Differential Transmission of Parvovirus B19 in a Twin Gestation: A Case Report

Raymond T. Foster Sr. and Steven R. Allen

Department of Obstetrics and Gynecology, Scott and White Hospital and Clinics, Texas A&M University System Health Science Center College of Medicine, Temple, Texas, United States of America

Maternal infection with parvovirus B19 during pregnancy can cause aplastic anemia in the fetus. Severe anemia may lead to nonimmune hydrops or fetal demise. In the case reported, the demise of one twin was diagnosed by ultrasonography in an asymptomatic 21-year-old para 1-0-2-1 African American at the gestational age of 25 weeks. The deceased twin (A) was grossly hydropic with anasarca, ascites, pleural and pericardial effusions, and a thickened placenta. Parvovirus B19 DNA was found in the amniotic fluid of Twin A using the polymerase chain-reaction technique. Serial scans of Twin B showed normal growth and no evidence of hydrops. The pregnancy was managed expectantly until 29 weeks when delivery was indicated by maternal disseminated intravascular coagulation. Maternal IgM antiparvovirus B19 antibodies were detected at the time of delivery. Antiparvovirus B19 IgM antibodies were not present in Twin B. These serologic studies suggest a recent acute maternal infection and refute such an infection in Twin B. We present a case of differential transmission of parvovirus B19 in a twin pregnancy with *in utero* death of the infected twin and subsequent maternal disseminated intravascular coagulation.

Parvovirus B19, the only parvovirus that infects humans, commonly causes a trivial infection in children and adults. In children, the self-limited infection typically presents as an erythematous macular rash, whereas in some adolescents and adults polyarticular arthritis develops, mimicking rheumatoid arthritis. Because parvovirus B19 infects red blood cell progenitors, patients with hematologic disorders such as sickle cell anemia can develop a transient aplastic crisis.

Fetal infection can lead to anemia, hydrops fetalis, and even fetal death. Fetal mortality after maternal parvovirus B19 infection has been previously reported between 5% and 16% (Miller, Fairley, Cohen, & Seng, 1998; Public Health Laboratory Service Working Party on Fifth Disease, 1990; Rodis et al., 1990) in three separate prospective trials. We identified a single prior report of differential parvovirus B19 transmission in a twin gestation (Pustilnik & Cohen, 1994). A twin gestation with differential fetal infection leading to death of the infected twin and subsequent maternal disseminated intravascular coagulation (DIC) is herein reported.

Case Report

A 21-year-old para 1-0-2-1 African American with a diamnionic/dichorionic twin pregnancy was found to have an intrauterine fetal demise of one twin during an ultrasound exam performed to assess fetal growth at the gestational age of 25 weeks. The exam showed concordant normal interval growth and normal amniotic fluid volume for each twin. The deceased twin (A) was hydropic, with anasarca, ascites, pleural and pericardial effusions, and a thickened placenta. Twin B's ultrasound exam was unremarkable.

Maternal serum studies (Kleihauer-Betke stain, TSH, total thyroxine, VDRL, rubella IgG antibody, human immunodeficiency virus antibody, and an indirect coombs test) were normal, and therefore failed to identify an etiology for the fetal hydrops and demise. An amniocentesis was performed on Twin A, yielding a normal karyotype, negative amniotic fluid polymerase chain reaction (PCR) assay for cytomegalovirus and toxoplasmosis, and a positive PCR for parvovirus B19 DNA. Parvovirus B19 IgM antibodies were detected in maternal serum at delivery, suggesting a recent primary maternal infection.

At the gestational age of 29 weeks, the patient was hospitalized for preterm, premature rupture of membranes without preterm labor. Twin B had normal interval growth and a reassuring biophysical profile. Betamethasone was given (12 mg IM q 24h X 2) to promote fetal lung maturity. Coagulation studies were normal (platelets 161×10^3 cells/μl, fibrinogen 348 mg/dL, and minimally-elevated fibrin-split products, 10–20 μg/mL).

Received 10 June, 2003; accepted 11 June, 2004.

Address for correspondence: Raymond T. Foster Sr., M.S., M.D., Fellow, Division of Urogynecology, Department of Obstetrics and Gynecology, Box 3192, Duke University Medical Center, Durham, North Carolina 27710, USA. E-mail: raymond.foster@duke.edu

Table 1

Maternal Serum Laboratories Documenting Consumptive Coagulopathy

Date	5/16/01	6/1/01	6/4/01	6/14/2001 (1417 hrs)	6/14/2001 (2200 hrs)	6/15/01	6/17/01
Estimated gestational age	25 weeks	27–3/7 weeks	27–6/7 weeks	29–2/7 weeks	29–2/7 weeks	29–3/7 weeks	29–5/7 weeks
Platelets (10^3 cells/μL)	118	178	161	138	146	132	127
Fibrinogen (mg/dL)	341	348		212	228	202	
Fibrinogen split products (μg/mL)		> 10, < 20		> 40	> 40	> 40	
Protime	12.6			12	12.6	12.8	
aPTT	36.1			38.2	36.3	39	
INR	1.1			1.1	1.1	1.1	

At the gestational age of 29 and 2/7 weeks, 4 weeks after Twin A's demise had been documented, the patient developed minimal vaginal bleeding. Laboratory studies consistent with DIC (Table 1) prompted a decision to proceed with delivery.

The patient underwent a misoprostol induction of labor and delivered a liveborn Twin B with a birthweight of 1302 grams. Twin B had no identifiable antiparvovirus B19 IgM antibodies or parvovirus DNA on the day of birth; in its absence the positive serum IgG assay represents transplacentally acquired maternal antibody. The patient did well after delivery with no clinical evidence of ongoing consumptive coagulopathy. The baby was discharged home on its 18th day of life.

The patient declined an autopsy of the stillborn infant. The placenta was sent to pathology for gross and microscopic investigation. The pathologist confirmed diamnionic/dichorionic gestation. Microscopic evaluation did not provide information that would alter suspicion of fetal death resultant from intrauterine infection with parvovirus B19.

Discussion

We present a case of a fatal congenital parvovirus B19 infection that affected only one fetus in a twin gestation. This case demonstrates that parvovirus B19 infection in the gravid patient has potentially devastating obstetric consequences. Fetal anemia, caused by parvovirus infection of hematologic progenitor cells and/or cardiac myocytes, can lead to nonimmune hydrops fetalis and death. Fetal infection develops in up to 20% of infected gravidas, with nearly half of these fetal infections leading to fetal demise or spontaneous abortion (Miller et al., 1998). The risk of pregnancy loss is greatest when maternal infection occurs during the first half of pregnancy, although nonfatal congenital infection is more likely as maternal infection occurs closer to term (Miller et al., 1998).

The pregnancy discussed herein was further complicated by maternal disseminated intravascular coagulation, developing approximately 4 weeks after identification of the fetal demise. We have identified only two prior reports of overt DIC in the mother of a multiple gestation with a fetal demise (Romero, Duffy, Berkowitz, Chang, & Hobbins, 1984; Skelly, Marivate, Norman, Kenoyer, & Martin, 1982). In both previously reported cases, heparin therapy was successfully used to reverse deteriorating laboratory abnormalities (fibrinogen and fibrin degradation products), which allowed 7 to 9 weeks prolongation of the periviable pregnancies (Romero et al., 1984; Skelly et al., 1982); heparin was not used in our patient due to a more advanced gestational age. To our knowledge, this patient is the first reported case of differential transmission of parvovirus B19 in a twin gestation which resulted in maternal DIC after expectant management of a single-twin demise.

In this case, a good outcome was obtained with expectant management. The literature supports intervention, that is, heparin therapy, to prolong the pregnancy in cases of extreme prematurity (Romero et al., 1984; Skelly et al., 1982). We are convinced, however, that such treatment would not have improved the outcome of Twin B in this patient.

References

Miller, E., Fairley, C. K., Cohen, B. J., & Seng, C. (1998). Immediate and long term outcome of human parvovirus B19 infection in pregnancy. *British Journal of Obstetrics and Gynaecology, 105,* 174–178.

Public Health Laboratory Service Working Party on Fifth Disease. (1990). Prospective study of human parvovirus (B19) infection in pregnancy. *British Medical Journal, 300,* 1166–1170.

Pustilnik, T. B., & Cohen, A. W. (1994). Parvovirus B19 infection in a twin pregnancy. *Obstetrics and Gynecology, 83,* 834–836.

Rodis, J. F., Quinn, D. L., Gary, G. W., Anderson, L. J., Rosengren, S., Cartter, M. L., Campbell, W. A., & Vintzileos, A. M. (1990). Management and outcomes of pregnancies complicated by human B19 parvovirus

https://doi.org/10.1375/twin.7.5.412 Published online by Cambridge University Press

infection: A prospective study. *American Journal of Obstetrics and Gynecology, 163,* 1168–1171.

Romero, R., Duffy, T. P., Berkowitz, R. L., Chang, E., & Hobbins, J. C. (1984). Prolongation of a preterm pregnancy complicated by death of a single twin in utero and disseminated intravascular coagulation. Effects of treatment with heparin. *The New England Journal of Medicine, 310,* 772–774.

Skelly, H., Marivate, M., Norman, R., Kenoyer, G., & Martin, R. (1982). Consumptive coagulopathy following fetal death in a triplet pregnancy. *American Journal of Obstetrics and Gynecology, 142,* 595–596.

https://doi.org/10.1375/twin.7.5.412 Published online by Cambridge University Press