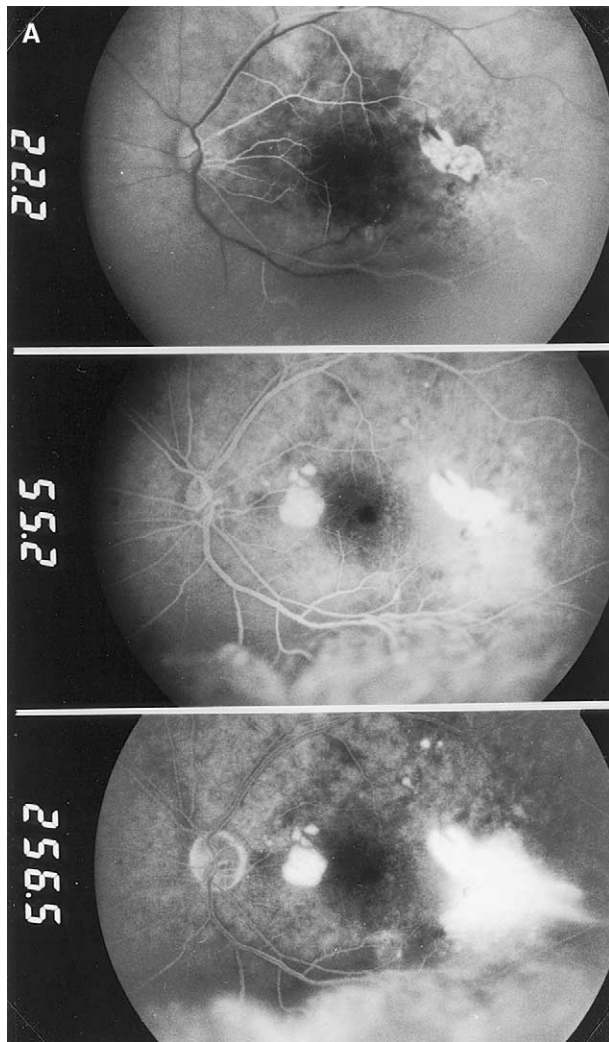


Figure 3. Case 3: 49-year-old woman with bullous retinal detachment and retinal pigment epithelial tear. **(A)** Fluorescein angiography of left eye showing marked dye leakage through retinal pigment epithelial tear. **(B)** Indocyanine green angiography of left eye showing dilatation of choroidal vein in posterior pole. Arrowheads indicate lesion in retinal pigment epithelial tear. **(C)** Late-phase indocyanine green angiography of left eye showing intrachoroidal hyperfluorescence in posterior pole (arrowheads).

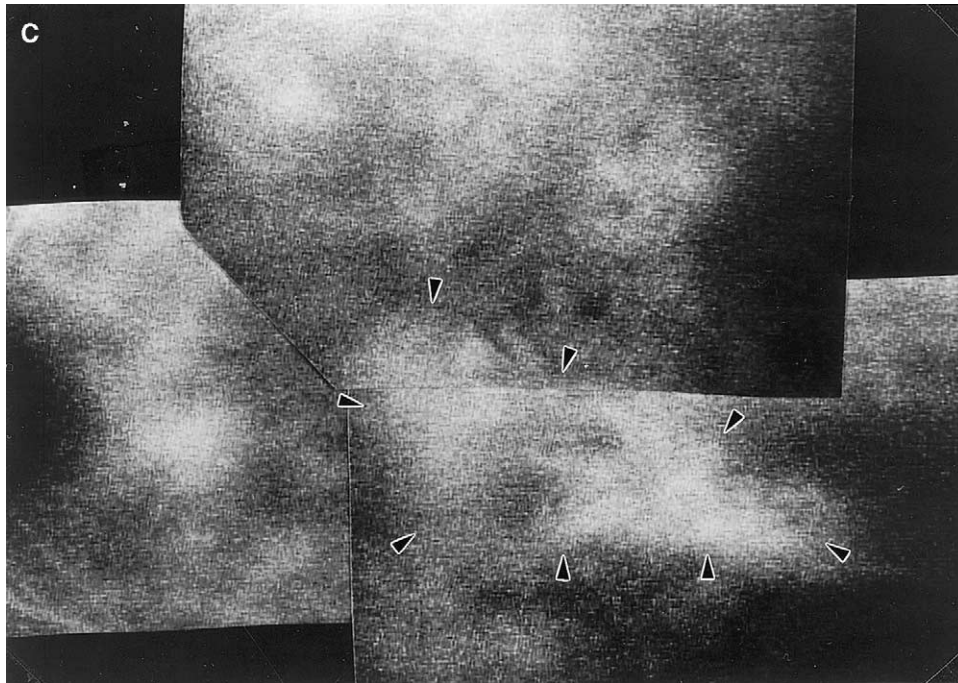


Figure 3. *Continued*

rous retinal detachment would not occur in the presence of choroidal circulatory abnormality alone. For the development of serous retinal detachment, other factors, including breakdown of the outer blood-retinal barrier in the retinal pigment epithelium, would be needed in addition to the presence of choroidal circulatory abnormality. Wakakura et al¹¹ suggested that corticosteroid administration was a predisposing factor, rather than a direct cause, in the disruption of the outer blood-retinal barrier.

On the other hand, it has been thought that serous retinal detachment develops independently in patients with malignant hypertension or pre-eclampsia. In such patients, choroidal circulation seems to be more severely disturbed than in CSC. Severe choroidal circulatory disturbance itself might damage retinal pigment epithelium, resulting in the development of serous retinal detachment.

In the present cases, corticosteroid might play a role in breaking down the outer blood-retinal barrier in the retinal pigment epithelium, if some choroidal circulatory abnormality preexisted before systemic corticosteroid administration. The problem is that the choroidal circulatory abnormality might congenitally or spontaneously develop before the occurrence of serous retinal detachment in patients with CSC and bullous retinal detachment, with or without systemic corticosteroid administration. It is

also possible that systemic corticosteroid administration might cause the occurrence of choroidal circulatory abnormality. Further investigation is needed.

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