Congenital Toxoplasmosis With Unusual Retinal Findings

Toxoplasmosis is the most common cause of infectious retinitis in otherwise healthy individuals. In the United States, 70% to 80% of women at childbearing age are at risk of developing a primary infection. Infants with congenital toxoplasmosis may or may not have clinical evidence of disease. Active disease is characterized by encephalitis, lymphadenopathy, hepatitis, jaundice, rash, thrombocytopenia with petechiae, and a range of neurological manifestations that include microcephaly and seizures. A classic triad of convulsions, intracranial calcifications, and chorioretinitis has been associated with congenital infection with Toxoplasma. The presence of this triad of findings, however, is not necessary to establish the diagnosis. Congenital toxoplasmosis can be made on the basis of serologic evidence (IgG and IgM titers) and central nervous system manifestations. Chorioretinal scarring is the most common ocular manifestation of congenital toxoplasmosis, but occasionally acute chorioretinitis is seen in the early neonatal period.

We report an unusual case of congenital toxoplasmosis with panuveitis and an associated retinal fold in one eye and a total retinal detachment in the fellow eye.

Report of a Case. A 1-month-old Asian girl with a history of irritability and fever was referred for evaluation of asymmetric red reflexes. The infant’s mother had a history of fever and lymphadenopathy involving the cervical lymph nodes at the fourth gestational month, but she had not been medically evaluated. She also had a history of eating undercooked meat early in the pregnancy. There was no family history of inherited retinal diseases.

On ophthalmic examination, the infant had light response in the right eye but no light response in the left eye. The pupils were 3 mm OD with a brisk response to light and 1 mm OS with no light response. There was a left afferent pupillary defect. Anterior segment examination of the right eye revealed moderate cells with no other abnormalities. Anterior segment examination of the left eye revealed a clear cornea, a flat anterior chamber with 360° of iris neovascularization, and seclusio pupilla. Intraocular pressure was 14 mm Hg OU. Findings from the dilated fundus examination of the right eye revealed a retinal fold extending from the optic nerve through the macula to the vitreous base at the 9-o’clock position associated with mild vitri-
nystagmus and a sensory esotropia. Follow-up examination 9 months later demonstrated no active iridocyclitis, vitritis, or retinitis, but there was a stable retinal fold in the right eye.

Comment. Toxoplasmic chorioretinitis is the most common ocular manifestation of both congenital and acquired toxoplasmosis. Retinal detachment, mostly of a tractional origin, occurs in only 10% of cases of congenital toxoplasmosis.

Among the entities that were considered in the differential diagnosis of this case were viral retinides, toxocariasis, familial exudative vitreoretinopathy, Norrie disease, and retinoblastoma. A normal family medical history, negative findings for organisms on serologic testing and culturing, and the clinical picture helped to exclude these other possibilities. Although clinical manifestations are usually sufficient to diagnose both congenital and acquired toxoplasmosis, occasionally it presents a diagnostic challenge. In this case, the presence of a macular retinal fold in the absence of the typical chorioretinal lesions and the IgM titer that was initially negative for toxoplasmosis presented a diagnostic challenge. In this context of other nonocular manifestations helped us reach the proper diagnosis. The prevalence of congenital toxoplasmosis is decreasing and clinicians may be less likely to think of it as a cause of disease, especially when it is atypical in appearance. We report this case of congenital toxoplasmosis because of its atypical ocular presentation.

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The Utility of 0.5% Apraclonidine in the Diagnosis of Horner Syndrome

In 1999, Morales et al1 reported that 1.0% apraclonidine hydrochloride (Iopidine; Alcon, Ft Worth, Tex) could be used to diagnose Horner syndrome. Apraclonidine caused reversal of anisocoria (the miotic pupil with Horner syndrome became larger than the normal pupil) in all patients in their study. Apraclonidine is primarily an α2-receptor agonist, but it does have some weak α1 affinity, as evidenced by conjunctival blanching. The authors postulated that the reversal of anisocoria was due to denervation hypersensitivity of α1-receptors in the pupil dilator muscle. The purpose of our study is to determine whether 0.5% apraclonidine, which is less expensive and more readily available than the 1.0% formulation, might also be used to diagnose Horner syndrome in the same lighting conditions.

Report of Cases. Patients with known or newly diagnosed Horner syndrome in 2 of our practices (those of R.A. and K.A.F.) were invited to participate, and institutional review board approval was obtained. Cases were consecutive, and all were confirmed by pharmacologic testing. Demographic data collected included patient age, sex, etiology and duration of Horner syndrome (if known), lesion location (preganglionic or postganglionic, if known), and results of previous pharmacologic testing.