Congenital infection by human parvovirus B19 ascites-anaemia

Ch. Giannakopoulou[1], E. Hatzidaki[1], K. Giannakopoulos[2], N. Papanicolaou[2], A. Patrikalakis[2], H. Kokori, K. Relakis[2]

[1]Department of Neonatology of the University of Crete; [2] Obstetrics and Gynaecological Clinic, Medical School, University of Crete (Greece)

Summary

A case of intrauterine infection by human parvovirus B19 (HPV B19) manifested as ascites during pregnancy is presented. Ascites was diagnosed by ultrasound at 27 weeks' gestation. A caesarean section was performed at 37 weeks' owing to affected mobility of the fetus. A pale, female infant with low haemoglobin and bradycardia was delivered. Polymerace Chain Reaction (PCR) lab tests revealed that the mother and the fetus were infected by HPV B19.

The neonate was born with low haemoglobin (Hb=10g/dl) and with ascites; it was discharged in good general condition 50 days after delivery.

Key words: Human parvovirus B19; Ascites; Anemia.

Introduction

Congenital infection by HPV B19 was initially described in 1983 during the outbreak of the Fifth Disease in Scotland [1, 2]. Since then a number of reports on congenital infection and fetal loss have been published.

Fetal loss may be observed during the first, second and third trimester of gestation owing to the infection of the fetus by HPV B19. However, intrauterine infection is not always fatal. It can also be transient and the fetus may be born healthy or unhealthy.

It has been more 15 years since the first report of infection by HPV B19 during pregnancy and the study of congenital infection. However, rates have yet to be established as far as fetal infection and outcome of infected fetuses are concerned. In addition, there is no consensus in terms of proper treatment methods.

Here we describe a case of congenital infection of a full term neonate by HPV B19.

Case Report

A female infant of 37 weeks' gestation, 2770-gr, was born to healthy parents of two other children. Those pregnancies were unremarkable and no febrile or unfebrile episodes associated with infection were reported. The siblings, 4 years and 20 months of age, respectively, are in good health. During this pregnancy standard ultrasound at 27 weeks' gestation revealed the presence of fluid in the peritoneal cavity of the neonate; no other pathological findings were observed. Periodic ultrasound examinations up to 37 weeks' gestation were also done. At 37 weeks' gestation, owing to affected fetal mobility, a caesarean section was performed. Upon delivery the infant was pale with bradycardia (pulse <50/min).

The infant was intubated and remained under mechanical support for two days. Following examination of the systems, the clinical condition of the neonate was unremarkable except for paleness and distension of the abdomen.

Laboratory tests: Mother's blood type: A Rh+, indirect Coombs (–), mother's serologic tests manifested IgM, anti-HPV B19, and PCR (+) for parvovirus B19. Respective infant's lab tests: Blood type A Rh+, direct Coombs (–), PCR (+) for HPV B19 infection, Hb 10 g/dl, WBC = 6800/mm^3, PLT = 198,000/mm^3, reticulocyte count = 6.2%, CRP (–), SGOT = 31 IU/ml, SGPT = 8 IU/ml, globulin 2.9 gr/100 ml, Na = 145 meq/L, K = 5.1 meq/L, Ca = 8.5 mgr/100 ml, total bilirubin = 1.2 mgr/dl, serum creatinine = 1 mg/dl, serum LDH = 337 IU/L.

During the second day of life, the neonate was transfused owing to mechanical support requirements. Ultrasound of the brain showed sub-dense imaging of the white matter round the ventricle, probably due to incomplete maturation of the nervous system. CT of the brain was normal, so were fundoscopy and ultrasound of the heart.

During the third day of the neonate's life, we proceeded with the surgical removal of the ascetic fluid. The fluid was yellowish with small mature lymphocytes. An immuno-histochemical test showed the presence of B and T indices in the lymphocytes, a fact which sustains the view that the fluid was exudative.

During the seventh day of life, the neonate presented with convulsions and was administered phenobarbitale (25 mg/kgr of body weight). The neonate remained in the clinic for 50 days because of feeding difficulties.

On discharge the neonate was in good general condition while objective examination of the various systems did not show anything pathological. Lab test results were: Hb = 8.2 g/dl, Ht = 24.9%, Reticul. = 7.9%, WBC = 7,100/mm^3.

Discussion

Ascites may be the initial stage of hydrops. In this sense the differential diagnosis should include, following exclusion of alloimmunization, the etiology of nonimmune hydrops as represented by congenital infections, particularly by cytomegalovirus, toxoplasmosis, etc. [3].

In the case at hand, the immunologic aetiology of ascites and anemia were excluded following specific investigations while PCR lab tests showed recent HPV B19 infection of the mother and neonate. It is obvious that intrauterine infection by HPV B19, a cause for nonimmune hydrops or fetal mortality during pregnancy, is in this case responsible for the ascites [1, 4].

Received April 7, 1998
revised manuscript accepted for publication May 12, 1998

For pregnant women, clinical manifestations of HPV B19 infection are similar to those observed in the general population. This infection should be suspected at the onset of rush or arthritic pain in pregnant women. The diagnosis is confirmed with serologic evidence of IgM antibodies. Diagnosis of fetus infection is established with DNA investigation of the virus in the amniotic fluid or by serologic investigation of blood samples received with paracenticis from the umbilical vessels.

PCR has been used by many researchers to this end [5]. In many cases, as well as in this case, infection in the mother is asymptomatic [1]. There is no consensus in the literature regarding the seriousness of the risk for fetuses infected by their mothers. Infection of a pregnant woman by HPV B19 may result in miscarriage, death of the fetus, and the presence of hydrops and anemia in the neonate.

Assessments of intrauterine infection vary, however, there seems to be a consensus over a 33% rate, while the risk for fetal loss is estimated at 9% or less [6, 7]. A recent estimate of fetal loss is 5% while hydrops is estimated at 26% for fetuses carried by women infected with HPV B19 [7, 8].

The period between infection of the mother and fetal loss ranges from 1 to 16 weeks. This implies that women should have periodic ultrasound investigations. Generalized edemas, pericardium development, pleuritis or peritoneum fluid – as in the present case – may develop. These are non-specific findings which are manifested in other fetal pathologies [3, 7, 9].

In maternal B19 parvovirus infection fetal loss may be attributed to severe anemia with fetal-hydrop hypoxemia and heart failure as its main manifestations. There are also reports of HPV affecting the cardiac muscle [7]. Hydrop aetiology is not well understood since hydrops may occur outside severe anemia. In our case, cardiology investigation by ultrasound showed nothing pathological, therefore heart insufficiency cannot be suspected as the cause of ascites. The fate of fetuses that escape death during maternal B19 parvovirus infection is rather good [10-13]. Even with the manifestation of ascites or hydrops, prognosis can be good. During the neonatal period infected fetuses can be very healthy, or they may develop rush, thrombopenia of an immunologic type, and anemia. Encephalopathy has been reported in 2 neonates with mothers infected by the B19 parvovirus during 21-24 week's gestation. The neonates presented with convulsions, intracranial calcifications and absence of cells in the anterior horns of the CNS [14].

Whether the convulsions in our neonate during the seventh day of her life indicate cerebral damage owing to infection remains to be established.

Intrauterine infection by HPV B19 does not seem to predispose the manifestation of congenital anomalies [7, 10].

Initially intrauterine transfusion was considered to protect and treat severe anemia in fetuses. This method was used with good results [8]. However, the value of this method was later disputed since in many cases of intrauterine infection automatic remission has been observed. In addition, there is the risk of fetal haemorrhage due to possible thrombopenia and to the fact that transfusion is responsible for further suppression of erythropoesis, besides that caused by HPV B19 [7, 15].

An alternative gammaglobulin treatment has been proposed both for the mother and the fetus [16-18]. The results of this method of treatment are pending.

During an epidemic of the Fifth Disease, pregnant women should avoid contact with children with rush. In this case prevention is much safer than other methods of treatment for intrauterine infection by HPV B19.

References

[1] Anand A., Gray E. S., Brown T., Clewley J. P., Cohen B. J.: "Human parvovirus infection in pregnancy and hydrops fetalis". *New Engl. J. Med.*, 1987, *316*, 4, 183.

[2] Bora Ch., Giannacopoulou Ch.: "Parvovirus B19 infection in immunocompromised patients". *Iatriki.*, 1993, *63*, 1, 36.

[3] Jordan J. A.: "Identification of human parvovirus B19 infection in idiopathic nonimmune hydrops fetalis". *Am. J. Obstet. Gynecol.*, 1996, *174*, 37.

[4] Rodis J. F., Hovick T. J. Jr., Quinn D. L., Rosengren S. S., Tattersall P.: "Human parvovirus B19 infection in pregnancy". *Obstet. Gynecol.*, 1988, *72*, 5, 733.

[5] Torok T. J.: "Human parvovirus B19". In: "Infectious Disease of the fetus & newborn infant". Remington J. S., Klein J. O., Philadelphia, Pennsylvania, W. B. Saunders Company, 1995, 668.

[6] Prospective study of human parvovirus B19 infection in pregnancy. Public Health Laboratory Service Working Party on Fifth Disease. *B. M. J.*, 1990, *300*, 6733, 1166.

[7] Levy R., Weissman A., Blomberg G., Hagay Z. J.: "Infections by parvovirus B19 during pregnancy: a review". *Obstet. Gynecol. Surv.*, 1997, *52*, 4, 254.

[8] Schwarz T. F., Roggendorf M., Hottentrager B., Deinhart F., Enders G., Gloning K. P. *et al.*: "Human parvovirus B19 infection in pregnancy". *Lancet.*, 1988, *2*, 8610, 566.

[9] Holzgreve W., Curry C. J., Golbus M. S., Callen P. W., Filly R. A., Smith J. C.: "Investigation of nonimmune hydrops fetalis". *Am. J. Obstet. Gynecol.*, 1984, *150*, 7, 805.

[10] Keyserling H. L.: "Other viral agents of perinatal importance. Varicella, parvovirus, respiratory syncytial virus and enterovirus". *Clin. Perinatol.*, 1997, *24*, 1, 193.

[11] Rodis J. F., Rodner C., Hansen A. A., Borgida A. F., Deoliveira I., Shulman Rosengren S.: "Long term outcome of children following maternal human parvovirus B19 infection". *Obstet. Gynecol.*, 1998, *91*, 1, 125.

[12] Pryde P. G., Nugent C. E., Prigjian G., Barr M., Faix R. G.: "Spontaneous resolution of nonimmune hydrops fetalis secondary to human parvovirus B19 infection". *Obstet. Gynecol.*, 1992, *79*, 859.

[13] Wright I. M., Williams M. L., Cohen B. J.: "Congenital parvovirus infection". *Arch. Dis. Child.*, 1991, *66*, 2, 253.

[14] Conry J. A., Torok T., Andrews I.: "Perinatal encephalopathy secondary to in utero human parvovirus B19 infection". *Neurology*, 1993, *43*, 4, A346.

[15] Centers for Disease Control.: "Risks associated with human parvovirus B19 infection". *MMWR.*, 1989, *38*, 6, 81.

[16] Selbing A., Josefsson A., Dahle L. O., Lindgren R.: "Parvovirus B19 infection during pregnancy treated with high-dose intravenous gammaglobulin". *Lancet.*, 1995, *345*, 8950. 660.

[17] Schwarz T. F., Roggendorf M., Hottentrager B., Modrow S., Deinhardt F., Middeldorp J.: "Immunoglobulins in the prophylaxis of parvovirus B19 infection". *Infect. Dis.*, 1990, *162*, 5, 1214.

[18] Rodis J. F., Quinn D. L., Gary G. W. Jr., Anderson L. J., Rosengren S., Gartter M. L. *et al.*: "Management and outcomes of pregnancies complicated by human B19 parvovirus infection: a prospective study". *Am. J. Obstet. Gynzecol.*, 1990, *163*, 1168.

Address reprint requests to:

C. GIANNACOPOULOU, M. D.
37 Atlandithos St.
71305 Heraklion, Crete, Greece