CONGENITAL TOXOPLASMOSIS PRESENTING WITH DEEP ANEMIA: A CASE REPORT

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ABSTRACT

Toxoplasmosis is a serious infectious disease caused by the obligate intracellular protozoan Toxoplasma gondi, which leads to serious sequela and/or death fetuses. This is a report of a Turkish girl neonate in the first day with congenital toxoplasmosis who presented with only anemia and low hematocrit as a sole physical and laboratory finding respectively. As far as we now this is the first report of such a case in the literature. Even though there are no clinical signs, congenital toxoplasmosis requires treatment. In this way any other neurological sequela could be prevented. The patient was treated with primetamin, sulfadiazine, and leukovarin. She was followed for 12 months without blindness, deafness and any neurological sequela.

Key words: Congenital toxoplasmosis, anemia

INTRODUCTION

Toxoplasmosis is a serious infectious disease caused by the obligate intracellular protozoan Toxoplasma gondi, which induces sequel and/or death in newborns and fetuses. The disease is not easily recognized because the mother presents with silent form and only 15% of infected babies present clinical signs at birth. Auditory and visual disabilities and mental retardation as a sequela of disease can be observed in these infected babies at childhood and adolescence 1. That is why, it is important to diagnose and treat the infected babies at birth.

Although, the traditional clinical signs of the congenital toxoplasmosis are the triad of hydrocephalus, chorioretinitis, and intracranial calcifications, there are congenital toxoplasmosis cases in the literature accompanied with anemia 2. The rarely seen case with a diagnosis of sole anemia in newborn period is presented here.
CASE REPORT

A 33-year-old mother with G1P1 in the 36th gestational week gave birth to a baby weighing 2700 g (50th percentile), 47 cm in length (50th percentile), with a head diameter of 32 cm (50th percentile). A Turkish girl neonate in the first day with congenital toxoplasmosis is presented with only anemia and low hematocrit as a sole physical and laboratory finding. Blood analyses were: Hgb: 7.2 g/dL, Hct: 21%, WBC: 14800/mm³, Platelet: 256000/mm³.

Mother’s medical history revealed neither placenta previa nor abruptio placenta including any other acute bleeding. The regular examinations she had during the pregnancy showed that she did not have any specific disease and was not on any medication. The baby’s laboratory analysis for bleeding diathesis were normal (Protrombine time: 11.8 seconds, INR: 0.848, aPTT: 26.8 seconds, bleeding time: 3 minutes, fibrinogen: 200 mg/dL), fecal occult blood results were negative, and total urine microscopic analysis showed 2-3 erythrocytes. Immediately after delivery, the baby intramuscularly received 1 mg vitamin K.

There was not a mismatch since both mother and the baby had the same blood group type (O Rh +). The baby’s direct Coombs test was negative, reticulocyte was 2%, and in the peripheral blood smear erythrocytes were normochromic and normocytic, and glucose-6-phosphate dehydrogenase was 5.6 U/gHb (4.6-13.5), and the HPLC electrophoresis were Hbf: 79.5%, Hba: 16.1%, HbA2: 4.4%. Sepsis markers were negative (CRP: 4.99 mg/L [0-6], and negative hemoculture results).

When evaluated for in utero infections, like VDRL, CMV IgM., Rubella IgM, HSV Type 1, Type 2 IgM, Parvovirus B19 IgM with enzyme immune assay (EIA), the results were negative for all. Toxoplasma IgM was 0.7 index (normal:<0.55 ), IgG >300 IU/ml (normal:< 4 ), and thus we ran serological analyses on the mother’s blood. The mother’s results were: toxoplasma IgM 0.7, IgG >300. The Turkish Perinatology Committee does not offer routine toxoplasma serologic analyses during pregnancy, that’s why we did not have the chance to obtain these results before delivery. The mother’s medical history did not include any serious disease or cat contact in or out of the house during pregnancy. The only possible indicator of cat contact was that the mothers’ meals contained a large serving of not well cooked meat in the last two months of pregnancy.

The physical exam did not show chorioretinitis, one of the classic signs of congenital toxoplasmosis. Autoacoustic emission test results were bilaterally positive. All of the following analyses were negative: calcification in craniography, hydrocephalus or bleeding in cranial USG, organomegalies in abdominal USG. In cerebrospinal fluid there were 2-3 erythrocytes, 7-8 leukocytes, protein: 46 mg/dL, glucose: 43 mg/dl (at the same time, blood glucose: 80).

The diagnosis of toxoplasmosis with EIA-specific IgM, IgG, IgA tests might not show the early or late time period of disease, so we used a specific IgG avidity test in this case to show the primary infection time. IgG avidity test is based on the adhesion power of multivalent IgG to toxoplasma antigens. This adhesion power is lower in the early period and grows in the course of weeks and months. Proteolytic reactants, like urea, are used to separate the antigen-antibody complex. Avidity results were determined using a titration curve that showed the ratio of separated and intact antigen-antibody complex. High IgG avidity results show
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that there is no infection in the first 12 weeks of pregnancy. Conversely, low IgG avidity could show acute infection.

In this case, the avidity test shows an acute infection (IgG: 0.172 T.V. (<0.200 T.V.). Therefore, this case was diagnosed as congenital toxoplasmosis and we administered one dose of 10 mg/kg erythrocyte suspension. Congenital toxoplasmosis requires treatment even if there are no clinical signs; so we administered Primetamin 2 mg/kg/day for two days, and then lowered the dose to 1 mg/kg/day; Sulfadiazin 100 mg/kg/day twice a day; Leukovarin 5 mg three times a week. In this case, we observed the patient for 12 months and found that there was no blindness or deafness. The planned total therapy period was 1 year.

DISCUSSION

Congenital infection may present as a mild or severe neonatal disease or with sequelae or relapse of a previously undiagnosed and untreated infection later in infancy or even later in life. There is a wide variety of manifestations of congenital infection, ranging from hydrops fetalis and perinatal death to small size for gestational age, prematurity, peripheral retinal scars, persistent jaundice, mild thrombocytopenia, cerebrospinal fluid pleocytosis, and the characteristic triad of chorioretinitis, hydrocephalus, and cerebral calcifications. Autopsy of neonatal hemolytic anemia shows congenital toxoplasmosis in most cases. However, anemia is rarely reported in live cases of congenital toxoplasmosis. The majority of cases did not report anemia but rather physical signs, such as organomegalies and direct hyperbilirubinemia.

In this case there are no clinical or physical signs, only paleness. According to laboratory results, hemoglobin levels were low, toxoplasmic IgM was positive and avidity test was low, but there is nothing more to explain the anemia etiology. Chorioretinitis does not occur at delivery, but almost all such children develop ocular involvement later in life if they are not treated during infancy. In this case, we observed the patient for 12 months and found that there was no blindness or deafness.

CONCLUSION

If paleness is the only abnormality in a careful physical exam and if laboratory results show only anemia, we have to suspect the rare anemia etiology, congenital toxoplasmosis. Thus, we can prevent the pathologic early stages of serious sequel, such as blindness and deafness, caused by congenital toxoplasmosis.

CONSENT

Written informed consent was obtained from the patient for publication of this manuscript and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.
COMPETING INTERESTS

The authors declare that they have no competing interests.

REFERENCES