We recorded the FMERGs elicited by a 15° stimulus spot with duration of 100 ms preoperatively and at 1 week and 1, 3, and 5 months postoperatively (Fig. 2A). The amplitudes of the a and b waves were reduced with longer implicit times in the affected eye than in the unaffected eye. There was no change in their amplitudes at 1 week after treatment; however, 1 month after PDT, the descending limb of the b wave did not decrease to the baseline but remained elevated above the baseline (arrow). At 3 months after PDT, the FMERG showed a depolarizing pattern, and at 5 months, the ERG waveform resembled that recorded pre-PDT. The right BCVA recovered to 0.2 with resolution of the blurred vision and central scotoma.

Preoperative optical coherence tomography showed polypoidal structures accompanied by a serous retinal detachment (Fig. 2B). After PDT, these lesions gradually disappeared. At 3 months after treatment, IA demonstrated closure of the polypoidal lesion without choroidal hypoperfusion corresponding to the treated area (Fig. 1D).

Comments

Our results showed that the amplitudes of the FMERGs were not reduced soon after the PDT as reported by Ishikawa et al.[2] They reported that the FMERG amplitudes were transiently reduced, and the reduction was correlated with the degree of choroidal hypoperfusion. The absence of a reduction in the FMERG amplitudes in our patient was probably because we reduced the total energy of the PDT to one-half of the standard level, which prevented the choroidal hypoperfusion following PDT in our case.

The depolarizing pattern of the cone ERG is an unusual waveform, and has been recorded in a case of unilateral cone dystrophy.[3] Kondo and Miyake[4] reported a case of macular degeneration with a depolarizing pattern of the FMERG. In both these cases of a depolarizing ERG pattern, the abnormalities were mainly in the outer retina. Experimentally, ERGs with a depolarizing pattern have been recorded after the signals from the cones to the OFF-bipolar cells are blocked by glutamate analogs, *cis*-2,3-piperidine dicarboxylic acid (PDA), or kynurenic acid.[1] It is reported that the synapses between the photoreceptors and bipolar cells are altered in experimentally induced retinal detachment in cats.[5] In cases where the time course of the recovery of the synaptic alternation is different between the ON- and OFF-pathways, an imbalance between the ON- and OFF-responses of the FMERGs may occur with a resolution of the exudative retinal detachment of the macula. This should then result in a depolarizing ERG pattern. Alternatively, the PDT may have affected the balance between the ON- and OFF-responses of the macula. However, the waveform of the FMERG was unchanged 1 week after PDT, and the depolarizing pattern developed gradually postoperatively, suggesting that the PDT most likely did not alter the signal transmission of the middle retina, and thus did not contribute to the development of the depolarizing pattern of the FMERGs.

Keywords: AMD, depolarizing pattern, focal ERG, PCV, PDT

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Vascular Compressive Optic Neuropathy Caused by Hypertensive Intracranial Ophthalmic Artery

Owing to advances in magnetic resonance imaging (MRI) techniques such as spoiled gradient acquisition steady state (SPGR) and fast imaging employed steady state, it is now possible to show that vascular compression of the cranial nerve is responsible for some neuro-ophthalmologic diseases for which the etiology is still unknown.[1,2] Optic nerve compression by the intracranial carotid artery itself is a well-known problem.[3] However, there are only a few reports of optic nerve compression by the ophthalmic artery, except for carotid–ophthalmic aneurysms. We report a patient with vascular compressive optic neuropathy caused by a hypertensive intracranial ophthalmic artery.

Case Report

A 40-year-old man who is a pilot reported acute visual loss OD during night flight. He had systemic hypertension (average, 160/90 mmHg) not controlled by medication and impending central retinal vein occlusion (CRVO) OS. On examination, his best-corrected visual acuity was 0.4 OD and 1.0 OS. Funduscopic examinations showed normal OD and