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Indocyanine green angiography of central serous chorioretinopathy

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Abstract

Indocyanine green angiograms of central serous chorioretinopathy in 30 eyes were evaluated in order to demonstrate the pathologic features of the retinal pigment epithelium. In 5 of the cases, the dye leakage or the pigment epithelial degeneration was seen to be associated with areas of choroidal circulatory insufficiency. This implies that local choroidal ischemia is one of the causes of the pigment epithelial disturbance.

Introduction

Indocyanine green (ICG) angiography, which was developed by Flower and Hochheimer (1) in 1973, is a useful method for basic studies on choroidal vascular hemodynamics. However, this method is less appropriate for brown eyes, which have more pigment than blue eyes, and has only limited clinical applications because of its failure to provide helpful findings in the diagnosis and management of chorioretinal diseases.

ICG angiography by means of an infrared sensitive video camera, which was developed by us (2) in 1984, is so sensitive to ICG fluorescence that it is possible to take ICG angiograms also from brown eyed subjects (like Japanese) and to evaluate the pathologic characteristics of chorioretinal diseases.

The purpose of this study is to demonstrate the pathologic features of central serous chorioretinopathy (CSC) as shown by this method.

Patients and methods

Thirty-six patients (38 eyes) with suspected CSC in the macula were selected for this study. The age of the patients ranged from 32 to 61 years. After injection of an ICG solution into a cubital vein, ICG angiography was performed on each patient using a modified Topcon fundus camera fitted with an infrared sensitive video camera. The apparatus and method have been described in more detail elsewhere (2). After the ICG angiography, fluorescein angiograms were recorded by means of a standard Kowa fundus camera.

Results and discussion

In 38 cases, 30 eyes with the fluorescein dye leaking through the damaged pigment epithelium were evaluated for the purpose of this study. In those

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ICG angiograms, we found two things: 1. ICG was leaking from the choriocapillaris through a defect in the pigment epithelium into the subretinal space. 2. Hyperfluorescent areas existes close to or surrounding the leaking point. We now proceed to report in more detail on those findings.

1. ICG dye leaking

It has been believed (3) that ICG dye does not tend to leak from the fenestrated choriocapillaris into the choroidal space as opposed to fluorescein dye. 98% of the injected IC is bound to serum albumin, while on the other hand, 20 to 40% of sodium

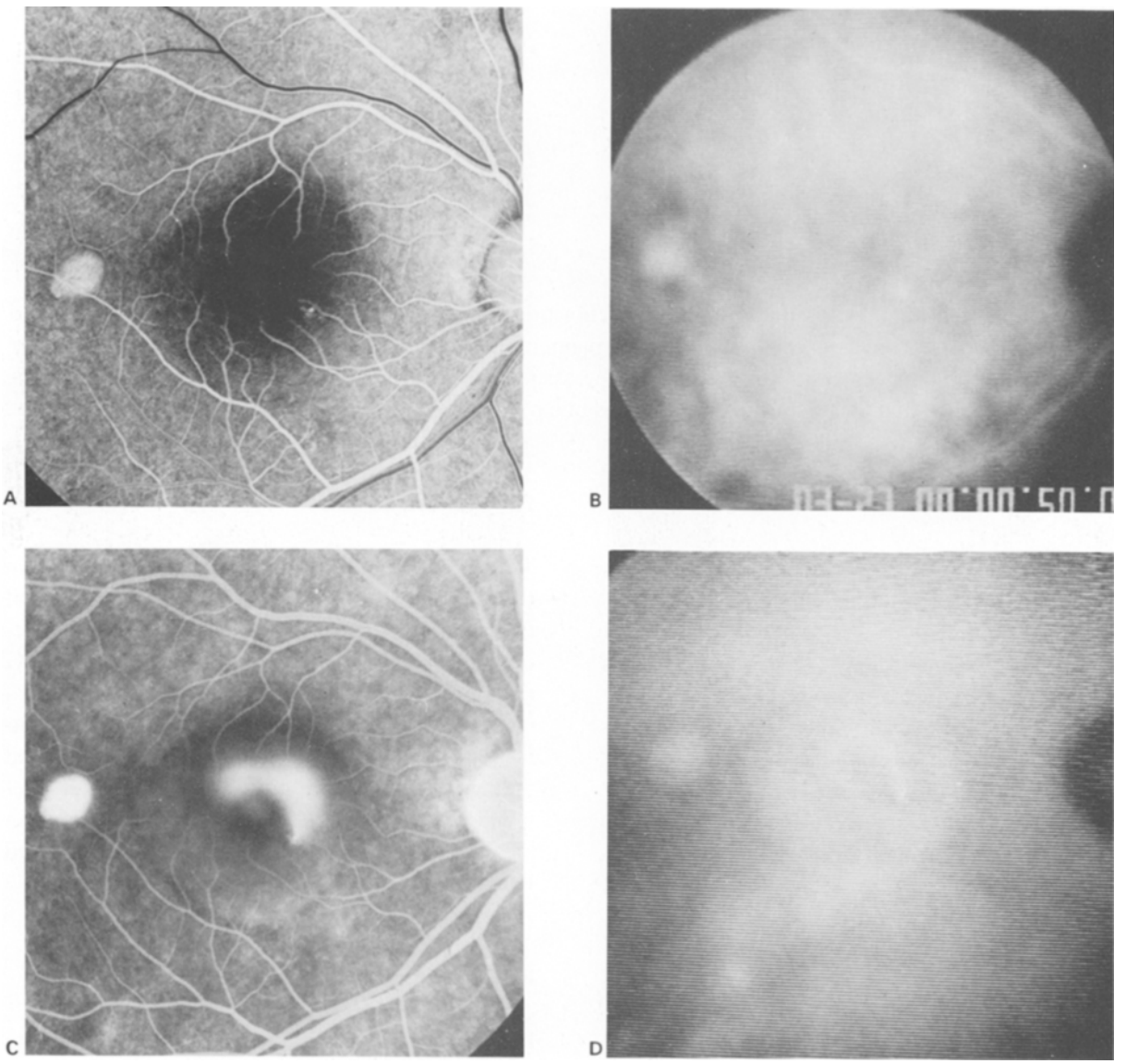

Fig. 1. The right eye of a 33-year-old male with central serous chorioretinopathy. (a). A pinpoint leaking spot naso-inferior to the fovea and a small pigment epithelial detachment temporal to the macula are visible in the early retinal venous phase of fluorescein angiography. (b). In the ICG angiogram 50 seconds after dye injection, a bright fluorescent spot and a hyperfluorescent region are demonstrated corresponding to those in the fluorescein angiogram. (c). In the late fluorescein angiogram 17 minutes after dye injection, pooling of fluorescein dye in the subretinal space of the macula was observed. (d). In the ICG angiogram, a hyperfluorescent area which corresponds to the pooling of fluorescein dye appears in the macula.

fluorescein is unblound in the circulating blood. As to the permeability of the choriocapillaris, Bill (4) has postulated that the choroidal blood vessels are highly permeable even to substances as myoglobin, albumin and gamma globulin (molecular weight 17.000, 67.000, 156.000 respectively). Therefore, it would be possible that albimin-bound ICG can pass through fenestration of the choriocapillaris, because the molecular weight of ICG (775) is sufficiently small.

In the ICG angiograms of 24 out of 30 eyes with the fluorescein dye leakage, a bright fluorescent spot, which corresponded to the leaking spot seen in the fluorescein angiograms, gradually appeared during 1 to 3 minutes after injection of the ICG dye, and ICG dye diffused from the bright spot to the

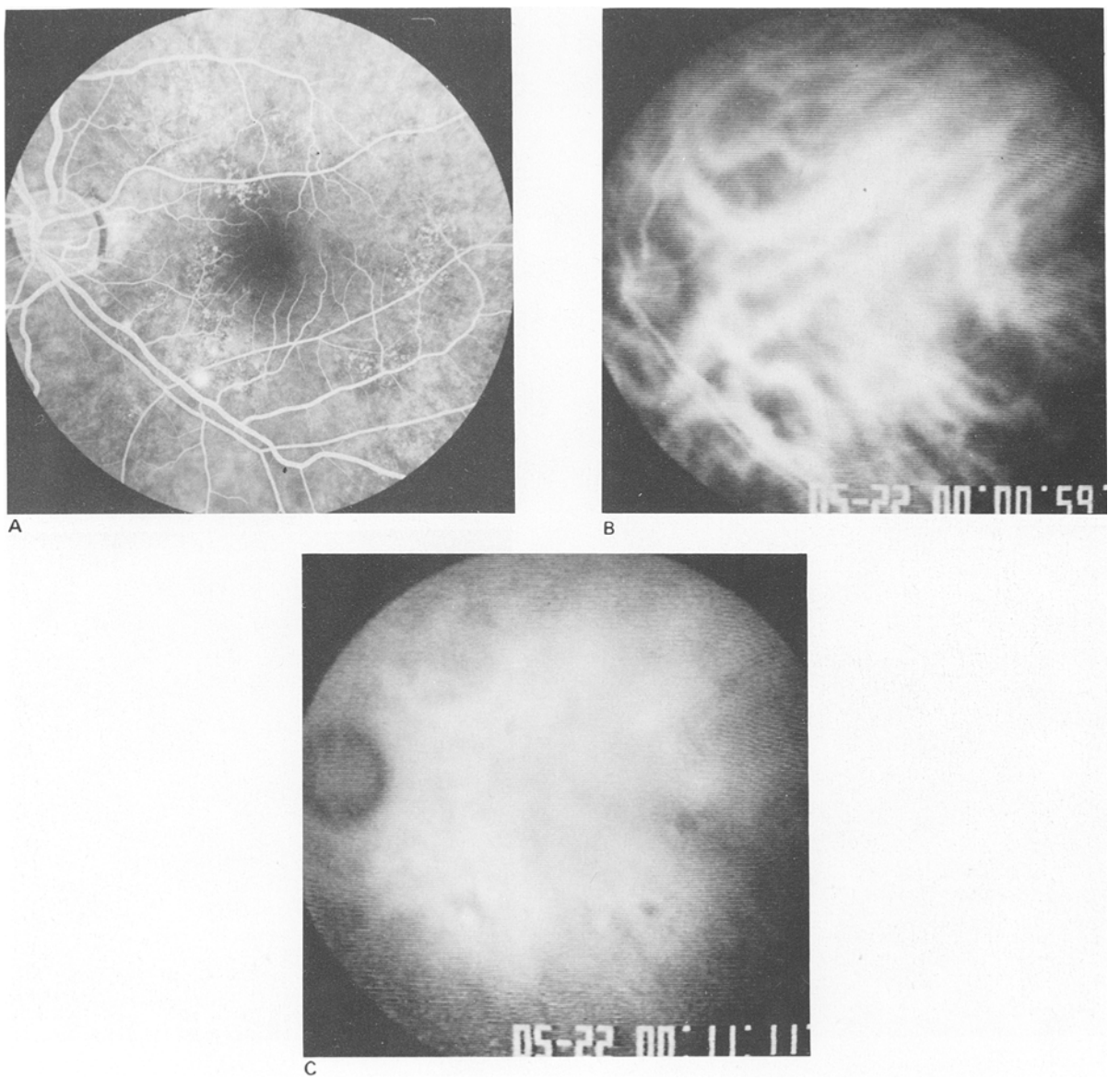

Fig. 2. The left eye of a 47-year-old male with central serous chorioretinopathy. (a). The fluorescein angiogram shows a leaking spot and spotty hyperfluorescense in the macula. (b). In the retinal venous phase of ICG angiopraghy, there are neither abnormal choroidal fluorescence nor window defect in the pigment epithelium. (c). In the late ICG angiogram, a regional hyperfluorescent area was clearly visible closed to and surrounding the leaking spot.

surrounding area. In the late ICG angiograms, accumulation of ICG dye, showing up as a hyperfluorescent area, was observed (Fig. 1). The extent, shape and size of the hyperfluorescent area corresponded to the pooling of fluorescein dye in the subretinal space. This would suggest that ICG dye, the greater part of which is bound to protein, can leak through a defect in the pigment epithelium. In 6 cases, we failed to find dye leakage in the ICG angiograms. The reason might be that the spatial separation of the video system is worse than that of the infrared black and white film, or that the background choroidal fluorescence disturbs the fluorescence from the subretinal space. However, we as-

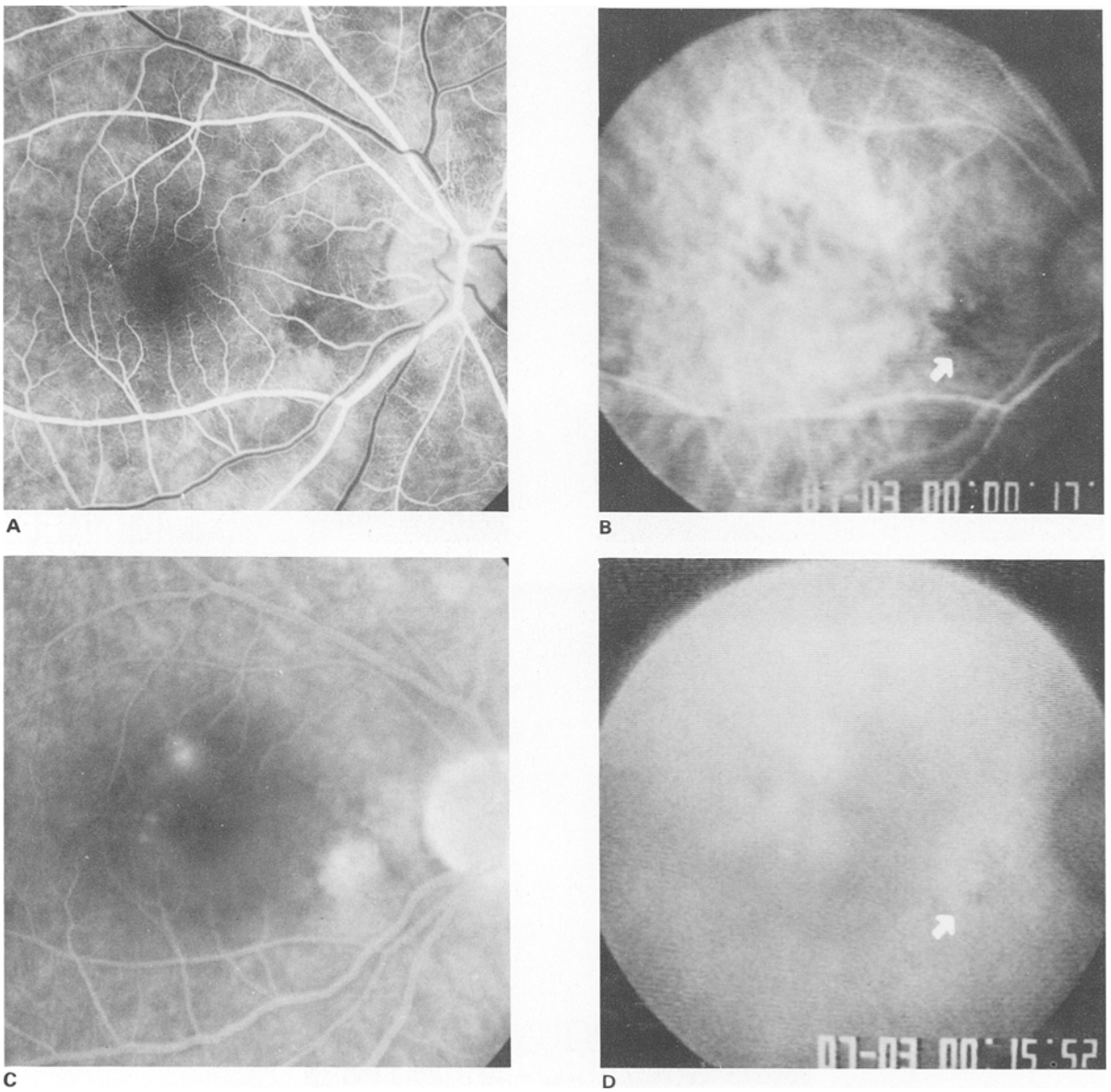

Fig. 3. The right eye of a 45-year-old male with central serous chorioretinopathy. (a). No specific pattern of delayed choroidal filling is demonstrated in the early venous phase of fluorescein angiography. (b). A localized and delayed choroidal filling pattern is observed temporal to the papilla (arrow) in the retinal venous phase of ICG angiography. (c). Diffuse fluorescein dye leakage is shown in the region which cerresponds to the hypofluorescence in (b). (d). In the late ICG angiogram, the hyperfluorescent region becomes obscure but still remains (arrow).

sume that the severity of the disturbance of the pigment epithelial barrier function determines whether ICG dye leaks through the defect of the pigment epithelium. Further investigation will be necessary to determine if the ICG fluorescence is caused by free or bound ICG.

2. Hyperfluorescent area

In 21 out of 30 cases with fluorescein dye leakage, the fluorescein angiograms showed window defects of the pigment epithelium through spotty, hyperfluorescent areas existing close to the leaking point. We found the hyperfluorescent area in ICG angiograms of those 21 eyes. In the early ICG angiograms of those cases it was difficult to see any hyperfluorescent area. During the course of angiography, however, hyperfluorescent regions gradually appeared close to, or surrounding the leaking point, and became prominent in the late angiograms. The shape of the hyperfluorescent region did not correspond to the choroidal vascular architecture existing under that area, and no anomalies (eg. choroidal hemangioma) in the choroidal vasculature were found under it (Fig. 2). Therefore, we assume that the hyperfluorescent region indicates a pigment epithalial degeneration. Because ICG angiography is able to show areas of disturbed pigment epithelium larger than in case of fluorescein angiography, it has an advantage over fluorescein angiography.

The reason why the pigment epithelial disturbance occurs in the macula is still not known. However, we assume that choroidal hypoxia in localized areas must have been one of the causes of the pigment epithelial damage. In 5 out of a total of 21 eyes with hyperfluorescent area the delayed choroidal filling pattern was found on ICG angiograms although this was absent in fluorescein angiograms. In those ICG angiograms, a localized hypofluorescent area was clearly presen during the early phase, and was clearly demarcated until the middle phase, in which the large retinal veins start to fill. In the late phase the hyperfluorescent area became obscure compared with that of previous phase but still remained (Fig. 3). There were no lesions in the hyperfluorescent area, which could explain the hypofluorescence through absorption. Therefore, the hypofluorescence area, which could explain the hypofluorescence through absorption. Theerefore, the hypofluorescence would be caused by reduced emission. We may consider this hypofluorescence to result from choroidal circulatory insufficiency. The extent of the hypofluorescent regions corresponded approximately with mottled hyperfluorescent area seen in the fluorescein angiograms.

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