

Indocyanine Green Angiographic Findings of Lacquer Cracks in Pathologic Myopia

Kyoko Ohno-Matsui, Naoto Morishima, Mutsuko Ito and Takashi Tokoro

*Department of Ophthalmology, School of Medicine,
Tokyo Medical and Dental University, Tokyo, Japan*

Abstract: Lacquer cracks are thought to represent healed mechanical breaks in the retinal pigment epithelium, Bruch's membrane, and choriocapillaris complex. In this study, we analyzed the indocyanine green (ICG) angiographic features of lacquer cracks and compared them with findings using fluorescein angiography. Complete ophthalmologic examinations, fluorescein angiography, and ICG angiography were performed in 29 consecutive patients (37 eyes) with lacquer cracks. Fluorescein angiograms of the cracks revealed linear hyperfluorescence in all 37 eyes. Using ICG angiography, we observed linear hypofluorescence in all 37 eyes. In 15 of 37 eyes, the length of the hypofluorescent lesion detected by ICG angiography was longer than the hyperfluorescent lesion observed by fluorescein angiography. In 17 of 37 eyes, more lacquer cracks were observed by ICG angiography than by fluorescein angiography. These findings indicate that ICG angiography can detect the development of the lesion more precisely, and may provide useful information for diagnosing pathologic myopia. **Jpn J Ophthalmol 1998;42:293-299** © 1998 Japanese Ophthalmological Society

Key Words: Fluorescein fundus angiography, indocyanine green angiography, lacquer cracks, pathologic myopia.

Introduction

Lacquer cracks are yellowish linear lesions found in the posterior fundus of 4.3% of highly myopic eyes.¹⁻⁵ Klein and Curtin⁶ suggested that lacquer cracks represented healed mechanical breaks of the retinal pigment epithelium, Bruch's membrane, and choriocapillaris complex, based on the fluorescein angiographic findings in 22 eyes with lacquer cracks.

The importance of lacquer cracks lies mainly in their prognostic significance. The vision-threatening complication of lacquer cracks is choroidal neovascularization (Fuchs' spot), which occurs in 57% of affected eyes.² We reported that lacquer cracks showed progression into various forms of myopic macular degeneration in a relatively short period.⁷ Therefore, it is important to detect the exact length

of lacquer cracks to identify which of these highly myopic eyes will develop complications.

Fluorescein fundus angiography has been the only diagnostic technique to confirm the appearance of lacquer cracks, so far. It is sometimes difficult to detect the exact length of lacquer cracks by fluorescein fundus angiography because of the simultaneous staining of surrounding myopic chorioretinal degeneration and because of the masking effect of subretinal hemorrhages, which are accompanied by the formation of fresh lacquer cracks.⁸

Recent improvements in the technology of indocyanine green (ICG) videoangiography have enhanced our ability to image the lesions that occur beneath the retinal pigment epithelium, such as lacquer cracks or choroidal neovascularization.⁹ In this study, we analyzed the ICG angiographic features of lacquer cracks, and compared these findings with those of fluorescein angiography.

Materials and Methods

A total of 37 eyes with lacquer cracks (29 patients) were examined in the high myopia outpatient clinic,

Received: April 11, 1997

Address correspondence and reprint requests to: Kyoko OHNO-MATSUI, MD, Department of Ophthalmology, Tokyo Medical and Dental University, 1-5-45 Yushima, Bunkyo-ku, Tokyo 113, Japan

Tokyo Medical and Dental University between January 1996 and December 1996, and were included in this study. Patients with prior ocular surgery or a specific condition, such as glaucoma, dense cataract, diabetes, or other form of retinal vascular disease, were excluded. These patients consisted of 10 men (13 eyes) and 19 women (24 eyes), ranging in age from 16 to 63 years (mean: 41.4 years). Refractive errors ranged from -6.0 to -27.0 D (mean: -15.3 D), and axial lengths, from 26.2 to 32.0 mm (mean: 29.8 mm).

All patients underwent general ophthalmologic examinations including indirect ophthalmoscopy and slit-lamp biomicroscopy with spherical $+78$ D or $+90$ D lens (Volk, Mentor, OH, USA). Axial length measurement by A mode ultrasonic scan (Alpha 2, Storz, St Louis, MO, USA) was performed in all patients. Color fundus photography, fluorescein fundus angiography, and ICG videoangiography were performed on the same day in each patient. Fluorescein angiograms were obtained with the use of a fundus camera (Pro 1, Kowa, Tokyo) after the intravenous injection of 5 mL of 10% sodium fluorescein. We performed ICG videoangiography for a 30-minute period after the injection of 50 mg ICG dye, using an ICG camera (TRC-50IA, Topcon, Tokyo) and a real-time video image quality improvement system (DVS-1000, Hamamatsu Photonics, Hamamatsu). The ICG videoangiographic sequence was videotaped on a videocassette recorder and was observed continuously on a videoscreen. Selected images were printed by a film recorder (FR-1100, Avio, Tokyo).

Results

The clinical findings of lacquer cracks are summarized in Table 1. Fundus examination revealed that lacquer cracks were yellowish, yellowish-white, or grayish irregular linear lesions in the posterior pole of each patient. The number of lacquer cracks in each eye varied from 1-13 (mean: 3.4). Twenty-nine of the 37 eyes had lacquer cracks involving the macular avascular area. Nine of the 37 eyes had choroidal neovascularization (Fuchs' spot) within the area of the lacquer cracks. All the Fuchs' spots were in the inactive stage, and none was accompanied by retinal edema or subretinal bleeding.

In all cases, fluorescein angiography revealed linear hyperfluorescence corresponding to the lacquer cracks from the early to the late phase of the angiogram. Fluorescein angiography revealed lacquer cracks more clearly than did ophthalmoscopy.

In the early phase of the ICG angiograms, we observed no abnormal fluorescence corresponding to

lacquer cracks in 36 of 37 eyes. In the left eye of Patient 1, however, middle-sized to large choroidal vessels behind the lacquer crack showed bright hyperfluorescence indicating window defect (Figure 1).

In the late phase of the ICG angiograms, we observed linear hypofluorescence in all eyes studied. The hypofluorescence became more apparent in the late phase (later than 20 minutes after dye injection), than in the early phase. Moreover, in 15 of 37 eyes (40.5%), the hypofluorescent lesions seen in the ICG angiograms were longer than the hyperfluorescent lesions observed in the fluorescein angiograms. In 17 of 37 eyes (45.9%), more lacquer cracks were observed in the late phase of ICG angiography than by fluorescein fundus angiography.

We describe two representative cases.

Patient 1: Hypofluorescence in the Late Phase of the ICG Angiogram

A 31-year-old woman was examined for high myopia in her left eye. Corrected visual acuity was right eye (RE): 20/30 and left eye (LE): 20/20. Ophthalmoscopic examination disclosed lacquer cracks in the macular area bilaterally (Figures 1A, 1B) and Fuchs' spot in her right eye. Fluorescein angiography showed linear hyperfluorescence corresponding to the lacquer cracks (Figures 1C, 1D). Although no lacquer crack was evident in the early phase of the ICG angiogram, middle-sized to large choroidal vessels were observed as bright hyperfluorescent spots (Figure 1E). In the late phase of the ICG angiogram, lacquer cracks disclosed a well-delineated linear hypofluorescent lesion (Figures 1F, 1G). This lesion continued to the temporal edge of the myopic crescent of the optic disc and was longer than the hyperfluorescent lesion observed by fluorescein angiography.

Patient 11: Numerous Hypofluorescent Areas on the ICG Angiogram

A 58-year-old man was examined for high myopia. Corrected visual acuity was RE: 20/20 and LE: 20/20. Ophthalmoscopic examination disclosed lacquer cracks and surrounding diffuse chorioretinal atrophy in the posterior fundus bilaterally (Figures 2A, B). Fluorescein angiography showed linear hyperfluorescence (Figure 2C) that was not obvious because of simultaneous tissue staining in surrounding diffuse chorioretinal atrophy. In the late phase of the ICG angiogram, numerous lacquer cracks were clearly detected as linear hypofluorescent lesions (Figure 2D).

Table 1. Clinical Findings in Patients With Lacquer Cracks

Patient Number	Age (y)	Sex	Eye	Visual Acuity	Refraction	Axial Length	Site of Lacquer Cracks	Number of Lacquer Cracks	Type of Fluorescence			
									FA Findings	ICG Findings		Fuchs' Spot
										Early Phase	Late Phase	
1	31	F	R	20/30	-7.5	28.3	M	2	hyper	none	hypo	+
			L	20/20	-6.0	27.9	M	1	hyper	dot hyper	hypo	-
2	27	M	L	20/20	-15.0	31.8	M	1	hyper	none	hypo	-
3	47	F	L	20/20	-26.0	32.0	M, E	5	hyper	none	hypo	-
4	27	M	R	20/60	-27.0	31.3	M	2	hyper	none	hypo	-
5	44	M	L	20/20	-9.0	26.5	M	1	hyper	none	hypo	-
6	38	M	R	20/25	-15.0	31.2	M, E	3	hyper	none	hypo	-
7	30	F	L	20/20	-10.5	28.5	M, E	4	hyper	none	hypo	-
8	58	M	R	20/20	-9.5	29.5	M	3	hyper	none	hypo	-
			L	20/20	-10.0	29.8	M	3	hyper	none	hypo	-
9	46	M	R	20/20	-14.0	30.8	M, E	2	hyper	none	hypo	-
			L	20/20	-14.0	30.9	M, E	4	hyper	none	hypo	-
10	32	F	L	20/25	-11.8	29.8	M, E	3	hyper	none	hypo	-
11	49	M	R	20/30	-19.0	30.0	M, E	3	hyper	none	hypo	-
12	51	M	R	20/50	-11.5	29.3	M	2	hyper	none	hypo	-
			L	20/60	-12.0	29.7	M	3	hyper	none	hypo	-
13	29	F	R	20/30	-20.5	30.1	M, E	13	hyper	none	hypo	+
			L	20/30	-18.0	30.7	E	12	hyper	none	hypo	-
14	59	F	R	20/40	-14.0	30.0	M	1	hyper	none	hypo	+
15	55	F	R	20/200	-16.0	30.2	M	4	hyper	none	hypo	+
16	63	F	L	20/30	-16.0	29.4	M	3	hyper	none	hypo	-
17	26	F	R	20/20	-19.5	30.8	E	1	hyper	none	hypo	-
			L	20/20	-20.0	30.7	E	2	hyper	none	hypo	-
18	16	F	L	20/40	-10.0	28.5	M, E	4	hyper	none	hypo	-
19	58	F	R	20/40	-22.0	31.1	M	5	hyper	none	hypo	-
20	38	F	R	20/20	-15.5	31.0	E	7	hyper	none	hypo	-
			L	20/20	-14.0	31.8	E	5	hyper	none	hypo	-
21	63	F	L	20/200	-12.0	26.2	M	1	hyper	none	hyp	+
22	43	M	L	20/20	-17.0	28.3	M	3	hyper	none	hypo	-
23	50	F	R	20/100	-23.0	30.9	M	2	hyper	none	hypo	+
24	30	F	R	20/20	-18.0	29.5	E	3	hyper	none	hypo	-
			L	20/40	-17.0	29.3	E	2	hyper	none	hypo	-
25	34	F	R	20/30	-13.0	29.2	M	3	hyper	none	hypo	-
			L	20/20	-13.5	29.0	E	4	hyper	none	hypo	-
26	32	F	L	20/20	-14.0	31.2	M, E	6	hyper	none	hypo	-
27	29	F	R	20/40	-9.5	28.8	M	1	hyper	none	hypo	+
28	50	F	L	20/70	-15.5	29.2	M	2	hyper	none	hypo	+
29	47	M	R	20/40	-25.0	30.9	E	2	hyper	none	hypo	-

ICG: indocyanine green. FA: fluorescein angiography. M: macular (including foveal avascular zone). E: extramacular. hyper: hyperfluorescence. Hypo: hypofluorescence.

Discussion

Until recently, ophthalmoscopy and fluorescein fundus angiography were the only technique to evaluate lacquer cracks. Klein and Curtin⁶ reported that fluorescein angiography of lacquer cracks showed continuous hyperfluorescence from the early to the late angiographic phase, which is compatible with our findings. The fluorescein angiographic finding appears as a window defect because of atrophy of

the overlying retinal pigment epithelium in the early phase of the angiogram and as tissue staining because of scar tissue in the late phase of the angiogram. These findings led Klein and Curtin⁶ to suspect that lacquer cracks were healed mechanical breaks of the retinal pigment epithelium, Bruch's membrane, and choriocapillaris complex.

On the other hand, almost all lacquer cracks in our patients showed linear hypofluorescence in the late

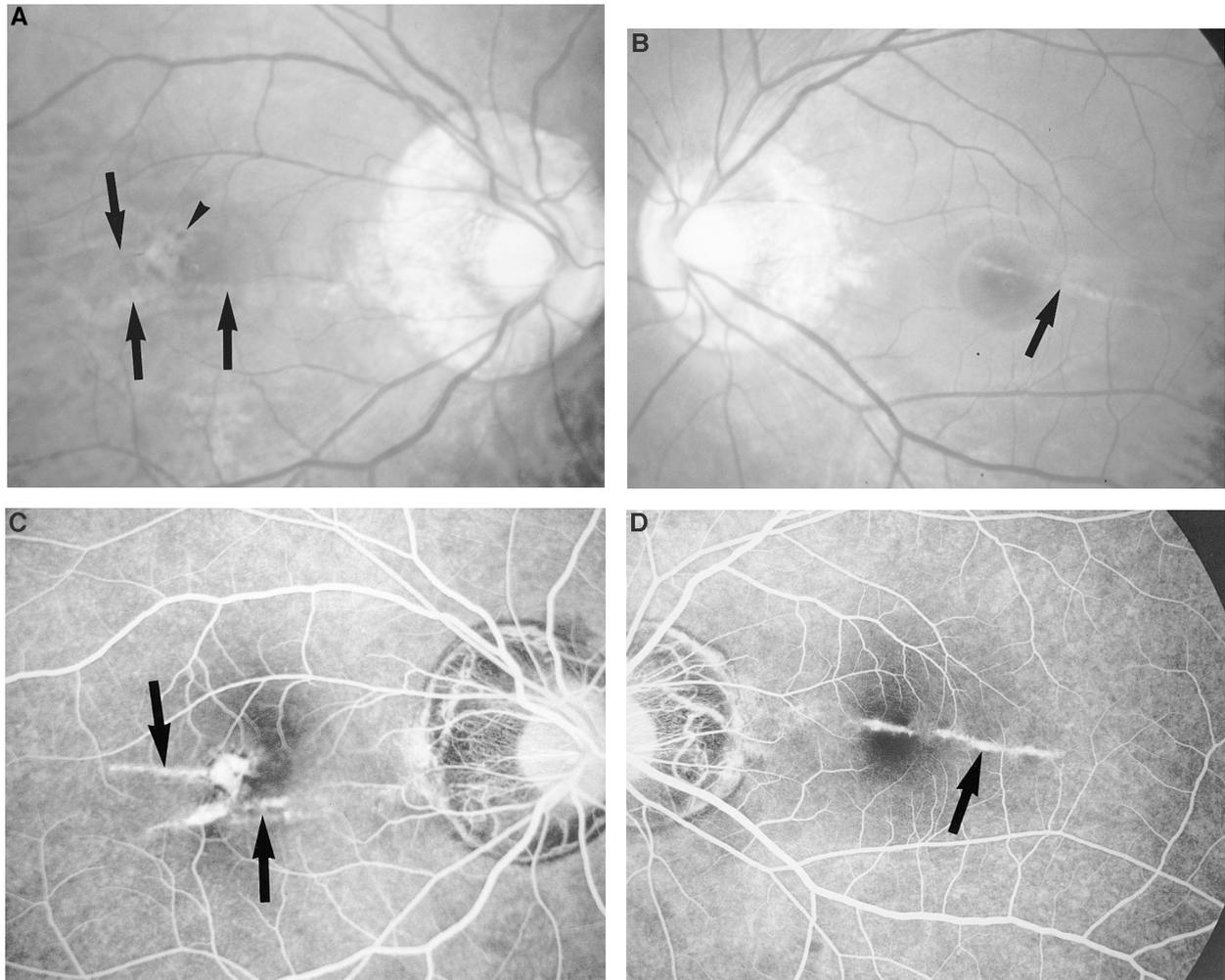


Figure 1. Case 1. (A) Right fundus shows lacquer cracks (arrows) with Fuchs' spot (arrowhead). (B) Left fundus shows one lacquer crack (arrow). (C) Fluorescein angiogram of right eye. (D) Fluorescein angiogram of left eye. Fluorescein angiograms of each eye show linear hyperfluorescence corresponding to lacquer cracks (arrow). (E) Early phase of indocyanine green (ICG) angiogram of left eye. No lacquer crack is evident. Middle-sized to large choroidal vessels behind lacquer crack are observed as bright hyperfluorescent spots (arrow). (F) Late phase of ICG angiogram in right eye. (G) Late phase of ICG angiogram in left eye. Lacquer cracks disclose well-delineated linear hypofluorescent lesion (arrows) in late phase of ICG angiogram. This lesion continues to temporal edge of myopic crescent of optic disc, and is longer than hyperfluorescent lesion observed by fluorescein angiography (Figure 1D).

phase of the ICG angiogram, which was contrary to the fluorescein angiographic findings that showed hyperfluorescence. On fluorescein angiography, rapid dye leakage from the surrounding normal choriocapillaris occurs, and the scar tissue within lacquer cracks is stained immediately by fluorescein dye. However, the large ICG molecule is bound almost completely to protein,¹⁰ rendering it impermeable in the choriocapillaris, which prevents the diffusion of ICG dye in the lesion of lacquer cracks. The free fraction of ICG dye, which can diffuse unrestrained,

has only a minimal capacity to fluoresce, in comparison with the bound fraction, because of a low albumin concentration. As a result, lacquer cracks do not show hyperfluorescence by ICG angiography.

There are still some controversies in the interpretation of hypofluorescence observed in the late phase of the ICG angiogram. This late hypofluorescence may result from the masking effect of the scar tissue within lacquer cracks. In the early phase of the ICG angiogram, the intense fluorescence of large to middle-sized choroidal vessels prevents the observa-

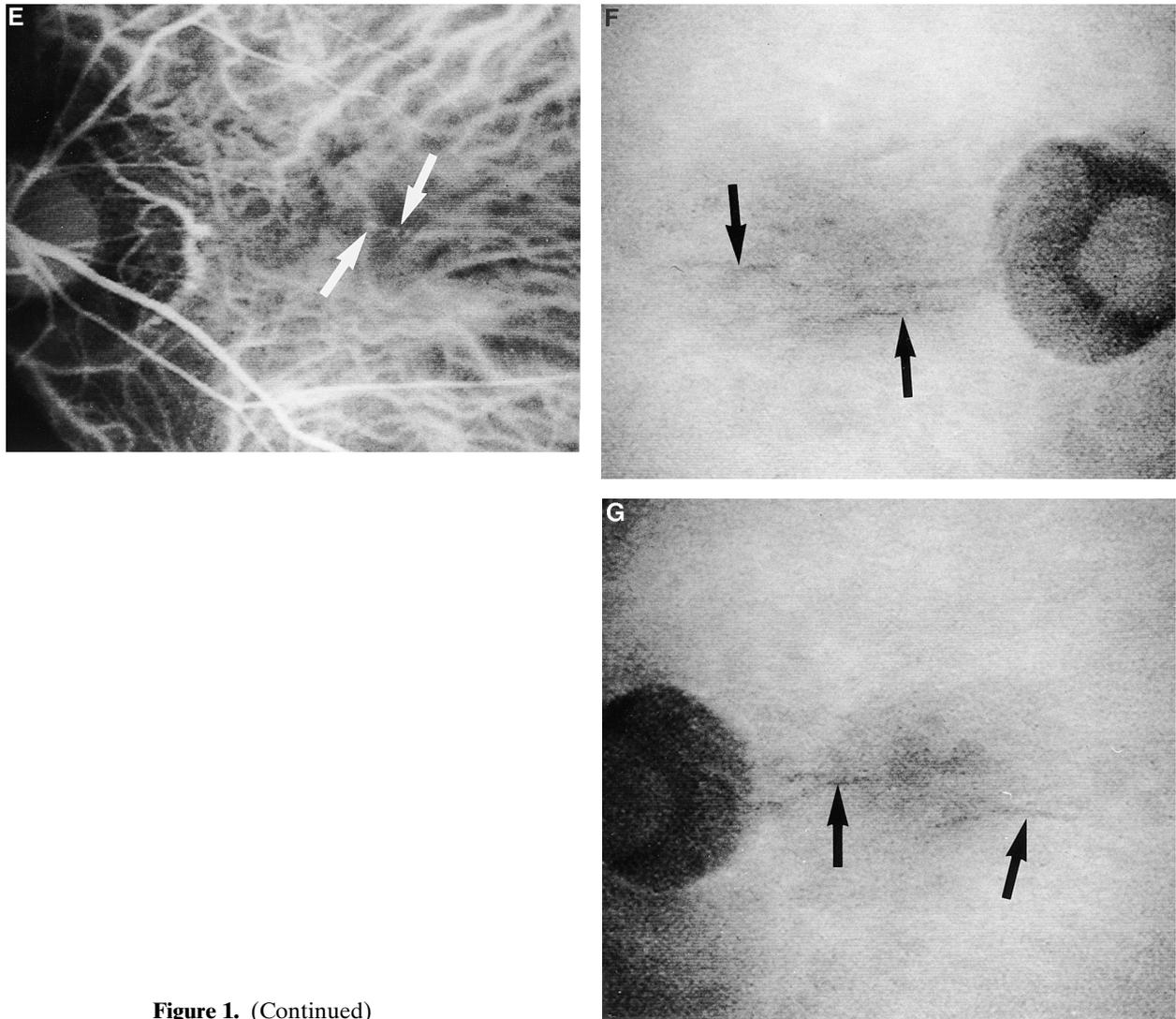


Figure 1. (Continued)

tion of mild hypofluorescence, like lacquer cracks. However, in the late phase of angiogram, when the fluorescence in the overall ocular fundus predominantly decreases, even mild scar tissue could show hypofluorescence. Alternately, choroidal filling defect beneath lacquer cracks may have additional hypofluorescence. Hypofluorescence in the ICG angiogram could be observed just after the onset of the rupture in Bruch's membrane complex.⁸ Based on these findings, ICG angiography may show earlier lesions, such as rupture of the Bruch's membrane-choriocapillaris complex with mild scarring, before the development of atrophy of the retinal pigment epithelium. Interpretation of ICG angiograms is still controversial because the behavior of the dye and its

diffusion across vascular barriers into extravascular space have not been clarified.

In this study, ICG angiography revealed the size, number, and extent of lacquer cracks more clearly than fluorescein angiography. Indocyanine green angiography is less affected by alteration of the retinal pigment epithelium, the simultaneous staining of the surrounding myopic chorioretinal degeneration, and subretinal bleeding, factors that occasionally accompany lacquer cracks because of the characteristics of the dye. Therefore, ICG angiography can yield different information than fluorescein angiography.

Lacquer cracks are reported to progress into various myopic macular lesions, including myopic choroidal neovascularization.⁷ Indocyanine green angiography

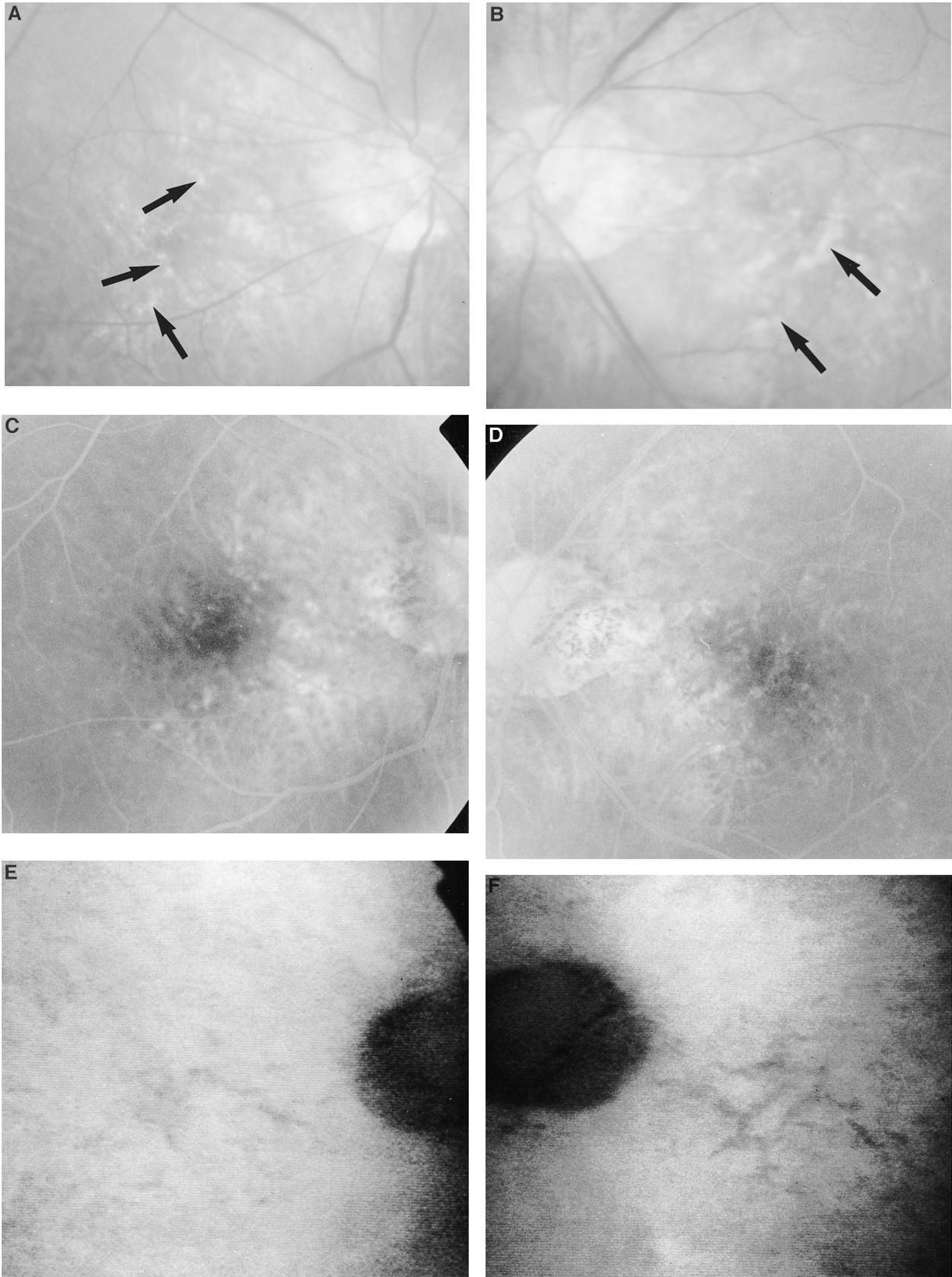


Figure 2. Case 2. **(A)** Color fundus photograph of right eye. **(B)** Color fundus photograph of left eye. Ophthalmoscopic examination discloses lacquer crack (arrows) and surrounding diffuse chorioretinal atrophy in posterior fundus bilaterally. **(C)** Fluorescein angiogram in right eye. **(D)** Fluorescein angiogram in left eye. Fluorescein angiograms show faint hyperfluorescence corresponding to lacquer cracks. Hyperfluorescence is not obvious because of simultaneous tissue staining of surrounding diffuse chorioretinal atrophy. **(E)** Late phase of indocyanine green (ICG) angiogram in right eye. **(F)** Late phase of ICG angiogram in left eye. Late phase of ICG angiogram detects more lacquer cracks that are clearly linear hyperfluorescent lesions.

provides more exact information for the diagnosis of lacquer cracks in patients with pathologic myopia.

This work was supported by a grant for Retinochoroidal Atrophy Research from the Ministry of Health and Welfare of Japan.

References

1. Curtin BJ, Karlin DB. Axial length measurements and fundus changes of the myopic eye. *Am J Ophthalmol* 1971;71:42–53.
2. Curtin BJ. Ocular findings and complications. In: Burgower W, Winters R, Barishek VM, eds. *Myopias*. Philadelphia: Harper & Row, 1985:309–16.
3. Hayashi K. Macular lesions in pathologic myopia. *Jpn J Clin Ophthalmol* 1978;32:271–84.
4. Salzmann M. The choroidal changes in pathologic myopia. *Arch Ophthalmol* 1902;31:41–2.
5. Tokoro T, Maruo T, Kanai J, Hayashi K. Report of Research Committee on Choroidal Degenerations. Tokyo: Ministry of Health and Welfare of Japan, 1987:1–14.
6. Klein RM, Curtin BJ. Lacquer crack lesions in pathologic myopia. *Am J Ophthalmol* 1975;79:386–92.
7. Ohno-Matsui K, Tokoro T. The progression of lacquer cracks in pathologic myopia. *Retina* 1996;16:29–37.
8. Ohno-Matsui K, Ito M, Tokoro T. Subretinal bleeding without choroidal neovascularization in pathologic myopia. A sign of new lacquer crack formation. *Retina* 1996;16:196–202.
9. Flower RW, Hochheimer BF. A clinical technique and apparatus for simultaneous angiography of the separate retinal and choroidal circulations. *Invest Ophthalmol Vis Sci* 1973;12:248–61.
10. Cherrick GR, Stein SW, Leevy CM, Davidson CS. Indocyanine green: observations on its physical properties, plasma decay, and hepatic extraction. *J Clin Invest* 1960;39:592–600.