Electrophysiology of the retinal pigment epithelium in central serous chorioretinopathy

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Abstract. The pathophysiology of central serous chorioretinopathy is incompletely understood but appears to involve the retinal pigment epithelium. We recorded consecutively the fast oscillation, hyperosmolarity response, acetazolamide response, and light peak from four patients with active central serous chorioretinopathy and three normal subjects to determine whether the affected eyes showed any electrophysical abnormalities. We found essentially no differences in any of the four responses between the active and the inactive eyes of the patients or between patients and normal subjects. Whatever retinal pigment epithelial dysfunction exists in central serous chorioretinopathy is unassociated with clinically evident changes in these retinal pigment epithelial electrophysiologic responses.

Abbreviations: CSC-central serous chorioretinopathy.

Introduction

The pathophysiology of central serous chorioretinopathy (CSC) is incompletely understood, but there is evidence that it may involve a diffuse abnormality of fluid transport across the retinal pigment epithelium (RPE), an abnormality that may itself be secondary in many instances to underlying choroidal disease [1-5]. The resultant inability of the RPE to absorb fluid efficiently allows fluid to accumulate in the subretinal space if there is a site of leakage.

This study was conducted to see whether patients with active CSC demonstrated any coexistent abnormality in the electrical responses of the RPE, which might help to diagnose or classify the disease [4]. The nonphotic drug-induced electrical responses of the RPE [5] to acetazolamide and hyperosmolarity were of particular interest insofar as they may give information about the behavior of the RPE membrane independent of light and retinal activity. We used a recently developed clinical protocol for recording both photic (fast oscillation and light peak) and nonphotic (acetazolamide and hyperosmolarity) RPE responses in a single recording session [6]. These responses are all generated across the basal membrane of the RPE, but they involve different
ionic mechanisms that might relate selectively to the pigment epitheliopathy in CSC [7–9].

Subjects and methods

Recordings were elicited from both eyes of three normal subjects (age range, 25–51 years; two men and one woman) and four patients with active CSC (age range, 27–39 years; three women and one man). Best corrected visual acuity in the normal group ranged from 20/15 to 20/20, and no funduscopic abnormalities were present. Best corrected visual acuity in the eyes with CSC ranged from 20/25 to 20/50; visual acuity in all of the unaffected fellow eyes was 20/20. Visual symptoms included blurred vision, metamorphopsia, dyschromatopsia, micropsia, and paracentral scotoma. Three of the patients were in good general health but reported a recent increase in emotional or physical stress; the fourth patient was in remission from chronic myelogenous leukemia, having undergone bone marrow transplantation 2 months previously. She was normotensive with stable laboratory counts (hematocrit, 31.0%–32.8%, platelets, 48,000–62,000/mm³; white blood cells, 3,400–5,200/mm³; glucose, 145 mg/dl; blood urea nitrogen, 18/dl and creatinine, 1.0 mg/dl. Her medications included nifedipine (90 mg daily), cyclosporine (350 mg twice daily), prednisone (20 mg twice daily), norethindrone acetate (20 mg daily), and trimethoprim-sulfamethoxazole (1 double strength tablet twice daily).

All of the patients with CSC had an elevated serous retinal detachment involving the macula (Fig. 1A); fluorescein angiography confirmed the presence of an active leak in every case (Fig. 1B). Three of the patients also showed RPE defects in the unaffected eye, suggestive of previous or inactive CSC. The electrophysiologic recordings were performed from 1½ to about 9 weeks after the onset of symptoms, but all of the patients were still symptomatic and had ophthalmoscopically visible detachments.

These studies were approved by our institutional Human Studies Committee, and informed consent was obtained before each recording session. The protocol excluded subjects who were pregnant or had cardiopulmonary disease.

Our recording conditions and method have been described previously [6]. With the pupils undilated, the fast oscillation was elicited first, followed sequentially by the hyperosmolarity response for 25–30 min, the acetazolamide response for 25–30 min, and the light response. The fast oscillation was expressed as a peak/trough electro-oculogram (EOG) amplitude ratio. The magnitude of the nonphotic responses was quantified by means of the formula $(V_o - V_{\text{min}})/V_o \times 100$, where $V_o$ represents the baseline EOG amplitude in the dark and $V_{\text{min}}$ the nadir after intravenous injection of 12.5 g