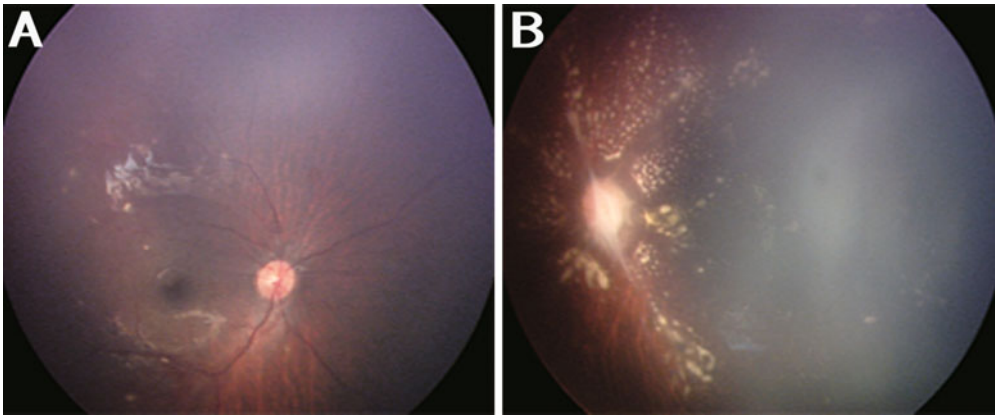

Figure 2A, B. Fundus photographs of the **A** right and **B** left eyes 1 month after administration of fluconazole. **A** The size of the white lesions had decreased significantly. **B** Retinal reattachment with hard exudates, white vessels, and optic disc pallor were observed.

in a patient with *Candida* vaginitis and onychomycosis; both patients were treated with antibiotics.[2,3] The current patient had no systemic abnormalities but evidently had endogenous *Candida* endophthalmitis because PCR analysis detected sufficient quantities of *Candida* DNA in the vitreous and the cerebrospinal fluid to diagnose the infection. FA findings of abnormal vasculature in the peripheral retina are usually seen in eyes with retinopathy of prematurity or familial exudative vitreoretinopathy, which prompted us to suspect that the *Candida* infection in the present case was congenital. Generally, a congenital *Candida* infection occurs by vertical transmission through the uterus or vagina and is associated with systemic involvement, including dermatitis, meningitis, anomaly of the brain, and oral mucositis.[4] However, the patient was delivered by Caesarean section, and no signs of *Candida* infection were detected in the mother. Thus, acquired *Candida* infection was the most likely diagnosis in the present case. Intravenous antibiotics delivered 2 weeks before the onset of bilateral endophthalmitis likely caused iatrogenic *Candida* infection because of inadvertent manipulation. Possible insufficient growth of the retinal vasculature might have facilitated the proliferation of *Candida* in the patient's retina.

To diagnose and treat such a difficult case, broad-range PCR for the 18S ribosomal RNA sequence is a good screening tool.[5] Moreover, real-time PCR can examine the quantity of the pathogen and determine its relation to the endophthalmitis. Early treatment of infectious endophthalmitis is essential in infants, in whom vision develops rapidly. Thus, a broad-range, real-time PCR system using ocular samples is useful when the patient has uveitis or endophthalmitis of unknown origin.

Keywords: *Candida* chorioretinitis, *Candida* infection, polymerase chain reaction

Makiko Ito[1], Tadashi Yokoi[1], Sunao Sugita[2], Naomi Shinohara[3], Sachiko Nishina[1], and Noriyuki Azuma[1]
[1] Department of Ophthalmology, National Center for Child Health and Development, Tokyo, Japan; [2] Department of Ophthalmology and Visual Science, Tokyo Medical and Dental University Graduate School of Medicine and Dental Sciences, Tokyo, Japan; [3] Department of Pediatrics, Tokyo Dental College Ichikawa General Hospital, Chiba, Japan

Received: January 14, 2010 / Accepted: June 8, 2010
Correspondence to: Noriyuki Azuma, Department of Ophthalmology, National Center for Child Health and Development, 2-10-1 Okura, Setagaya-ku, Tokyo 157-8535, Japan
e-mail: azuma-n@ncchd.go.jp

DOI 10.1007/s10384-010-0871-4

References

1. Palmer EA. Endogenous Candida endophthalmitis in infants. Am J Ophthalmol 1980;89:388–395.
2. Williams MA, McMullan R, Hedderwick S, Mulholland DA, Best RM. Diagnosis and treatment of endogenous fungal endophthalmitis. Ophthalmologica 2006;220:134–136.
3. Kostick DA, Foster RE, Lowder CY, Meyers SM, McHenry MC. Endogenous endophthalmitis caused by *Candida albicans* in a healthy woman. Am J Ophthalmol 1992;113:593–595.
4. Michael JM. Fungal infections. In: Remington JS, Klein JO, editors. Infectious diseases of the fetus and newborn infant. Philadelphia: WB Saunders; 1983. p. 466–469.
5. Jaeger EEM, Carroll NM, Choudhury S, et al. Rapid detection and identification of *Candida*, *Aspergillus*, and *Fusarium* species in ocular samples using nested PCR. J Clin Microbiol 2000;38:2902–2908.

Choroidal Neovascularization in a Child Following Laser Pointer-Induced Macular Injury

Laser pointer-induced macular injury is characterized by a decrease in visual acuity and metamorphopsia.[1] High-energy lasers can cause chorioretinal damage, which can lead to choroidal neovascularization (CNV) in animals.[2] Case reports of the development of a CNV following laser-induced macular injury have also been published.[3,4] We report the case of a child with a CNV that developed following a macular injury caused by repeated exposure to a green laser pointer. The prevalence of CNV in children is low, but it is still an important cause of visual impairment.[5] To the best of our knowledge, this is the first report of a child developing CNV following a macular injury caused by exposure to a green laser pointer.

Jpn J Ophthalmol
Vol 54: 622–637, 2010

Case Report

An 11-year-old boy with decreased visual acuity in the right eye was referred to our hospital for consultation. The parents reported that the child stared directly at a commonly used green laser pointer. He did not understand the cautionary statement, and from the age of 2 to 3 years stared at it with his dominant right eye every day for more than 10 s at a time, as if it were a toy, at a distance of 30 cm. Although he had a congenital hearing loss and mental retardation, his visual functions developed normally up to the time of the injury. When he was 7 years old, his visual acuity was 1.0 OU, after correction of bilateral astigmatism, –4.0 diopters.

When he was 11 years old, his best-corrected visual acuity (BCVA) was 0.2 OD and 1.0 OS. No abnormalities were found in the anterior segment of either eye. Ophthalmoscopy identified a yellow exudate-like lesion or fibrous tissue surrounded by subretinal hemorrhage in the right macula (Fig. 1A). The left eye was completely normal. Two years later when he was 13 years of age, the fundus showed a yellow fibrous lesion in the right macula (Fig. 1B) that demonstrated leakage on fluorescein angiography (Fig. 1C, D). A STRATUS optical coherence tomography image showed a highly reflective mass that extended from the outer retinal layer through the retinal pigment epithelium and Bruch's membrane into the choroidal tissue of the right macula (Fig. 1E). The left eye was normal. Investigations for ocular infectious diseases did not reveal any disease.

We elected to follow the patient with careful observation and not to perform invasive therapy because of his age and mental condition. He is now 14 years old, and his BCVA and the appearance of the fibrous tissue are unchanged.

Comment

By the results of the ophthalmological examinations and the history of events, we diagnosed the patient as having laser pointer-induced macular injury. An accurate diagnosis of laser pointer-induced macular injury did not come easily, because it was difficult to interpret the complaints of the

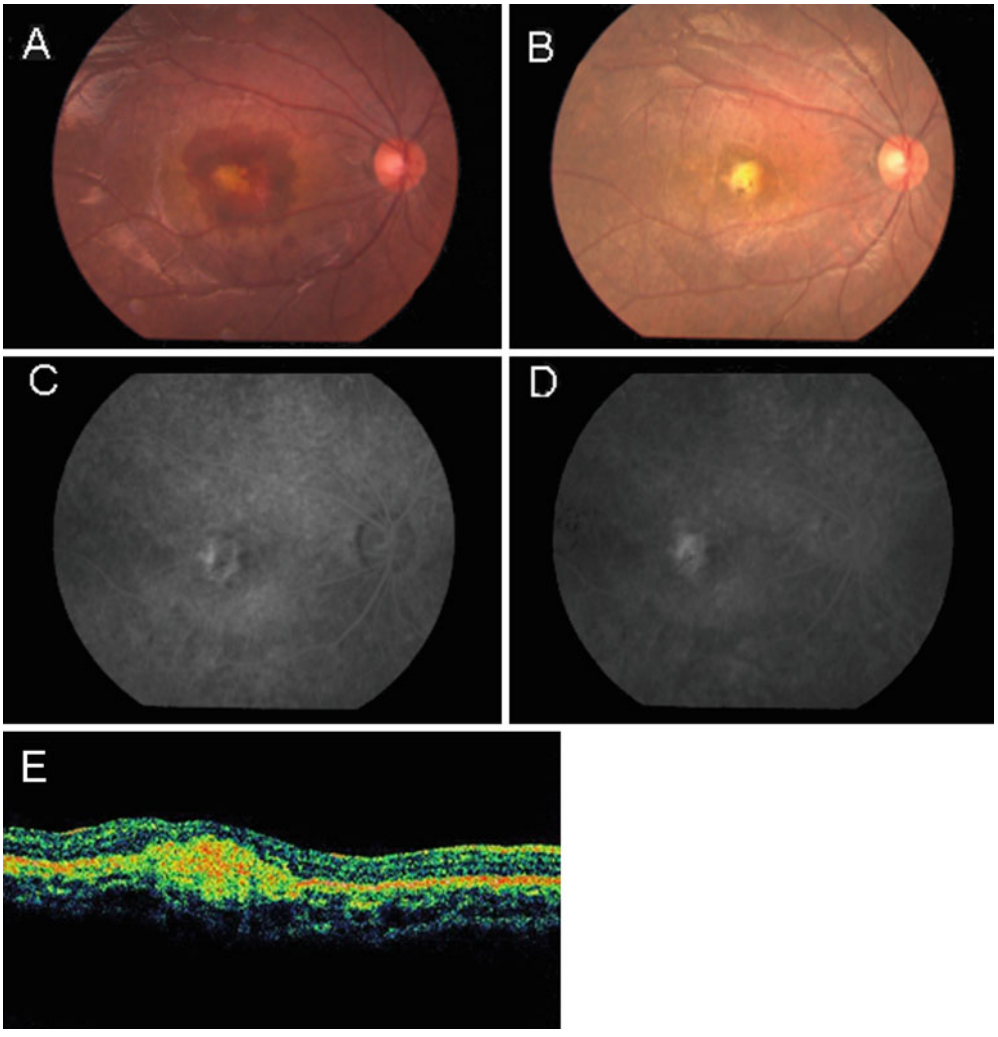

Figure 1A–E. Fundus images of the current case**. A** When the patient was 11 years old, the fundus of his right eye showed a yellow lesion resembling exudates or fibrous tissue surrounded by subretinal hemorrhage in the macula. **B** When he was 13 years old, the fundus showed a yellow lesion resembling fibrous tissue. **C, D** Fluorescein angiography showed leakage with fibrous tissue remaining in the right macula (**C** early phase; **D** late phase). **E** Optical coherence tomography demonstrated a highly reflective mass extending not only to the outer retinal layer and retinal pigment epithelium but also to the Bruch's membrane and choroidal tissue.

patient, and the time of the injury and initial examination were prolonged.

There is a correlation between the energy of a laser and the degree of chorioretinal damage it can cause. The output power of handheld laser pointers is commonly from 1 to 5 mW. Mild thermal retinal injuries might be caused by a 5-mW laser, if it is stared at for more than 10 to 20 s;[1] this suggests that the chorioretinal damage in our patient, which probably induced the CNV, was caused by the frequent and repeated exposure to the low-energy laser beam.

The prognosis of this patient is unclear, because the interval between the first laser exposure and the development of the CNV was long in comparison to previously reported cases. The patient is being carefully followed for the possible reactivation of the CNV.

Keywords: choroidal neovascularization, laser pointer, macular injury

Kaoru Fujinami[1,2], Tadashi Yokoi[2], Miina Hiraoka[2], Sachiko Nishina[2], and Noriyuki Azuma[2]
[1] National Institute of Sensory Organs, National Hospital Organization, Tokyo Medical Center, Tokyo, Japan; [2] Department of Ophthalmology, National Center for Child Health and Development, Tokyo, Japan

Received: February 25, 2010 / Accepted: June 29, 2010
Correspondence to: Noriyuki Azuma, Department of Ophthalmology, National Center for Child Health and Development, 2-10-1 Okura, Setagaya-ku, Tokyo 157-8535, Japan
e-mail: azuma-n@ncchd.go.jp

DOI 10.1007/s10384-010-0876-z

References

1. Barkana Y, Belkin M. Laser eye injuries. Surv Ophthalmol 2000;44:459–478.
2. Shen WY, Lee SY, Yeo I, et al. Predilection of the macular region to high incidence of choroidal neovascularization after intense laser photocoagulation in the monkey. Arch Ophthalmol 2004;122:353–360.
3. Roider J, Buesgen P, Hoerauf H, Schmidt-Erfurth U, Laqua H, Birngruber R. Macular injury by a military range finder. Retina 1999;19:531–535.
4. Sun Z, Wen F, Li X, Wu D. Early subfoveal choroidal neovascularization secondary to an accidental stage laser injury. Graefes Arch Clin Exp Ophthalmol 2006;244:888–890.
5. Sivaprasad S, Moore AT. Choroidal neovascularisation in children. Br J Ophthalmol 2008;92:451–454.

Spontaneous Closure of a Stage 2 Macular Hole Without Detachment of the Posterior Hyaloid

Stage 2 macular holes occasionally close spontaneously after hyaloid membranes with pseudo-opercula become separated from the surface of the retina. However, we observed spontaneous closure of a stage 2 macular hole without release of the vitreofoveal traction. This case was documented by means of optical coherence tomography (OCT).

Case Report

A 52-year-old man complained of metamorphopsia in his left eye. He was referred to a nearby clinic, and a macular hole in the left eye was diagnosed. He did not report any trauma. About 2 weeks later, he came to our clinic at Akita University Hospital. His best-corrected visual acuity was 20/16 in the right eye and 20/160 in the left. Slit-lamp examination showed no remarkable findings. Biomicroscopic examination did not reveal posterior vitreous detachment (PVD). OCT (Zeiss OCT3; Zeiss-Humphrey Systems, Dublin, CA, USA) showed the presence of a stage 2 macular hole with perifoveal cyst formation (Fig. 1A). The hole measured 336 μm in diameter and was partially covered with a retinal flap. A posterior hyaloid was present and adhered to the edge of the hole (Fig. 1B). Around the macular hole, there was a shallow PVD. He did not have any other ocular diseases such as diabetic retinopathy, retinal vein occlusion, macular telangiectasia, or uveitis.

Four months later, the patient's best-corrected visual acuity had improved to 20/30. OCT seemed to show the presence of an outer retinal bridge over the macular hole (Fig. 1C, D), indicating a spontaneous macular hole closure in process. The perifoveal cysts were no longer apparent. However, the patient still felt metamorphopsia in his left eye, and the posterior hyaloid remained adhered to the retinal flap (Fig. 1E). To release this adhesion and close the hole completely, we performed a pars plana vitrectomy. During the operation, we used triamcinolone acetonide to visualize the vitreous and observed the hyaloid attachment to the macular hole. We did not peel the internal limiting membrane because the macular hole was already bridged and we thought that releasing the attachment was sufficient to close the hole completely. At the end of the operation, air tamponade was used.

Seven days after the surgery, OCT showed the presence of a thick bridge and a well-defined retinal hyporeflective space interrupting the inner high-reflective layer (Fig. 2A). Seven months after surgery, the patient's best-corrected visual acuity remained at 20/30. The hyporeflective space had become quite small. Two and a half years after surgery, his best-corrected visual acuity was 20/20. OCT (Spectralis HRA+OCT; Heidelberg Engineering, Heidelberg, Germany) showed that the hyporeflective space no longer existed and the foveal morphology had progressed to almost normal (Fig. 2B).

Comments

As the use of OCT has become more common, many cases of spontaneous closure of macular holes have been reported.[1–4] Four explanations have been proposed for the