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Indocyanine Green Angiographic Findings in 3 Patients with Traumatic Hypotony Maculopathy

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Purpose: Little is known about the choroidal circulation in human eyes with ocular hypotony. Recently, indocyanine green angiography (IA) became a useful method for examining choroidal circulation. The present study using IA was designed to determine choroidal circulatory disturbances in patients with traumatic hypotony maculopathy.

Methods: Indocyanine green angiography was performed on 3 consecutive patients (3 eyes) with traumatic hypotony. One patient underwent IA using an infrared fundus camera only during the hypotony stage. The other 2 patients underwent IA using a scanning laser ophthalmoscope before and after recovery of intraocular pressure (IOP).

Results: During the hypotony stage, IA revealed multiple hypofluorescent spots in many parts of the fundus, sector hypofluorescent areas, dilatation, and tortuosity of the choroidal vessels in the posterior pole. These findings had not been detected by fluorescein angiography. After surgical treatment, IOP returned to the normal range and visual acuity improved. Indocyanine green angiography showed improvement of the sector hypofluorescent areas, and dilatation and tortuosity of choroidal vessels in the posterior pole. However, most of the hypofluorescent spots and regional delay of choroidal filling remained.

Conclusions: Indocyanine green angiography revealed that choroidal circulatory disturbances occurred during the hypotony stage and that some remained during the recovery stage. **Jpn J Ophthalmol 2000;44:283–289**

Key Words: Blunt trauma, choroidal circulation, hypotony maculopathy, indocyanine green angiography.

Introduction

An excessive decrease in intraocular pressure (IOP), induced by trauma or surgery, is known to cause hypotony maculopathy,[1–3] which leads to severe visual loss. In animal experiments, a decrease in IOP influenced choroidal perfusion as well as retinal circulation.[4–6] However, little data concerning choroidal circulation in human eyes with ocular hypotony have been obtained because there has not been a suitable method for the clinical observation of choroidal circulation.

In recent years, indocyanine green angiography (IA) was developed to investigate choroidal circulation and has been applied to many choroidal disorders.[7–9] Indocyanine green (ICG) has two properties that make it superior to sodium fluorescein for investigating choroidal circulation.[7–9] First, the absorption peak of ICG is at wavelength 790 to 805 nm and the emission peak is at 835 nm, both of which readily penetrate the melanin in the retinal pigment epithelium and the xanthophyll. Second, approximately 98% of ICG binds to plasma proteins, and the protein-bound ICG molecules can barely pass through the fenestration of the choriocapillaris. These properties facilitate the visualization of the choroidal vasculature.

In this study, the disturbance of choroidal circula-

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tion under ocular hypotonic conditions caused by blunt trauma and the recovery were evaluated by IA.

Materials and Methods

Patients

Three eyes of 3 consecutive patients (3 men aged 40, 56, and 21 years) with hypotony maculopathy were studied. Hypotony maculopathy had been caused by blunt trauma, and the patients had been treated by several surgical procedures.

This study was conducted according to the principles established in the Declaration of Helsinki. Informed consent was obtained from each patient.

Case 1. On April 10, 1991, a 40-year-old man was referred to Kochi Medical School Hospital because of hyphema in his left eye after he had been punched in the face 3 days previously. The best corrected visual acuity in his left eye was 0.04, and the IOP in this eye was 2 mm Hg. When the hyphema disappeared 3 days after the visit, ophthalmoscopy revealed a swollen disc and chorioretinal folds in the macular area. Gonioscopy showed a wide cyclodialysis cleft. Because medical treatment with topical atropine sulfate (1%) had no effect on the hypotony, several surgical procedures (argon laser photocoagulation,[10] scleral buckling, and SF6 gas injection into the vitreous cavity) were attempted, but the IOP pressure in the patient's left eye remained low. Fluoroscein angiography and IA were performed on October 9, 1991 (Figures 1,2), and FA revealed cystoid macular edema. The IOP was 4 mm Hg, and the best corrected visual acuity was 0.03. Intraocular pressure was normalized after direct suturing of the cyclodialysis clefts,[3,11] on October 21, 1991. At the last follow-up examination on June 13, 1995, visual acuity was 0.3 and IOP was 10 mm Hg.

Case 2. On September 26, 1996, a 56-year-old man was referred to Kochi Medical School Hospital for hypotony maculopathy in his right eye. On June 26, 1996, a small stone had hit his right eye, and the patient underwent treatment at an eye clinic with topical atropine and argon laser photocoagulation to the cyclodialysis cleft, but the IOP did not return to normal. When the patient first visited our clinic, the best corrected visual acuity in his right eye was 0.2, and the IOP in that eye was 4 mm Hg. Ophthalmoscopy revealed a swollen disc and chorioretinal folds in the macular area. Fluorescein angiography showed leakage from the right optic disc and black striae throughout the posterior segment of the fundus (Figure 3). Gonioscopy showed a definite cyclodialysis cleft in two quadrants extending from 4:00 to 8:30 o'clock. On October 21, 1996, SF6 gas injection into the vitreous cavity was attempted. Fluorescein angiography and IA were performed on October 18, 1996 (Figures 4,5,7A), before, and on March 19, 1997 (Figures 6,7B), after this procedure. After the IOP changed from 6 to 12 mm Hg, the best corrected visual acuity returned to 1.0.

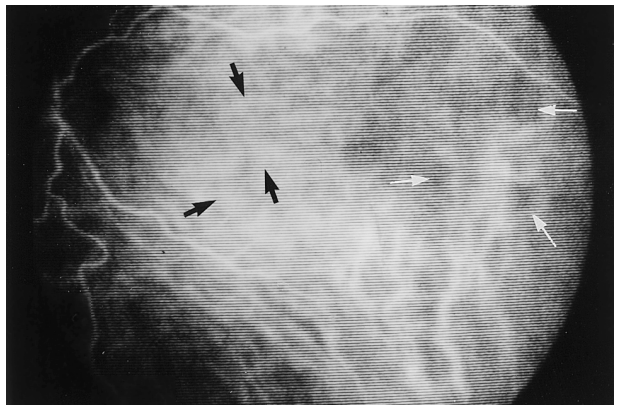

Figure 1. Case 1. Indocyanine green angiogram showing dilatation and tortuosity of choroidal vessels (black arrows) in posterior pole and scattered hypofluorescent spots (white arrows) at 4.5 minutes after dye injection.

Case 3. On September 4, 1996, a 21-year-old man was referred to Kochi Medical School Hospital for hypotony maculopathy in his left eye. He had fallen from a ladder on May 25, 1996, and sustained blunt trauma to his left eye. He had been treated at an eye clinic with topical atropine and argon laser photocoagulation to a cyclodialysis cleft. When he visited our clinic, the best corrected visual acuity of his left eye

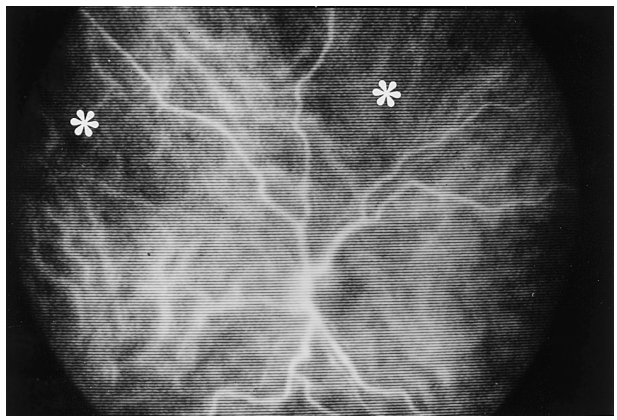

Figure 2. Case 1. Indocyanine green angiogram showing sector hypofluorescent areas (asterisks) at 4 minutes after dye injection.

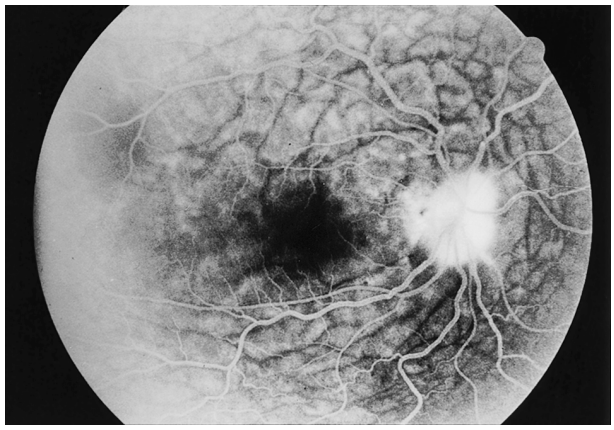

Figure 3. Case 2. Fluorescein angiogram showing leakage from the right optic disc and hypofluorescent lines at sites with corresponding choroidal folds.

was 0.1, and the IOP was 1 mm Hg. Ophthalmoscopy revealed a swollen disc and chorioretinal folds in the macular area. Gonioscopy showed a cyclodialysis cleft extending from 8:00 to 12:00 o'clock. On November 18, 1996, phacoemulsification, intraocular lens implantation, and SF6 gas injection into the vitreous cavity were performed, and IOP was normalized. The best corrected visual acuity returned to 1.5. Fluorescein angiography and IA (Figure 8) were performed before and after these procedures.

Indocyanine Green Angiography

Indocyanine green angiography was performed after FA. For case 1, a fundus camera (TRC 50-IA; Topcon Instruments, Tokyo) was used and IA was carried out when hypotony continued despite intervention with several surgical procedures. We used a 50° field for observation. The IA images were stored on a 2.5-inch floppy disk. For cases 2 and 3, IA was performed during the follow-up period as well as at the initial visit by using a scanning laser ophthalmoscope (Rodenstock Instruments, Munich, Germany). We used a 40° field for observation. The images were recorded on S-VHS videotapes.

In all 3 cases, 25 mg of dye (Diagnogreen; Daiichi Pharmaceutical, Tokyo) dissolved in 1.0 mL of distilled water, was injected into the cubital vein, followed by a 5-mL flush with balanced saline solution. If necessary, 25 mg of dye was reinjected in the late phase of IA. Indocyanine green angiography is roughly divided into early, middle, and late phases.[7] In healthy cases, the early phase angiograms clearly show choroidal and retinal vessels. In the middle phase (6–15 minutes after injection), choroidal vessels become invisible because ICG fills the choriocapillaris. In the late phase (beyond 18–22 minutes), angiograms show choroidal vessels as hypofluorescent channels, and retinal vessels are no longer visible.

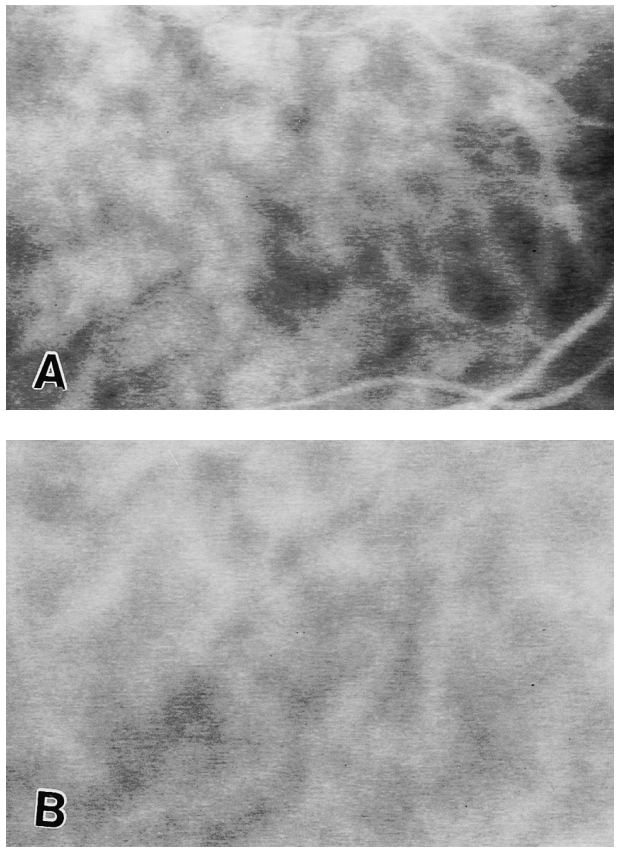

Figure 4. Case 2. (**A**) Indocyanine green (ICG) angiogram showing dilatation and tortuosity of choroidal vessels in the posterior pole at 53.5 seconds after injection. (**B**) ICG angiogram showing definite dilatation and tortuosity of choroidal vessels even at 21 minutes after dye injection.

Results

Indocyanine angiography revealed the following in all 3 cases during hypotony: scattered hypofluorescent spots (Figures 1,5), dilatation and tortuosity of the choroidal vessels in the posterior pole (Figures 1,4), and sector hypofluorescent areas (Figures 2,5). Scattered hypofluorescent spots and dilatation and tortuosity of the choroidal vessels in the posterior pole were seen from the early phase to the late phase of IA. Sector hypofluorescent areas were observed from the early to the middle phase of IA, but these areas disappeared in the late phase of IA. Because scattered hypofluorescent spots were very dark, they were identified even in the sector hypoflu-

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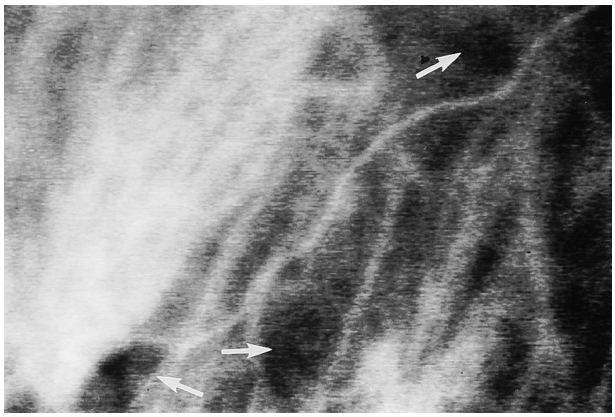

Figure 5. Case 2. Indocyanine green angiogram showing sector hypofluorescent area and scattered hypofluorescent spots (arrows) the same as in case 1, at 4 minutes after dye injection.

orescent areas (Figure 5) Although hypofluorescent spots were also seen in the opposite eye in case 2, the number of spots was much fewer than in the affected eyes. Except for the hypofluorescent spots, these findings could not be detected by FA; most of the hypofluorescent spots on FA were distinguishable only in the early phase and vanished in the late phase.

In contrast to the quick recovery of visual acuity and abnormal retinal findings (ie, retinal folds and dilatation and tortuosity of retinal vessels), recovery from the abnormal choroidal findings occurred slowly after the IOP was normalized by several surgical procedures (Figures 6,7). However, some hypofluorescent spots remained after IOP recovery (Figure 7). A part of the choriocapillaris in the posterior pole where choroidal filling delay was detected was seen as a lobular structure (Figure 8) in case 3.

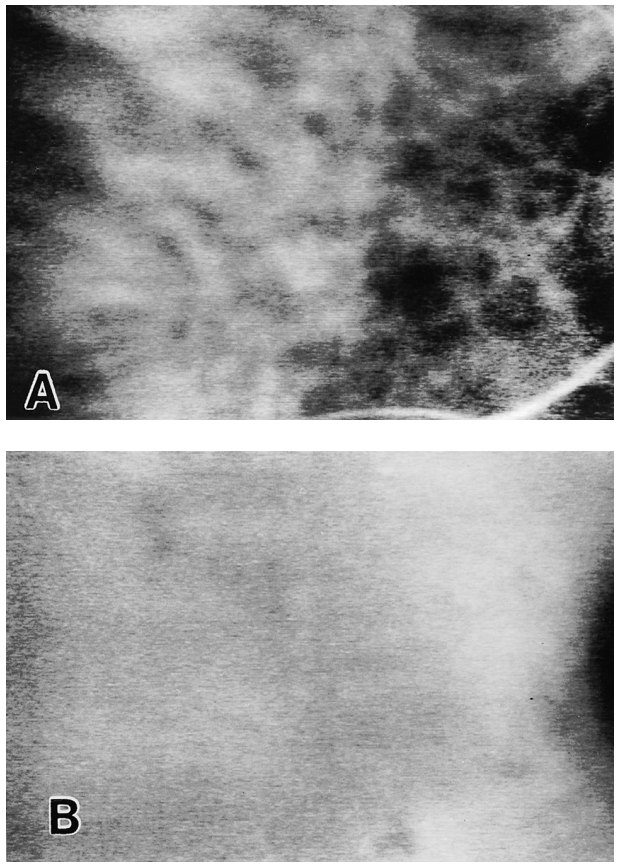

Figure 6. Case 2. (**A**) Indocyanine green angiogram taken when intraocular pressure returned to normal after a surgical procedure. After dye injection, choriocapillaris is filled with dye from temporal side of the macula. (**B**) Compared with Figure 4B, late-phase (24-minute) angiogram reveals homogeneous background choroidal fluorescence.

Discussion

In this study, IA revealed scattered hypofluorescent areas, sector hypofluorescent areas, dilatation and tortuosity of the choroidal vessels in the posterior pole, and the lobular filling of the choroid. These findings were not revealed by FA. Many hypofluorescent spots were seen from the early phase to the late phase of IA. Ophthalmoscopic examination demonstrated that pigment did not exist in the regions that were identified by IA to be hypofluorescent. In addition, IA revealed that medium or large choroidal vessels ran beneath these hypofluorescent spots. These hypofluorescent spots became smaller and obscured in the late phase of IA. From these results, we conclude that the scattered hypofluorescent spots were not caused by blockage by pigments but rather were caused by filling defects in the choriocapillaris. The hypofluorescent spots were detected in affected eyes in all cases and in the opposite eye in case 2. Similar hypofluorescent spots have been observed in elderly patients.[12] Case 2 was a 56-year-old man, and because he was older, the hypofluorescent spots could be observed in his opposite eye.

Sector hypofluorescent areas seemed to correspond to the areas supplied by the ciliary arteries.[13] These areas were roughly triangular with the apexes of the wedges pointed to the macula (Figure 2). These hypofluorescent areas became indistinct in the late phase of IA, further indicating that the sector hypofluorescent areas were caused by a disturbance of the supply from the ciliary arteries. However, the

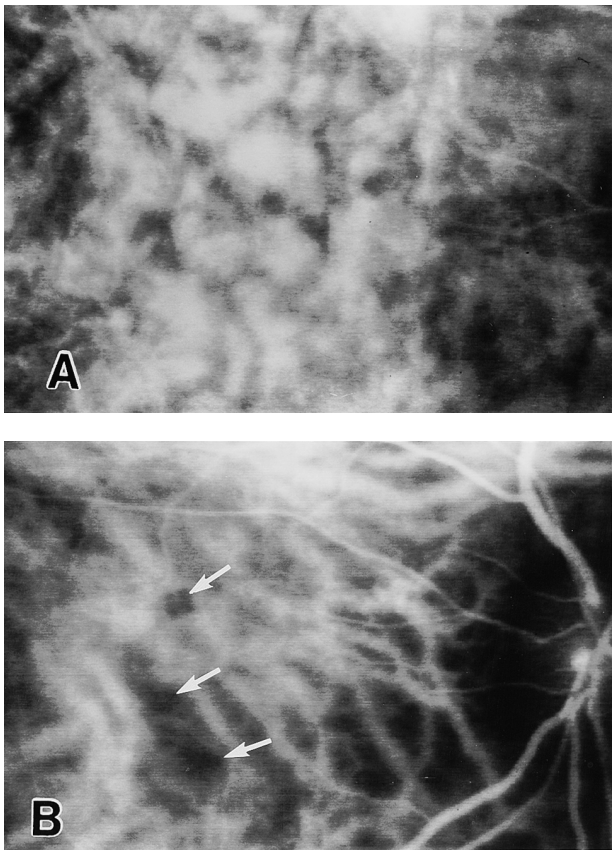

Figure 7. Case 2. (**A**) Indocyanine green (ICG) angiogram before recovery of intraocular pressure (IOP). Dilatation and tortuosity of choroidal vessels in posterior region become more definite after ICG was again injected in late phase of ICG angiography. (**B**) ICG angiogram taken 5 months after recovery of IOP. Choroidal vessels in posterior pole are almost normal. Note remaining scattered dense hypofluorescent spots (arrows).

severe damage seen in triangular syndrome did not develop.

Dilatation and tortuosity of the choroidal vessels in the posterior pole were also detected by IA. We could not determine whether these vessels were choroidal arteries or veins. In general, the choroidal arteries branch radially toward the periphery and the

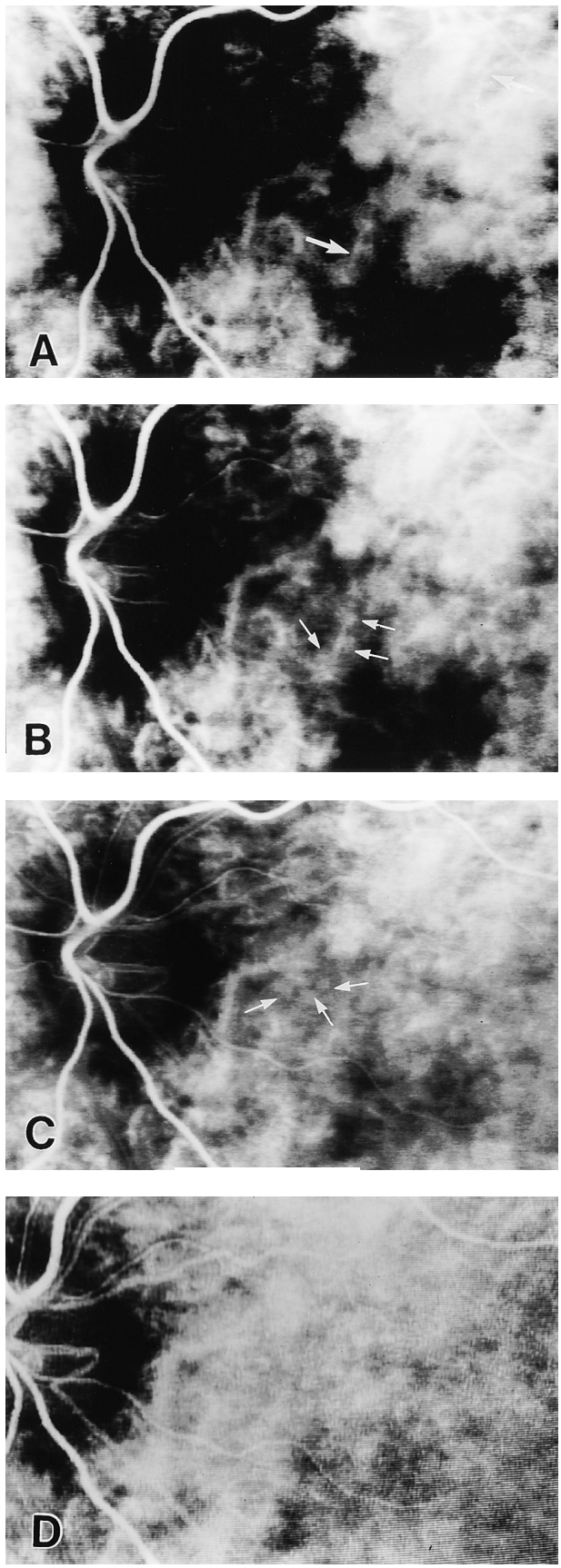

Figure 8. Case 3. Indocyanine green (ICG) angiograms taken 3 months after recovery of intraocular pressure. (**A**–**D**) ICG angiograms showing lobular filling pattern every second. (**A**) Artery filling phase, with arrows indicating choroidal arteries. Note distinct delay of choroidal filling in posterior pole. (**B**) After choroidal arterial filling, small hyperfluorescent spots (arrows) appear, looking like a bunch of grapes. (**C**) These hyperfluorescent spots appear one after another (arrows). (**D**) Area with delayed choroidal filling gradually becomes homogeneous background choroidal fluorescence.

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choroidal veins gather into a vortex vein as shown on IA.[14] Moreover, the arterial and venous filling phases of the choroidal vessels are different. These differences usually enable us to distinguish between choroidal arteries and veins. However, the characteristic arterial and venous phases were not apparent because the arterial filling phase became obscure in the early phase of IA when the dye was injected slowly. We suggest that this discrepancy was caused by a delay in choroidal circulation. Comparison of the two angiograms before and after recovery from dilatation and tortuosity of the choroidal vessels showed that choroidal veins were dominant only where abnormal vessels were detected. Therefore, we suggest that most of these abnormal vessels are choroidal veins (Figure 7). In ICG angiograms in healthy cases, choroidal vessels become conspicuous in relief as hypofluorescent channels in the late phase.[7] However, these abnormal vessels were prominent hyperfluorescent channels even in the late phase. We conclude that this finding was caused by the congestion of blood flow in the choroid. In normal conditions, the transmural pressure (the pressure in the choroidal veins minus the IOP) in the choroidal veins is very low.[15] From this, we conclude that the venous congestion was induced by the markedly decreased IOP.

The lobular filling of the choriocapillaris was seen during the recovery stage of hypotony in case 3 (Figure 8). The lobular filling of the choriocapillaris cannot be detected by FA or IA under normal conditions, because it occurs too quickly to be observed even with a video recorder in slow motion. Moreover, leakage of sodium fluorescein from fenestrations of the choriocapillaris obscures the lobular filling pattern of the choriocapillaris. Studies in primates showed that the lobular filling pattern could be visualized by elevating the IOP.[16,17] Hayreh hypothesized that elevating the IOP slowed down the choroidal circulation. In case 3, after the IOP returned to normal, the choriocapillaris filling pattern became apparent in the posterior pole. Transit of ICG dye into each lobule through small choroidal arteries appeared at different times. We conclude that this time lag of blood flow into successive lobuli enabled us to observe the lobular filling pattern.

In previous reports[4–6] that described the relationship between the IOP and choroidal perfusion, many researchers have asserted that a decrease in IOP will increase choroidal perfusion. Contrary to this, we observed local choroidal circulatory disturbance when IOPs were as low as that previously reported. The difference between previous reports and our cases was the duration of the low IOP (ie, transient or sustained). Therefore, we conclude that sustained, but not transient, hypotony was the cause of the choroidal circulatory disturbance. Impairment of the autonomic nervous system[15] may also have regulated choroidal circulation.

Visual acuity immediately improved after IOP became normal, but a part of the choroidal circulatory disturbance persisted. A careful follow-up is important here, because it is difficult to predict the influence of sustained choroidal circulatory disturbance on vision.

The findings revealed by IA in our cases were not observed by FA. Indocyanine green angiography was a useful method for detecting and recognizing the degree of choroidal circulatory disturbance in patients with traumatic hypotony.

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