

Case Report

Congenital Toxoplasmosis presenting as multiple ring enhancing lesions in brain on neuroimaging

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ABSTRACT

We report a case of congenital toxoplasmosis in a male child who presented at 1 month 21 days of life. He had a past history of meningitis on third day of life which resolved with intravenous antibiotics. This time, the child presented with progressive enlargement of head and CSF (Cerebrospinal fluid) showed elevated protein levels with cellular reaction and CT scan showed dilated ventricular system with aqueductal obstruction and multiple ring-enhancing lesions in bifrontal and bioccipital regions with a right porencephalic cyst. Repeat CSF after 14 days of intravenous antibiotics showed elevated protein with no cellular reaction. Fundus examination was normal. Toxoplasma blood serology (IgG and IgM) in baby and mother were positive. The child received treatment with Pyrimethamine, Clindamycin, Folinic acid and Prednisolone. CSF parameters got normalized and multiple ring-enhancing lesions disappeared on repeat CT scan. He underwent a Ventriculo-Peritoneal shunt surgery for the hydrocephalus and was discharged in a stable condition.

Keywords: Congenital toxoplasmosis, Intracranial calcifications, Ring-enhancing lesions

INTRODUCTION

Toxoplasmosis is one of the most common parasitic infections in humans, and infects up to one-third of the world's population.¹ Congenital toxoplasmosis has a wide spectrum of clinical manifestations, but it is subclinical in approximately 80 % of infected newborns.² Transmission to the fetus usually follows acquisition of primary infection by an immunologically normal pregnant women during gestation.³ There is an increasing risk of transplacental transmission and decreasing severity of infection with increasing gestational age.^{4,5} The classic triad consists of chorioretinitis, intracranial calcifications and hydrocephalus and is found in less than 10% of infected infants.⁶ In the present case, the child had hydrocephalus and multiple ring-enhancing lesions which subsequently improved with treatment.

CASE REPORT

A 1 month 21 days old child presented with complaint of progressive increase in size of head over past 15 days. The child was admitted on third day of life for meningitis and was treated with intravenous antibiotics. On admission the patient was stable, the child was playful and accepting feeds and had gained weight compared to birth weight. Systemic examination was unremarkable. The only positive finding was the presence of a large head (head circumference was 40.8cm) with bulging anterior fontanelle. CSF was suggestive of protein 198 mg/dL with 120 cells. The CT scan showed dilated ventricular system with aqueductal obstruction, multiple ring enhancing lesions in bifrontal and bioccipital regions with a right porencephalic cyst. After 14 days of intravenous antibiotics CSF examination was repeated,

which showed no cellular reaction but was showing persistently elevated protein (168 mg/dL). VDRL, HBsAg and tuberculosis work up were all negative. Fundus examination was normal. Mother's and baby's HIV were non-reactive. Toxoplasma IgG and IgM antibody titres were positive in both baby and mother.

A diagnosis of congenital Toxoplasmosis was made and Pyrimethamine, Clindamycin, Folinic acid and Prednisolone were started. After just 7 days of this regimen the CSF parameters were all within the range of normalcy (Protein-75 mg/dL). A repeat CT scan was done at this stage which revealed tiny calcific foci in periventricular region of right frontal and left parieto-occipital area, porencephalic cyst in right frontal lobe, with moderate to severe obstructive hydrocephalus with periventricular edema. The multiple ring enhancing lesions had disappeared. The patient was then operated, and a ventriculo-peritoneal shunt was placed. The patient recovered, and the head circumference showed decreasing head size. The patient was discharged in a stable condition having achieved social smile and partial head holding at 12 weeks of age

DISCUSSION

Toxoplasmosis gondii is a protozoan with worldwide distribution.⁷ Congenital toxoplasmosis is the most serious manifestation of the disease, resulting from transplacental infection of the fetus with *Toxoplasma gondii* during pregnancy.^{7,8} Infection with *T. gondii* occurs primarily from ingestion of inadequately cooked meat containing cysts or from ingestion of oocysts derived from food or water contaminated with cat feces.

Congenital transmission occurs when immunologically susceptible mother acquires infection during gestation. The general risk of transmission of acute infection from mother to foetus is estimated to be 40%; however, the actual risk and severity of congenital infection vary with gestational age.⁹ Of untreated maternal infections acquired in the first trimester, approximately 17% of fetus is infected, usually with severe disease. Of untreated maternal infection acquired during 3rd trimester, approximately 65% of fetuses are infected usually with mild disease at birth.¹⁰ Transmission is less frequent when infection is acquired before 10th week of gestation.

In normal children, infection may be asymptomatic. Infections at the early gestational age may cause anemia, jaundice, chorioretinitis, seizure and hydrocephalus of fetus. Late sequels of congenital toxoplasmosis are sensorineural deafness, micropcephaly, mental retardation, visual defect, and developmental delay. The characteristic triad of presentation is chorioretinitis, hydrocephalus and intracranial calcifications.¹¹ A CSF protein level of >1g/dl is characteristic of severe disease and is usually accompanied by hydrocephalus. The most sensitive and specific method for diagnosis is isolation of the organism in tissue cell lines or in animals; however, it

is cumbersome and time taking procedure. Serologic tests used widely are the indirect immunofluorescent assay, an ELISA, and the Sabin Feldman dye test. CT scans typically reveal hydrocephalus with asymmetric dilatation of ventricles. Intracranial calcifications especially in the peri-ventricular region are characteristic of the condition. Ring enhancing lesions have been described in adult toxoplasmosis, toxoplasmic abscess and toxoplasmosis in HIV patients.¹²

Table 1: Treatment of congenital toxoplasmosis.¹³

| Medication | Dosage | Duration of therapy |
|---------------|--|---|
| Pyrimethamine | Loading dose: 2 mg/kg per day for 2 days than 1 mg/kg per day for 2 or 6 months, then this dose every Monday, Wednesday, Friday. | 1 year |
| Sulfadiazine | 100 mg/kg per day in two divided doses. | 1 year |
| Leucovorin | 10 mg three times weekly | During and for 1 week after pyrimethamine therapy |

The specific postnatal therapy is based on pyrimethamine and sulfadiazine. The simultaneous use of both drugs is 8-fold more active than either pyrimethamine or sulfadiazine alone and has been the "gold standard."¹⁴ This regimen, described more accurately in the Table 1, has been associated with resolution of signs of active congenital toxoplasmosis, usually within the first weeks after initiation of therapy.¹⁵

CONCLUSION

We must, however, keep in mind the toxic effects of pyrimethamine, hence all patients treated with this drug should have a monitoring of blood counts twice every week and moreover should receive folinic acid (in the form of leucovorin calcium). Therapy should be continued for at least one year of age.

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