

Non-Immune Hydrops Fetalis Secondary to Parvovirus B19 Infection with Favorable Perinatal Outcome: Case Report and Review of the Literature

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Abstract

Parvovirus B19 infection is an uncommon yet feared cause of fetal complications, particularly anemia and non-immune hydrops. We present the case of a 23-year-old woman who acquired a primary infection during pregnancy; her fetus developed severe anemia that required two intrauterine transfusions, culminating in a favorable neonatal outcome. A literature review is included to contextualize the diagnostic and therapeutic challenges.

1 Introduction

Parvovirus B19 is a DNA virus belonging to the family *Parvoviridae*. The virus was first identified in 1975 in a blood-donor bag labeled “B19” [1]. It is transmitted principally via respiratory secretions, blood products, or transplacentally. Maternal infection during pregnancy is often asymptomatic, yet it can lead to severe fetal complications, notably profound anemia, non-immune hydrops fetalis, and even intrauterine fetal death (IUFD) [2].

7 Case report

A 23-year-old gravida 2 para 1 woman (blood type A Rh-positive) with an unremarkable medical history and a husband of blood type O Rh-positive was referred at 23 weeks’ gestation for suspected fetal hydrops. Obstetric ultrasonography revealed massive fetal ascites and an elevated middle cerebral artery peak systolic velocity of 1.63 multiples of the median (MoM), consistent with moderate-to-severe fetal anemia. Maternal serology demonstrated parvovirus B19-specific IgM antibodies, confirming a recent infection. A first intrauterine transfusion (IUT) of 30 mL was performed, resulting in clear sonographic improvement; a second 20 mL IUT was administered at 27 weeks’ gestation. The subsequent course was uneventful, and at 37 + 3 weeks an elective Caesarean section delivered a growth-appropriate neonate (birth weight 2.9 kg; Apgar scores 10/10 at 1 and 5 minutes). Six-month follow-up showed normal growth and development.

Discussion

Parvovirus B19 infection is common, with a seroprevalence estimated at 50 %–75 % in adults, yet it often remains underdiagnosed during pregnancy. It is transmitted chiefly via respiratory secretions, blood products, or transplacentally, and affects roughly 1 %–5 % of pregnant women, with a vertical transmission risk to the fetus of 17 %–33 % [3]. During epidemic periods, this risk can be even higher, particularly in professionally exposed women such as teachers or mothers of young children, in whom seroconversion rates may reach 16 % [4].

Erythema infectiosum, or fifth disease, is the typical presentation in children, characterised by moderate fever, malaise, and the pathognomonic “slapped-cheek” facial rash, followed by a

maculopapular eruption on the trunk and limbs. In adults, especially pregnant women, infection is often asymptomatic (in 30 %–50 % of cases) but may present with an influenza-like syndrome, a “megalo-erythema”-type rash, and, above all, arthralgia of the hands, wrists, or knees that can be disabling. Rare manifestations include myocarditis and heart failure, aplastic crisis in the context of chronic anaemia, and complications in immunocompromised individuals [5].

During pregnancy, parvovirus B19 infection generally does not compromise maternal health, ¹ it can have significant fetal consequences. The vertical transmission rate is estimated at 17 %–³³ %, with the highest risk between 9 and 20 weeks of gestation [6]. ^{Most} infected fetuses, however, recover spontaneously without sequelae.

The principal complications are:

1. **Spontaneous miscarriage:** the risk is 13 % when infection occurs before 20 weeks’ gestation, versus only 0.5 % thereafter. This difference ² may be attributable to multivisceral lesions, even in the absence of anemia or hydrops. [6]
2. **Non-immune fetal hydrops:** this is the most overt manifestation of congenital infection. The risk depends on the gestational age at the time of infection:
 - Less than ⁵ 10 % if infection occurs before 12 weeks’ gestation
 - About 5 % between 13 and 20 weeks’ gestation
 - Less than 1 % after 20 weeks’ gestation [7]

The mechanisms involved include severe fetal ¹² anemia resulting from infection of erythroid precursors, compounded by the short lifespan ³ of fetal red blood cells during hepatic hematopoiesis. Additional causes comprise fetal viral myocarditis, which can progress to heart failure, as well as hepatic involvement attributable either to direct hepatocyte destruction or to indirect toxicity from iron (hemosiderin) deposition. [7]

Diagnosis relies on a combination of ultrasound findings—⁵ ascites, edema, effusions, placentomegaly—together with Doppler assessment of the middle cerebral artery (MCA) peak systolic velocity, where a value > 1.5 multiples of ⁶ the median (MoM) is indicative of moderate-to-severe anemia, and detection of maternal anti-parvovirus B19 IgM antibodies. Fetal confirmation can be obtained by PCR on either fetal blood or amniotic fluid [3][5][7].

Management is centered on intrauterine ² transfusion (IUT), which rapidly corrects the anemia and markedly improves prognosis. Reported perinatal survival rates after IUT range from 67 % to 85 %, while spontaneous regression of hydrops occurs in approximately 30 %–34 % of cases when anemia is only moderate [7]. In our patient, two transfusions were required and achieved both clinical and ultrasonographic improvement, culminating in a favorable outcome. Long-term follow-up of affected infants has revealed no significant neurodevelopmental sequelae, provided that intervention is timely.

At present, no licensed vaccine against parvovirus B19 exists. Preventive measures therefore depend on strict hygiene practices and avoidance of high-risk exposures in pregnant women. Serologic screening at the onset of pregnancy may be considered for women at particularly high risk of exposure, although routine testing is not yet universally recommended [5].

Conclusion

Parvovirus B19 infection during pregnancy is generally benign but can lead to serious complications, such as fetal anemia or hydrops. Diagnosis is based on serologic testing and middle cerebral artery (MCA) Doppler assessment. Intrauterine transfusion (IUT) markedly improves the prognosis when anemia is confirmed. Appropriate and timely management helps ensure a favorable fetal outcome.



Figure 1 : Image échographique à 23 SA montrant une ascite fœtale.

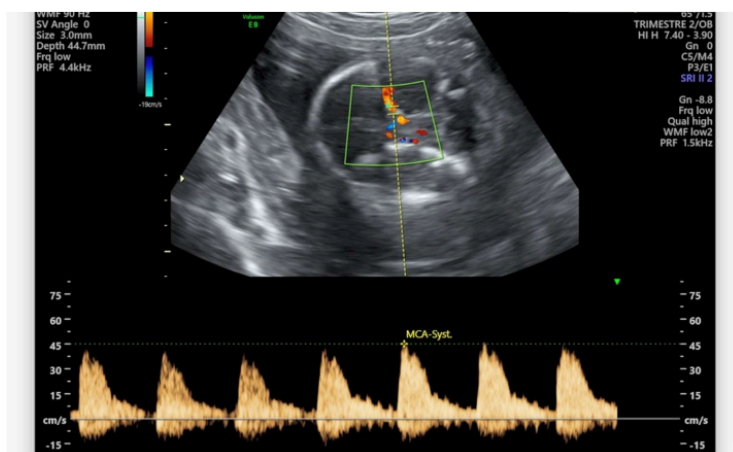


Figure 2 : Étude de la vélocimétrie de l'artère cérébrale moyenne à 23 SA objectivant un PSV à 1,6 MoM.

	Age gestationnel	Hb et Hte du sang fœtal en Pré transfusionnel		Volume transfusé	Hb et Hte du sang fœtal en post transfusionnel	
		Hb g/dl	Hte%		Hb g/dl	Hte%
1 TIU	23SA	10.3	31.5	30cc	12.8	39.1
2 TIU	27SA+6jr	8	23	25cc	13.4	53

Figure 3 : Tableau synthétisant les deux transfusions intra-utérines réalisées au cours de la prise en charge .

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