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LETTER TO THE EDITOR

Intrauterine parvovirus B19 infection: early prenatal diagnosis is possible

Human parvovirus B19 infection occurs commonly in childhood and is also found in previously seronegative adults. The prevalence of B19 seropositivity amongst adults is between 40—60%[1,2] and is between 35—55% in pregnant women.

Women of childbearing age in the USA show an annual seroconversion rate of 1.5%.[3] Transplacental transmission of parvovirus B19 to the fetus may be common following maternal infection. A large prospective study on 193 cases of confirmed B19 infection in pregnancy estimated that the fetal infection rate was 33%, and found a risk of non-immune hydrops fetalis and of fetal mortality of around 9%.[4,5] Moreover, sporadic cases of meconium peritonitis in hydropic fetuses have been reported.[6] However, it is not yet known whether some congenital abnormalities, such as ocular abnormalities and central nervous system abnormalities, are correlated with parvovirus B19 infection during pregnancy.[7,8]

During an outbreak of erythema infectiosum in Florence in the spring of 2002, a 37-year-old pregnant woman developed an erythema infectiosum in the 14th week of gestation. Maternal serum proved positive for anti-parvovirus B19 IgM and IgG (Parvo IgM and Parvo IgG EIA, Seiken, Tokyo, Japan) as well as for B19 DNA sequences by a nested polymerase chain reaction (PCR); this reaction amplifies B19 DNA sequences in the region coding for the NS1 protein, as already described.[9] Amniotic fluid collected two weeks after the onset of the rash was positive for B19 DNA by PCR. As maternal infection with parvovirus B19 is now recognized as a potential risk for adverse pregnancy outcome, especially during the first 20 weeks of pregnancy, ultrasound monitoring was performed and no fetal abnormality was observed. At 36 weeks, a boy of 4.6 kg was born, with Apgar scores of 7 and 8 at one and five minutes respectively, with normal hematological data and without any other abnormalities (echocardiography, cranial ultrasound and chest X-ray were normal). B19 DNA was absent in the newborn serum.

As far as we know, there are no data about the reliability of the nested PCR performed on amniotic fluid with regard to (1) gestational age and (2) the interval between maternal infection and amniocentesis. In this case, nested PCR on amniotic fluid was found positive at the 16th week of gestation, only two weeks after the onset of erythema infectiosum. We therefore believe that prenatal diagnosis of parvovirus B19 infection may be performed earlier compared to varicella, toxoplasmosis and cytomegalovirus infection, where the best results are achieved when amniotic fluid is taken after 18 weeks (21 weeks for cytomegalovirus) of gestation and 4—6 weeks after onset of maternal disease (6—9 weeks for cytomegalovirus).

Additionally in our case, B19 DNA levels were low, only detectable by a qualitative PCR, being under the sensitivity of the real time quantitative PCR used, either in maternal serum or in amniotic fluid. B19 DNA was negative in newborn serum. It is possible that a mild fetal infection was easily cleared before birth. More observations are required in order to assess the prognostic value of a quantitative detection of B19 DNA in maternal serum and amniotic fluid. We suggest that prenatal diagnosis (preferably early) could be useful because the real risk of congenital abnormalities connected to parvovirus B19 is unknown at this time and because many terminations of pregnancy, made for this reason, could be avoided.

Finally, this observation suggests that the performance of B19 infection diagnosis on maternal and newborn blood could lead to an underestimation of intrauterine B19 infection rate.

Conflict of interest: No conflicting interest declared.

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