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JCIMCR OPEN ACCESS Journal of Clinical Images and Medical Case Reports

ISSN 2766-7820

Case Report

Open Access, Volume 3

Congenital toxoplasmosis: About a serious and preventable case

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Received: Jul 25, 2022

Accepted: Aug 12, 2022

Published: Aug 19, 2022

Archived: www.jcimcr.org

DOI: www.doi.org/10.52768/2766-7820/2007

Keywords: Toxoplasma gondii; Congenital infection; Obstetric ultrasound.

Abbreviations: CMV: Cytomegalovirus; HVS2: Herpes Simplex Virus type 2; HVS1: Herpes Simplex Virus type 1; VDRL: Venereal Disease Research Laboratory; PCR: Polymerase Chain Reaction.

Abstract

Background: Congenital toxoplasmosis results from vertical transmission of the Toxoplasma gondii parasite from mother to fetus after a primary maternal infection. The risk of fetal contamination is higher the later the term of maternal infection. Conversely, the risk of severe fetal infection decreases with term; third trimester infections are most often asymptomatic at birth.

Despite the mandatory use of toxoplasma serology during pregnancy monitoring, some patients may be lost to follow-up. This can have serious consequences in the case of primary infection in early pregnancy, which may lead to a fatal embryo-fetopathy as in the case presented here.

Case presentation: We hereby report the uncommon case of a 29-year-old woman referred to our facility for premature rupture of membranes at six months of pregnancy. Obstetrical ultrasound on admission revealed moderate hydramnios, fetal brain lesions consistent with cerebral abscesses and fetal hydrops with pleurisy, prefrontal oedema and fetal ascites. The results of the biological tests requested were positive for IgG and IgM Elisa Toxoplasma. The evolution was marked the next day by the rapid onset of uterine contractions with premature delivery. The newborn, weighing 1280g, presented major respiratory distress and died at 17 minutes of life. The parents refused an autopsy, but the diagnosis of congenital toxoplasmosis was confirmed by a positive PCR of toxoplasma gondii in a placental sample and in a cord blood sample, in a context of primary infection.

Conclusions: Congenital toxoplasmosis is more serious when contracted in early pregnancy. A serological test for anti-toxoplasmic IgG is recommended in early pregnancy to establish the precise gestational age of seroconversion. It is imperative to remind practitioners of the dramatic complications of congenital toxoplasmosis as in our case so that they can ensure an effective prevention.

Background

Congenital toxoplasmosis results from vertical transmission of the Toxoplasma gondii parasite from mother to fetus after a primary maternal infection. The number of new cases of congenital toxoplasmosis is estimated at 700 per year in France, i.e., a rate of 1 per 1,000 live births [1]. The risk of fetal contamination is higher the later the term of maternal infection. Conversely, the risk of severe fetal infection decreases with term; third trimester infections are most often asymptomatic at birth.

Despite the mandatory use of toxoplasma serology during pregnancy monitoring, some patients may be lost to follow-up. This can have serious consequences in the case of primary infection in early pregnancy, which may lead to a fatal embryofetopathy as in the case presented here.

Case presentation

We hereby report the uncommon case of a 29-year-old female farmer living in a rural area who has never attended school. She has no significant pathological history, gravida 4 para 4 whose first three unattended pregnancies resulted in three healthy full-term vaginal births. For the current pregnancy, she had only one prenatal visit at three months of pregnancy which showed an evolving intrauterine pregnancy. There was no folic acid supplementation in early pregnancy and no prenatal check-ups, including serologies. Her already known blood type was Rhesus A positive. An obstetrical ultrasound was requested at the first consultation but was not performedsince she has stopped her follow-up.

She was referred to our facility for premature rupture of membranes at six months of pregnancy. She complained of amniotic fluid loss. On entry clinical examination, the patient was in good general condition, apyretic, with no oedema, flexible uterus, uterine height of 29 cm and present fetal heart sounds. On vaginal examination, the cervix was long, closed and posterior with clear amniotic fluid leakage. Obstetrical ultrasound on admission showed a single fetal pregnancy estimated at 26 weeks of amenorrhea according to the cranial perimeter with a fetus in cephalic presentation and positive fetal heart activity. Morphological study revealed cerebral lesions consistent with cerebral abscesses (Figure 1), moderate hydramnios and hydrops fetalis with pleurisy, prefrontal oedema and fetal ascites (Figures 1 and 2). The diagnosis was premature rupture of membranes at approximately 26 weeks of amenorrhea with hydrops fetalis and fetal cerebral abscess.

The results of the biological tests requested were as follows: Elisa IgG CMV: absent; Elisa IgM CMV: absent; Elisa IgG Toxoplasma: 288 IU/l (normal value below 150 IU/l; Elisa IgM-Toxoplasma: 39 (normal value sup. 15 IU/l); Elisa Rubella IgG: absent; HVS2: absent; Elisa HVS1: present; Elisa listeria M: absent; Elisa VDRL: Negative; Fasting blood glucose: 0.82 g/dl; Proteinuria: negative. In view of these results, we concluded to a fetal toxoplasmic infection complicated by hydrops fetalis with cerebral abscess.

The evolution was marked the next day by the rapid onset of uterine contractions with the threat of premature delivery. Before the case could be discussed between the pediatric and obstetric teams, the patient, who was multiparous, had already given birth to a polymal formed child with an Apgar score of 2/3/5 at respectively 1, 5 and 10 minutes of life. The newborn, weighing 1280 g, had a major respiratory distress and died at 17 minutes of life.

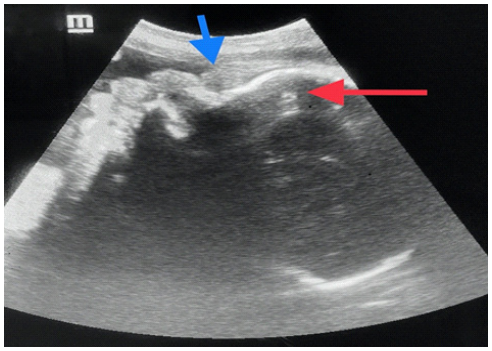

Figure 1: Ultrasound image of a sagittal slice of the fetal brain showing:
blue arrow: A prefrontal oedema.
red arrow: A cerebral lesion compatible with a cerebral abcess.

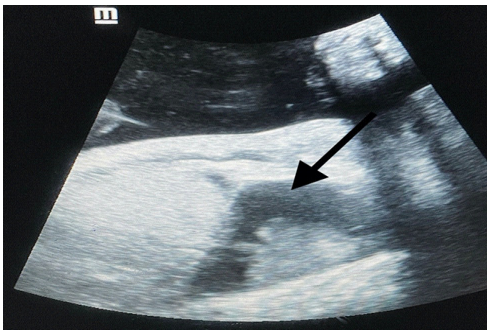

Figure 2: Ultrasound image of parasagittal fetal trunk showing fetal pleurisy (black arrow).

Macroscopic examination of the placenta revealed placentomegaly. The parents refused an autopsy but the diagnosis of congenital toxoplasmosis was confirmed by positive PCR of toxoplasma gondii in a placental sample and in a cord blood sample, in a context of primary infection.

Discussion

Seroprevalence rates of toxoplasmosis among pregnant women differ widely from a country to another depending primarily on their eating habits, their culture and climatic variations [2,3]. The highest incidence rates occur in South America, in some Middle Eastern and low-income countries [4]. In Morocco, the regional rates of Toxoplasmosis seropositivity effloresce 50% among pregnant women [5]. There is no maternofetal transmission when the infection occurs prior to conception unless the immune system is compromised. However, when the infection is acquired during pregnancy, the severity of fetal infection depends on the gestational age at seroconversion [6].

Pregnant women come across Toxoplasma Gondii usually from ingestion of one of its three forms: Oocysts, bradyzoites or tachyzoites. Fetoplacental infection resulting in morphological abnormalities decreases with increasing gestational age whereas the risk of vertical transmission increases as the pregnancy progresses [7,8]. Acute maternal infection is mostly insidious in pregnant women. At worst, it manifests itself as a viral illness (fatigue, headache, lymphadenopathy and myalgia) [9].

The most detrimental fetal implications of congenital toxoplasmosis occur early in pregnancy. First trimester seroconversion often leads to severe fetal sequelae with potential long-term debilitating effects or even miscarriage and stillbirth. Up to 90% of newborns with congenital toxoplasmosis are asymptomatic or phenotypically normal [10]. Otherwise, it may present itself in various forms including jaundice, fever, microcephaly, maculopapular rash, thrombocytopenia and seldom generalized lymphadenopathy. The classic characteristic triad consisting of chorioretinitis, hydrocephalus and cerebral calcifications, most commonly identifies the presence of active congenital disease [11].

Maternal diagnosis relies on documentation of recent seroconversion. Determiningthe exact timing of the infection is of paramount importance. The presence of elevated levels of Toxoplasma-specific IgG antibodies indicates infection occurred at some point. They remain detectable for life. IgM antibodies may appear within the first week of infection and normally decline within a few months [12]. However, they sometimes persist for years after the initial infection. Therefore, IgM antibodies should not be used to confirm nor deny a recent or acute infection. The measurement of IgG avidity may allow, in the presence of IgM, to exclude the risk of maternal-foetal transmission of toxoplasmic infection [13]. It studies the dissociation of the Ag/Ac bond by treatment with urea. The higher the avidity, the older the infection. A threshold of exclusion of recent infection can thus be established according to the techniques used. Conversely, a low avidity index does not necessarily prove a recent infection (the maturation of IgG can be more or less long depending on the individual) [13]. Regarding our patient, maternal serology revealed elevated IgG and IgM antibodies but the late gestational age and the absence of previous samples did not allow us to rule out the diagnosis of recent seroconversion. Suspected fetal infection based either on maternal seroconversion or fetal sonographic findings levies a fetal evaluation. Fetal ultrasound can show suggestive abnormalities including increased placental thickness, intracranial densities, intrahepatic densities and/or hepatomegaly, ascites, pericardial and/or pleural effusion and or ventricular dilatationnevertheless, it is under no circumstances a diagnostic tool. In our case, fetal Toxoplasmosis was suspected upon morphological abnormalities: cerebral abscess and hydrops fetalis associated with prefrontal edema, pleurisy and fetal ascites [14]. Given the severity of fetal manifestations, maternal seroconversion in the first trimester is highly suspected.In symptomatic newborns, Toxoplasmosis diagnosis should be differentiated from the other TORCH (Toxoplasma, Rubella, CMV, HSV). The diagnosis is led by a well conducted history and a meticulous physical examination associated with a lumbar puncture and a head CT (Computed Tomography). The parasite can either be isolated (in the placenta, the umbilical cord or the newborn's blood) or detected using PCR. Serologic tests are recommended to confirm the diagnosis [14].

Since there is no effective cure, prevention of primary infection is the key. Educating women especially during preconceptional counseling can reduce the seroconversion rate during pregnancy by 60% [15]. Gestational age is the determining factor of how to approach treatment therapy. In an effort to prevent fetal transmission if the presence of acute Toxoplasmosagondii infection is confirmed in a pregnant woman, treatment with Spiramycin can be initiated. Whilst in a confirmed fetal infection through amniocentesis, a switch to pyrimethamine and sulfadiazine is advocated after the first semester associated with folinic acid [16].

Conclusions

Congenital toxoplasmosis is more serious when contracted in early pregnancy. A serological test for anti-toxoplasmic IgG is recommended in early pregnancy to establish the precise gestational age of seroconversion. It is imperative to remind practitioners of the dramatic complications of congenital toxoplasmosis as in our case so that they can ensure an effective prevention.

Declarations

Guarantor of submission: The corresponding author is the guarantor of submission.

Acknowledgements: None.

Funding: There are no funding sources to be declared.

Competing interests: The authors declare that they have no competing interests.

Consent for publication: Written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

Ethics approval and consent to participate: Ethics approval has been obtained to proceed with the current study. Written informed consent was obtained from the patient for participation in this publication.

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