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Case Report

Congenital Toxoplasmosis - Suspicion Raised on Basis of Finding on Antenatal Scan

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Abstract

Infection with the protozoan parasite *Toxoplasma gondii* occurs globally and it usually manifests no symptoms. However, a primary infection in a pregnant woman may spread to the fetus through the placenta [1]. An estimated 190,100 cases of congenital toxoplasmosis are reported worldwide each year, with an incidence of 1.5 occurrences per 1000 live births [2]. Chorioretinitis, cerebral calcifications, and hydrocephalus make up the classic triad of neonatal congenital toxoplasmosis, however they are rarely visible at birth [3]. Most newborns with congenital toxoplasmosis don't exhibit any symptoms. Here, we discuss a case in which prenatal and postnatal scans revealed complaints of hydrocephalus. The majority of the symptoms of this condition were surprisingly discovered in our instance. The maternal serology report, which was positive for *Toxoplasma* IgM, provided additional assistance with the diagnosis.

Keywords: *Congenital Toxoplasmosis; Antenatal Scan; Toxoplasma gondii; Hydrocephalus; Toxoplasma IgM*

Introduction

Infection with the protozoan parasite *Toxoplasma gondii* occurs globally and it usually manifests no symptoms. However, a primary infection in a pregnant woman may spread to the fetus through the placenta [1]. An estimated 190,100 cases of congenital toxoplasmosis are reported worldwide each year, with an incidence of 1.5 occurrences per 1000 live births [2]. The incidence and severity of congenital infections depend on when the mother becomes infected during pregnancy [4]. Although congenitally infected newborns are usually asymptomatic at birth, they are at risk for complications such as blindness later in life. When congenital infection is evident, it can manifests as retinochoroiditis, cerebral calcifications, hydrocephalus, neurocognitive impairment [1]. Early diagnosis and treatment of acute *T. gondii* infection during pregnancy can decrease the risk of vertical transmission and risk of severe disease in newborn [5]. Furthermore, timely intervention can effectively lessen the severe long-term consequences in newborns with congenital toxoplasmosis [6].

Here we present a case of congenital toxoplasmosis suspected in neonate with presenting complaint of Hydrocephalus in antenatal scans.

Case Report

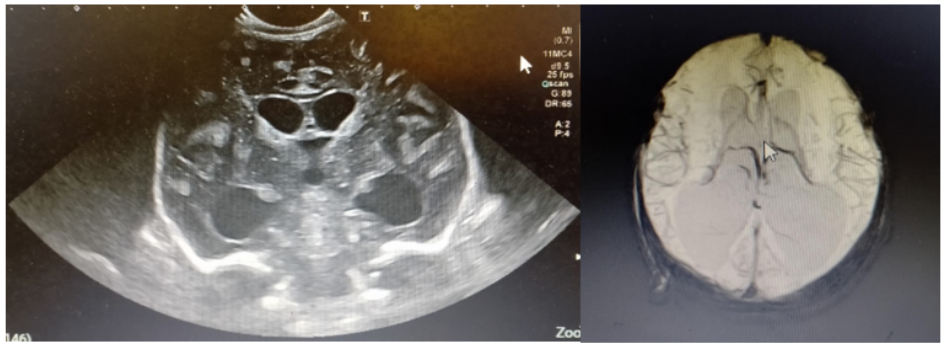
A male newborn was admitted in our hospital on 4th day of life via emergency department with the presenting complaints of hydrocephalus in antenatal and postnatal scans with V/H ratio of 33% and yellowish discoloration of sclera. He was born outside our hospital at 36+5 weeks of gestation via emergency lower segment caesarian section due to non progress of labor, to a mother who was the gravida 2nd at that time with previous history of miscarriage at 8 weeks. She had antenatal history of leaking for 10 hours with no other remarkable finding except her antenatal scans which has revealed bilateral mild hydrocephalus in neonate. Her blood group was B positive. There was no any history of contacts with animals or consumption of raw meat, no history of fever, rash or enlarged lymph nodes during pregnancy. Baby was born with good APGARS. He was admitted in hospital at 12 hours of life and was managed as RDS. He had issues of jitteriness or fits according to attendant.

Upon admission, findings of physical examination of the newborn to our department were as follows: Vitals were normal, in anthropometry- weight, 3.3 kg (between 90th and 97th centiles); head circumference, 33 cm (below 3rd centile); length, 40 cm (at 10th centile); active and alert; slight yellow coloration of the sclera; and anterior fontanelle wide and soft, and red reflex was positive. The newborn's abdomen was soft, not distended with Hepatosplenomegaly. The remaining examination results were unremarkable.

The child was started antibiotics and laboratory investigations were conducted. (Figure 1a) Ultrasound head revealed dilatation of both lateral ventricles and third ventricle with Levene index of 15 mm on Right side and 14.5 mm on left side. There were multifocal areas of meningeal thickening (due to which antibiotics were switched to meningitic dose) with multiple innumerable echogenic areas scattered in the bilateral cerebral hemispheres. Left caudate lobe was echogenic. Findings were suggestive congenital TORCH infection with non-communicating hydrocephalus. The white blood cell count was 14.5×10^9/l (neutrophils, 33.8%; lymphocytes, 44.6%), the hemoglobin concentration was 17.1 g/dl, the platelet count was 87×10^9/l, and the C-reactive protein concentration was 11.4 mg/l. The total serum bilirubin concentration was 7 mg/dl, and the indirect bilirubin concentration was 5.6 mg/dl. Due to the findings of hydrocephalus and thrombocytopenia, our initial impression was of congenital TORCH infection. Multidisciplinary approach was adopted involving neonatologists, pediatric neurologists and pediatric infectious disease specialists. (Figure 1b) MRI brain revealed multifocal ring-enhancing lesions in both supratentorial and infratentorial brain with communicating hydrocephalus and meningitis favoring cerebral infection (TORCH) some of the lesions were also in favor of calcific deposits. CSF studies were sent which revealed CSF glucose less than two-thirds of that of blood; proteins were more than 600 mg/dl and leucocytes were 0.093×10^9/l with 80% lymphocytes and 20% neutrophils. Eye exam revealed bilateral, multifocal, widespread retinitis. Neonate developed seizures during the stay and was managed with anticonvulsants. TORCH profile of both mother and baby were sent along with CSF bio fire. BERA was planned but attendants got discharged due to financial reasons with advise of follow up. Labs that were chased after discharge revealed negative TORCH IgMs for baby but positive IgM (3.54) for toxoplasmosis of mother. CSF and blood culture were showing no growth. CSF bio fire results were also negative (which tested for *E. coli, Hemophilus influenza, Listeria monocytogenes, Neisseria meningitidis, Streptococcus agalactiae* and *Pneumoniae, Cryptococcus neoformans, Enterovirus,* Herpes Simplex Virus 1, 2 and 6, Human Parechovirus, Varicella Zoster virus and Cytomegalovirus). Our final impression is of congenital toxoplasmosis due to the combination of mother's serology (Positive IgM for toxoplasmosis) with intracranial calcifications, hydrocephalus, chorioretinitis and unexplained mononuclear CSF pleocytosis or elevated CSF protein. Serology may be negative initially in infants due to delayed antibody production in some cases in which testing should be repeated.

Discussion

Congenital toxoplasmosis affects children of pregnant women who acquires new *Toxoplasma gondii* infection or experiences reactivation if the mother's immune system is impaired during pregnancy [6]. Toxoplasmosis in pregnant women is acquired by eating undercooked meat contaminated with tissue cysts or by consuming oocysts excreted by cats through contaminated food, vegetables, or water.



a= Postnatal ultrasound scan of the baby

b= SWI sequence of MRI (lesions showing faint susceptibility artefact indicating calcific deposits

Figure 1

The chance of maternal illness spreading to the fetus depends on weeks of gestation at that time; it is minimal in the first trimester and may rise up to 90% in the last days of gestation. However, when infection happens earlier in pregnancy, it causes more severe disease in fetus [1]. Chorioretinitis, cerebral calcifications, and hydrocephalus make up the classic triad of neonatal congenital toxoplasmosis, however they are rarely visible at birth [3]. Congenital toxoplasmosis in infants is often asymptomatic. In addition to the typical trio, those with symptoms may also exhibit aberrant CSF results, rashes, micro- or macrocephaly, seizures, sensorineural hearing loss, altered motor tone, delayed milestones, jaundice, hepatosplenomegaly, anemia, and thrombocytopenia.

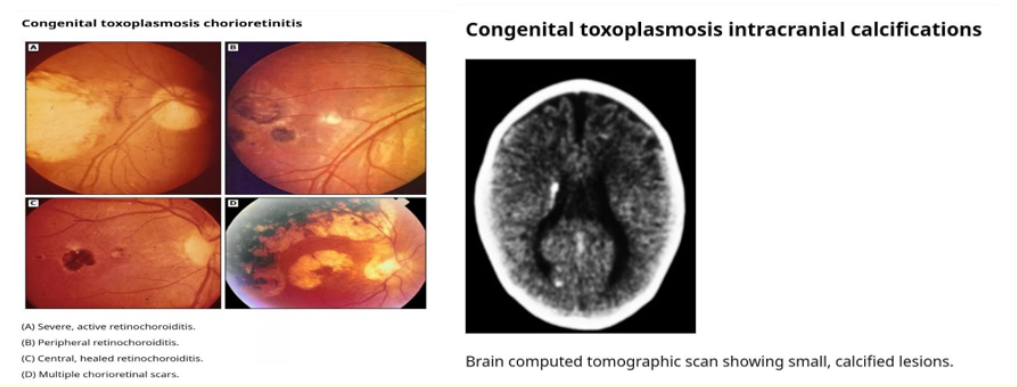


Figure 2

Positive results from the PCR test for *Toxoplasma* in the amniotic fluid and ultrasound imaging can prove fetal infection [7]. Evaluation of a newborn suspected of having congenital toxoplasmosis should include review of maternal history including risk factors and available serology, complete examination of newborn, Complete blood counts, lumbar puncture, ophthalmologic and audiologic evaluation and neuroimaging to look for abovementioned findings along with *Toxoplasma gondii* serologic testing of both mother and newborn (IgG, IgM and IgA). Based on maternal exposure, testing can also be done for cytomegalovirus and other congenital illnesses (since co-infection with toxoplasmosis and CMV can happen). When serologic testing findings are inconclusive, PCR testing might aid in making the diagnosis. CSF, peripheral blood, urine, vitreous fluid (ocular *Toxoplasma*), Broncho alveolar lavage fluid, cord blood, and placenta are all acceptable sample sources for PCR. Interpretation of these tests for diagnosis is mentioned in table 1.

Confirmed Congenital Toxoplasmosis	Presumed congenital Toxoplasmosis	Excluded congenital Toxoplasmosis
IgM + or IgA+ with IgG+ in newborn or compatible mother serology	Characteristic clinical findings	Negative IgG, IgM, and IgA.
Positive CSF PCR with symptoms or maternal history of acute infection	Positive IgG, but negative IgM and IgA	Negative IgM and IgA with positive IgG titer that declines over time
Increase in IgG titers or Increased titers in comparison to mother		
Positive IgG beyond 12 months of age		

Table 1

In some equivocal cases repeat testing is also required (Table 2).

Scenario	Cause	When to repeat
Asymptomatic infant, positive initial IgM and/or IgA	Small leaks through placenta	10days after birth 7 days after last transfusion
Positive IgG but not IgM or IgA	Past or currently infected mother	Serial testing during 1st year of life
Negative initial results but strong clinical suspicion	Delayed antibody production, antenatal treatment	2 - 4 weeks after birth, Then every 4 weeks till 3 months of age

Table 2

The initiation of therapy follows diagnosis. Prior to starting therapy, several additional tests are necessary, including baseline CBCs, LFTs, serum creatinine, and urine analysis (for dosage adjustments), as well as G6PD testing (medication with sulfadiazine may cause hemolysis in children with G6PD deficiency). Studies have demonstrated that antenatal therapy reduces the risk of severe infection along with frequency of early sequelae, that are found at birth as well as late sequelae. Mothers with acute primary toxoplasmosis are prescribed spiramycin in the first trimester of pregnancy and it should be continued till delivery. After fetal infection has been confirmed, pyrimethamine, sulfadiazine, and folinic acid combinations are given [8]. Due to its teratogenicity, this triple regimen should not be given during the first 14 weeks of pregnancy [9]. In the postnatal period, it is often treated with a regimen that includes pyrimethamine, sulfa-

diazine, and folinic acid, typically for one year. In certain situations, like in case when vision is threatened by active chorioretinitis or when CSF protein levels are above 1 g/dl, steroids may be administered until these problems are resolved.

To identify adverse reactions of medication, it is necessary to monitor changes in complete blood counts, renal function tests, and liver function tests. Long-term effects should be clinically followed up, including by routine ophthalmologic evaluation. A multidisciplinary approach including a neonatologist, pediatric infectious disease specialist, pediatric neurologist, ophthalmologist, pediatric ENT specialist and radiologist is recommended.

Untreated symptomatic infections have a poorer prognosis because they can cause major neurologic impairment, visual problems, and hearing loss. Early and prolonged treatment does, however, help to lessen the risk, although treated newborns still run the chance of developing long-term complications, which in part relies on the disease's severity at the time of diagnosis. In order to ensure compliance and ongoing care of the infant, it is crucial to properly advise the parents on each element. This will help to reduce the likelihood of unfavorable results.

Conclusion

High index of Suspicion for congenital infections on basis of their prominent features can help to facilitate early diagnosis and tailor appropriate diagnostic evaluation in a cost effective manner. So that, specific treatment can be started as start as soon as possible to prevent long term sequelae. Antenatal testing should be considered in pregnant women with suggestive risk factors, clinical or radiologic findings to improve the outcomes. Multidisciplinary approach should be followed. Proper counselling of parents should be done regarding each aspect to ensure compliance and ongoing care of newborn.

Ethical Approval and Consent to Participate and Publish

Informed consent was obtained from parents of patient for using the case details for publication in journal as well for other study purpose.

Availability of Data and Material

Case details are not publicly available because data is patient's medical records but are available from the corresponding author upon reasonable request.

Conflicts of Interests

None.

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