A 10 Month Old Infant with Nystagmus and Strabismus

Jedari Attya S., Maliky Z. and Daneshjou Kh.
Department of Pediatrics, Imam Khomeini Hospital,
Tehran University of Medical Science, Tehran, Iran

Abstract: This report present an infant with nystagmus, strabismus, salt and pepper and scars in fundoscopy; calcification in Brain CT scan and high titer of Anti Toxoplasmosis antibody. A 10 month old infant that referred with nystagmus, strabismus after fever which appeared five months ago. In fundoscopy of both eyes, salt and pepper and scars and in Brain CT scan multiple calcification were seen. The diagnosis of congenital Toxoplasmosis was established by positive serum Anti toxoplasma Ab (IgG) (>400). Toxoplasmosis may present with only nystagmus and strabismus and physicians should consider this infection in the differential diagnosis of a abnormal eye movement.

Key words: Nystagmus, strabismus, toxoplasmosis

INTRODUCTION

Toxoplasma gondii an obligate intracellular protozoan is acquired perorally, transplacentally or rarely parenterally by transfusion or transplantation (McLeod and Remington, 2004).

Newly infected cat and other felidae excrete infectious toxoplasma oocysts in their feces. Human infection is usually acquired by the oral route by eating undercooked or raw meat that contains cysts or by ingestion of cysts.

Congenital infection may present as a mild or severe neonatal disease with onset during 1st months of life or with sequelae or relapse at any time and the manifestations include small for gestational age, prematurity, peripheral retinal scars, persistent jaundice, chorioretinitis and cerebral calcification (McLeod and Remington, 2004).

More than half of these are considered normal in the perinatal period but almost all such children will have ocular involvement later in life and retinochoroiditis is the most common ocular manifestation of congenital toxoplasmosis. Other manifestations are convulsions, hydrocephalus, microphthalmia, microcephaly, hepatosplenomegaly, nystagmus, visual impairment, strabismus, developmental delay (McLeod and Remington, 2004). Ocular involvement existed in 30% of treated children with congenital involvement (Kodjikian and Fleury, 2006).

Ocular involvement in congenital toxoplasmosis is frequently bilateral but acquired toxoplasmosis in old children and adult is generally unilateral (Rosso et al., 2005; Kenneth et al., 2004).

The second most common symptom was microphthalmia and strabismus (Vutova et al., 2002).

In two sibling bilateral macular lesions were consistent with the diagnosis of congenital toxoplasmosis, there was considerable evidence that the lesions were scars from toxoplasmic retinochoroiditis (Stern and Roman, 1978).

The aim of this study was to describe a 10 month old infant with toxoplasmosis that was presented with ocular involvement.

CASE REPORT

A 10 month old boy infant was referred to our department owing of strabismus and nystagmus.

The infant had fever in 4th month of life and after 1 month Epiphora, abnormal eye movement, strabismus, nystagmus, abnormal lip movement (probably convulsions) is started. In fundoscopy salt and pepper in right eye and several scars in left eye were seen and the patient referred to me. In examination, the infant had strabismus in left eye and nystagmus in both eyes. Jaundice, abnormal lip movement (probably convulsion), epiphora and fever in 4th month were seen in the history, but the infant does not have development delay.

CSF analysis was normal, toxoplasma Ab (IgM) = 5.9 and toxoplasma Ab (IgG)>400.

EEG had abnormal wave and in Brain CT scan multiple calcification in periventricular (bilaterally) and in subcortical area were seen.
According to the results, toxoplasmosis was diagnosed and treatment was begun with pyrimethamine, Sulfadiazin, folinic acid, prednisolone and pyrimethamine for convulsions. He responded dramatically to the therapy and after 1 month, the eye lesions were inactivated and there was no progression in eye involvement.

**DISCUSSION**

Toxoplasmosis can involve multiple organ systems such as the nervous system, ear, lung, heart, skeletal muscle, LAP, skin, liver, kidney, bone, endocrine and any part of eye may be involved either unilaterally or bilaterally including macula, optic nerve and visual pathway in the brain and visual cortex and glaucoma. Extraocular muscle may also be involved.

Ocular toxoplasmosis is a recurrent and progressive disease that require multiple course of therapy. Our patient was well baby without any gross problem at birth, but in 10 month of age ocular toxoplasmosis was suggested by history of fever and epiphora that progress to nyctagmus and strabismus in 5 month, finding in brain CT scan (calcification) and positive serology were seen at 10 month And soon the clinical response to treatment were seen.

The diagnosis of toxoplasmosis can be established by isolation of *T. gondii* from blood and etc. or by culture or serology such as IgM-ELISA, IgA ELISA. PCR of amniotic fluid is the procedure of choice for establishing the diagnosis of congenital toxoplasmosis and sensitivity and specificity of this test are approximately 95% in 18 week of gestation. IgG antibody titers of 1/4 to 1/64 are usual in older children with active toxoplasmic chorioretinitis. When the retinal lesions are characteristic and serologic tests are positive the diagnosis is likely (McLeod and Remington, 2004).

At birth when a diagnosis of congenital toxoplasmosis is suspected, the following studies should be performed: general, ophthalmologic and neurologic examination, head CT scan, attempt to isolate *T. gondii* from placenta and infant’s leukocytes from umbilical cord blood anduffy coat, measurement of serum toxoplasma-specific IgG, IgM, IgA and IgE antibodies and the total amount of IgG and IgM in serum, lumbar puncture including analysis of CSF for cell, glucose, protein, toxoplasma-specific IgG and IgM antibodies and total amount of IgG and PCR and inoculation into mice. Presence of toxoplasma-specific IgM in CSF or local production of specific IgG in CSF establishes the diagnosis of congenital cerebral toxoplasma infection (McLeod and Remington, 2004).

**CONCLUSION**

Toxoplasmosis may present with several clinical feature but in congenital toxoplasmosis almost all such children will have ocular involvement later in life. The disease is in differential diagnosis of other infection such as *Treponema pallidum*, *Mycobacterium tuberculosis*, *Vasculitis* or *Cytomegalovirus*. All infected newborns should be treated, whether or not they have clinical manifestations of infection. In infant with congenital infection treatment may be effective in interrupting acute disease that damages vital organs. Infant should be treated for 1 year.

Ocular involvement existed in 30% of treated children with congenital involvement (Kodjikian et al., 2006).

Overall ocular prognosis is satisfactory when congenital damage is recognized early and treated appropriately (Rosso et al., 2005).

**REFERENCES**


