Verteporfin Photodynamic Therapy of Choroidal Neovascularization Secondary to Ocular Toxoplasmosis

Choroidal neovascularization (CNV) can arise secondary to the retinochoroiditis and macular scarring from ocular toxoplasmosis.1,2 Treatment of CNV due to toxoplasmosis can include corticosteroids, cryotherapy, laser photocoagulation, submacular surgery, and verteporfin photodynamic therapy (PDT).1,4 We describe 2 cases of CNV secondary to toxoplasmosis treated successfully with PDT.

Report of Cases. Case 1. A 20-year-old man with a diagnosis of congenital ocular toxoplasmosis with bilateral macular scars sought care because of a 9-month history of decreasing vision and metamorphopsia in the right eye. Visual acuity was 1/200 OD. Fundus examination results revealed a subfoveal choroidal scar with surrounding subretinal hemorrhage and exudate (Figure 1A). Fluorescein angiographic images identified central leakage from the CNV with surrounding blocked fluorescence corresponding to the hemorrhage. The eye was treated with PDT and the greatest linear dimension of the treatment spot included all of the hemorrhage.

One week posttreatment, visual acuity improved to 20/200 OD. Six weeks posttreatment, a flat scar was present in the central macula with resolution of the hemorrhage and exudate. Six months posttreatment, visual acuity improved to 20/60 OD and has remained stable for more than 2 years (Figure 1D-F).

Case 2. A 15-year-old boy with a diagnosis of bilateral macular scars secondary to congenital ocular toxoplasmosis reported decreasing vision in his left eye. On examina-
tion, visual acuity was 20/100 OS. Fundus examination results revealed a macular scar with adjacent fibrosis, subretinal fluid, and hemorrhage (Figure 2A). Fluorescein angiographic images identified leakage from the CNV and blocked fluorescence corresponding to the hemorrhage (Figure 2B and C). Optical coherence tomographic images revealed increased retinal thickening and a highly reflective layer under the retina consistent with CNV (Figure 2D). The patient underwent PDT.

One month following treatment, visual acuity improved to 20/80 OS. Fundus examination results showed a flat chorioretinal scar and complete resolution of subretinal fluid and blood. The patient’s visual acuity and clinical appearance remained stable through 18 months of follow-up (Figure 2E-H).

Comment. Compared with other treatments, PDT has the theoretical advantage of selectively occluding the CNV, even in the presence of small amounts of overlying blood, without causing significant damage to the unaffected retina and choroid. Thus, PDT has the potential for vision improvement, especially in younger patients as shown in our cases. Furthermore, only a single treatment was necessary in both of these cases, unlike the 3 to 4 treatments routinely needed during the first year in patients with neovascular age-related macular degeneration. The decreased need for retreatment with PDT in these toxoplasmosis cases is similar to the retreatment rate observed in younger patients with postinflammatory CNV. While improvement could have occurred without PDT, these lesions were not in the process of resolving as suggested by the duration and progression of vision loss and the presence of new hemorrhages. Photodynamic therapy was most likely responsible for the observed improvements owing to the temporal relationship between treatment and the resolution of the CNV. To our knowledge, this is the second report of CNV secondary to toxoplasmosis treated with PDT; the first report demonstrating a sustained visual acuity benefit from a single treatment with PDT, and the first successful use of PDT to treat a predominantly hemorrhagic lesion.

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Visual Impairment and Deafness in Young Children: Consider the Diagnosis of Congenital Infection With Cytomegalovirus, Even Years After Birth

Infection with cytomegalovirus (CMV) is the most common congenital infection in humans and affects 1% to 2% of all neonates. However, only 10% show symptoms at birth, varying from slight developmental complaints to serious neurologic, auditory, or ophthalmologic abnormalities. Although the other 90% of infected neonates are asymptomatic at birth, symptoms of congenital CMV may not be discovered until many years later. Later in childhood, 5% to 17% of these children will develop ocular, audiologic, neurologic, or developmental sequelae. Symptoms of congenital CMV can be indistinct and the diagnosis may be overlooked for years, leading to developmental disorders without adequate treatment. This report demonstrates how visual impairment detected many years after birth may lead to the diagnosis of congenital infection with CMV.

Report of a Case. A 3-year-old boy was suspected of decreased vision and sent to our clinic. At the age of 7 months the patient was diagnosed with deafness of unknown origin for which he received a right-sided cochlear implant. Ocular examination using the Kay picture test revealed decreased visual acuity OD (2/5) and normal vision in the left eye. Funduscoppy of the right eye revealed an atrophic chorioretinal scar of the macula (Figure), whereas the left eye was unremarkable.

Serologic tests were performed, the results of which showed both the mother and son to be positive for anti–CMV IgG antibodies. To differentiate between a congenitally and postnatally acquired CMV infection, the Guthrie card (containing neonatal dried blood drawn within 7 days after birth and used for the screening of inborn errors of metabolism) was retrieved (with the parents’ permission) from the regional screening center. The dried blood was eluted in isotonic sodium chloride solution and tested for CMV DNA (real-time polymerase chain reaction [PCR], Taqman ABI7700; sensitivity, 5 genome copies/100 µL; specificity >99.9%) and anti–CMV IgM antibodies (Enzygnost; Dade Behring; sensitivity on serum, 95%; specificity, >99%). Both tests’ results were positive, establishing the diagnosis of congenital infection with CMV.

Comment. Infants with asymptomatic congenital CMV infection are at high risk of developing visual impairment. In one study up to 22% of patients with symptomatic congenital CMV infection developed chorioretinitis or optic atrophy and in most cases bilaterally. Conversely, during a 5-year follow-up in a longitudinal study that included 445 children with congenital CMV, ocular defects were found in only 7% of children who were asymptomatic at birth. In all of the cases visual impairment was associated with optic atrophy, macular scars (chorioretinitis), or cortical damage. Although hearing impairment is the highest risk associated with asymptomatic congenital CMV infection, isolated neurodevelopmental delay and ophthalmologic lesions can also be signs of congenital CMV. One way to diagnose congenital CMV years after birth is by performing a CMV-PCR and/or anti–CMV IgM enzyme-linked immunosorbent assay on the neonatal blood stored on the Guthrie card. Note that negative results do not exclude a congenital infection; both tests have limited sensitivity when used in this setting.

In conclusion, patients with asymptomatic congenital CMV are at risk for serious sequelae that need proper care. Among children with visual or hearing impairment of unknown cause, CMV-PCR and serologic testing of neonatal blood stored on the Guthrie card enables the diagnosis of a congenital infection with CMV, even years after birth.

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