Original Article

MEDICINE PALLIATIVE CARE

DOI: 10.47582/jompac.1900670

J Med Palliat Care. 2026;7(3):475-481

Perinatal outcomes of maternal Parvovirus B19 infection during pregnancy: a tertiary referral center experience

Kübra Kurt Bilirer*[1], Cafer Yelkenci[2], Selvi Aydın Şenel[1], Barış Boza[1]

[1]*Department of Perinatology, Başakşehir Çam and Sakura City Hospital, İstanbul, Turkiye*

[2]*Department of Obstetrics and Gynecology, Başakşehir Çam and Sakura City Hospital, İstanbul, Turkiye*

Cite this article as: Kurt Bilirer K, Yelkenci C, Aydın Şenel S, Boza B. Perinatal outcomes of maternal Parvovirus B19 infection during pregnancy: a tertiary referral center experience. *J Med Palliat Care*. 2026;7(3):475-481. doi:10.47582/jompac.1900670

Received: 02.03.2026 ◆ **Accepted**: 08.05.2026 ◆ **Published**: 25.06.2026

ABSTRACT

Aims: To evaluate perinatal outcomes and the need for intrauterine transfusion (IUT) in pregnancies complicated by maternal Parvovirus B19 infection, and to assess the association between maternal serological patterns, fetal anemia, hydrops, and pregnancy outcomes.

Methods: This retrospective study included 18 pregnancies with laboratory evidence of acute Parvovirus B19 infection among 387 screened cases between 2021 and 2025 at a tertiary referral center. Maternal infection was defined by serological and/or molecular evidence. Fetal anemia was assessed using middle cerebral artery peak systolic velocity (MCA-PSV). IUT was performed when indicated.

Results: Hydrops was identified in 61.1% of cases and fetal anemia in 66.7%. IUT was performed in 55.6% of pregnancies. The overall live birth rate was 50.0%, while the live birth rate among cases undergoing IUT was 30.0%. All intrauterine fetal demises occurred in fetuses with both severe anemia and hydrops, whereas all cases without anemia or hydrops resulted in live birth.

Conclusion: Fetal prognosis in Parvovirus B19 infection during pregnancy is primarily determined by the development of anemia and hydrops. Although IUT is life-saving, it does not guarantee survival in advanced cases, particularly when severe anemia and hydrops coexist.

Keywords: Parvovirus B19, fetal anemia, hydrops fetalis, intrauterine transfusion, pregnancy outcome

INTRODUCTION

Parvovirus B19 (B19V) is a human pathogen belonging to the genus *Erythroparvovirus* within the family *Parvoviridae*, with a marked tropism for erythroid progenitor cells.[1] B19V infection is a common childhood viral illness known as erythema infectiosum (fifth disease). Transmission occurs predominantly via respiratory droplets and, less commonly, through blood transfusion or vertical transmission from mother to fetus during pregnancy.[2] In adults, infection is frequently asymptomatic or presents with mild fever, rash, and arthralgia.[3]

Globally, seroprevalence increases with age and exceeds 80% in the adult population. Data from European cohorts indicate that 26–43.5% of women are susceptible to B19V infection at the beginning of pregnancy.[4] The incidence of seroconversion during pregnancy is estimated at 1% in endemic periods, rising to as high as 13% during epidemic outbreaks.[5] Recent epidemic surges have renewed attention to B19V infection in pregnancy due to its potential impact on perinatal outcomes.[6]

Following acute maternal infection, the rate of vertical transmission to the fetus is reported to be approximately 30-50%.[7] The risk of fetal transmission and subsequent complications is highest during the first half of pregnancy, particularly between 9 and 16 weeks of gestation, which also represents the most vulnerable period for fetal loss.[8] Although most infected fetuses experience spontaneous resolution without adverse outcomes, fetal B19V infection has been associated with spontaneous abortion, severe anemia, nonimmune hydrops, myocarditis, and intrauterine fetal death.[9]

By targeting erythroid progenitor cells via the P antigen receptor, B19V suppresses erythropoiesis and induces apoptosis. During the second trimester, when erythrocyte lifespan is short and hematopoietic activity is high, transient aplasia may rapidly lead to severe anemia and high-output cardiac failure. Possible myocardial involvement may further aggravate cardiac dysfunction, contributing to the development of nonimmune hydrops fetalis.[10]

Nonimmune hydrops is defined by the accumulation of fluid in at least two fetal compartments, including ascites, skin edema, pleural effusion, and/or pericardial effusion. The most important determinant of hydrops development is the gestational age at which maternal infection occurs. When

***Corresponding Author:** Kübra Kurt Bilirer, kubrakbilirer@gmail.com

infection develops within the first 12 weeks of gestation, the reported risk of hydrops ranges between 5% and 10%, whereas after 20 weeks the risk decreases to below 1%.[11,12]

Although B19V is not considered a classical teratogen, rare associations have been reported. Whether these findings reflect direct viral effects or secondary hypoxic-ischemic injury due to severe anemia remains uncertain.[13,14]

The diagnosis of acute maternal infection is primarily established through serological testing. IgM antibodies become detectable approximately 7-10 days after infection and may decline over time, whereas IgG antibodies are regarded as markers of long-term immunity. However, the sensitivity of IgM testing decreases as infection progresses, and false-negative results may occur. In cases of clinical suspicion, detection of viral DNA in maternal blood by polymerase chain reaction (PCR) or repeat serological testing improves diagnostic accuracy.[15] For the diagnosis of fetal infection, detection of B19V DNA by PCR in amniotic fluid or cord blood represents the most reliable method.[15,16]

Noninvasive assessment of fetal anemia relies on measurement of the middle cerebral artery peak systolic velocity (MCA-PSV), which guides the timing of intrauterine intervention in cases of severe anemia.[9,11] When severe fetal anemia is identified, intrauterine transfusion (IUT) is life-saving and significantly reduces mortality.[10,12]

In the present study, we analyzed perinatal outcomes in pregnancies with suspected acute maternal B19V infection among women who underwent maternal B19V serological testing due to suspected exposure or ultrasound findings such as hydrops, fetal anemia, hyperechogenic bowel, or early-onset fetal growth restriction. The aim was to systematically evaluate the relationship between maternal serological patterns and the development of fetal infection, nonimmune hydrops, the need for IUT, and overall pregnancy outcomes, and to interpret these findings in light of the existing literature to provide evidence that may inform clinical management strategies.

METHODS

This retrospective study was conducted at a tertiary referral center between January 2021 and July 2025. This study was approved by Ethics Committee No. 1 of Başakşehir Çam and Sakura City Hospital, İstanbul Provincial Health Directorate (Date: 09.07.2025, Decision No: KAEC/09.07.2025.193). The study was conducted in accordance with the principles of the Declaration of Helsinki. Due to the retrospective design, informed consent was waived, and all patient data were anonymized prior to analysis.

Pregnant women referred due to suspicious sonographic findings and/or suspected B19V exposure and for whom maternal B19V serology (IgM and IgG) was requested were retrospectively evaluated. The presence of at least two of the following findings—skin edema, fetal ascites, pericardial effusion, or pleural effusion—was considered consistent with hydrops fetalis. A total of 387 patients who underwent maternal B19V IgM and IgG testing for these indications were screened, and 18 cases with laboratory evidence of acute maternal B19V infection were included in the study. The patient selection process and clinical outcome flow are illustrated in **Figure**.

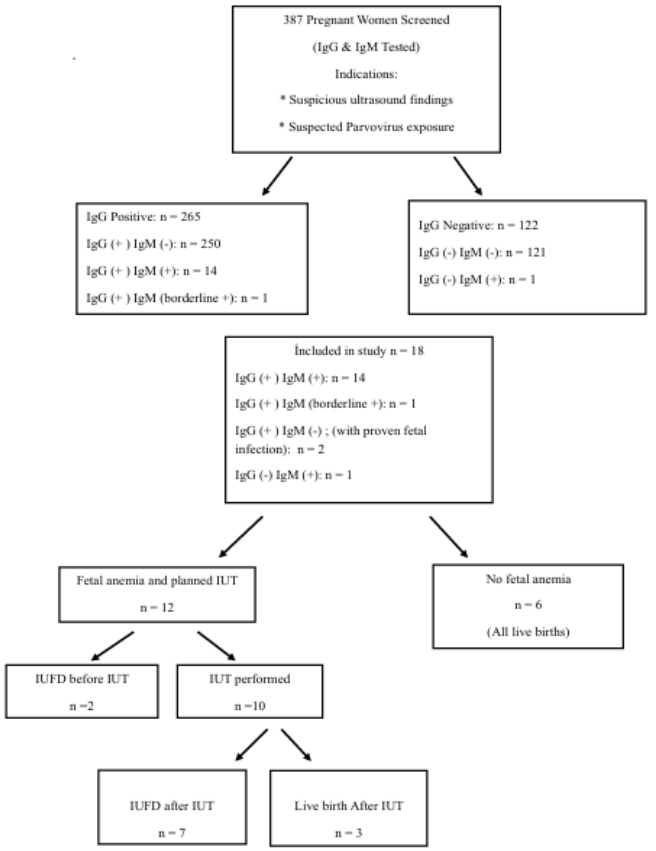

Figure. Flowchart of patient selection and clinical outcomes in pregnancies with suspected acute Parvovirus B19 infection
IUT: Intrauterine transfusion, IUFD: Intrauterine fetal demise

Acute maternal infection was defined based on maternal serological testing as the presence of IgM antibodies (with or without IgG positivity). In addition, cases with borderline IgM positivity according to institutional reference ranges were considered acute infection only if supported by positive maternal plasma PCR and/or confirmed fetal infection (positive B19V DNA PCR in amniotic fluid or cord blood). Cases with IgM negativity but IgG positivity were classified as acute infection only when supported by confirmed fetal infection. In such cases, IgM negativity was interpreted as reflecting a later stage of infection, in which IgM levels may have declined over time, and the diagnosis was supported by PCR positivity and/or confirmed fetal infection.

All ultrasound examinations were performed using a Hitachi Arietta ultrasound system by experienced perinatologists. Prenatal sonographic findings and other associated structural abnormalities were recorded. When feasible, Doppler assessment for fetal anemia was performed, and MCA-PSV measurements were obtained and interpreted according to gestational age-specific reference ranges and expressed as multiples of the median (MoM).

Maternal B19V IgM and IgG serologies were analyzed according to institutional laboratory protocols. Maternal plasma B19V DNA PCR was not routinely performed in all

patients and was obtained based on clinical availability and physician discretion. For the assessment of fetal infection, quantitative B19V DNA PCR was performed on amniotic fluid obtained via amniocentesis or on cord blood obtained via cordocentesis. Quantitative B19V DNA PCR results from maternal plasma, amniotic fluid, or cord blood were recorded as log10 copies/ml.

In cases with evidence of fetal anemia, IUT was planned. IUT procedures were performed by experienced perinatologists under local anesthesia using O Rh-negative packed red blood cells with a donor hematocrit of 88-90%. Intravascular transfusion via the umbilical vein under continuous ultrasound guidance was the standard approach in all cases and was performed by experienced operators.

For each patient, the indication for transfusion, number of transfusions, gestational age at each transfusion, pre-transfusion fetal hematocrit at the first IUT, and transfusion volume administered at each session were recorded.

Pregnancy outcomes were retrospectively evaluated, including intrauterine fetal demise, preterm live birth, and term live birth. For live-born neonates, gestational age at delivery, birth weight, and Apgar scores were documented.

RESULTS

A total of 18 pregnant women were included in the study. Detailed case-by-case maternal characteristics, prenatal ultrasound findings, MCA-PSV values, and B19V laboratory results are presented in **Table 1**.

The median maternal age was 29.5 years (range: 18-48), and the median gestational age at admission was 20.5 weeks (range: 15-29). All cases were diagnosed during the second trimester.

Hydrops fetalis, defined as fluid accumulation in at least two fetal compartments, was identified in 11/18 cases (61.1%). The most common ultrasound finding was ascites, observed in 13/18 cases (72.2%), followed by skin edema in 11/18 (61.1%), pericardial effusion in 9/18 (50%), pleural effusion in 8/18 (44.4%). Additional findings included cardiomegaly (11.1%), tricuspid regurgitation (22.2%), early-onset fetal growth restriction (11.1%), echogenic bowel (22.2%), hepatic calcifications (11.1%), placentomegaly (11.1%), mild ventriculomegaly (16.7%), long bone shortening (5.6%), and periventricular echogenic halo (5.6%).

MCA-PSV was assessed and the median MCA-PSV was 1.8 MoM (range: 0.85-2.6). MCA-PSV value >1.5 MoM was considered indicative of fetal anemia. Based on this threshold, fetal anemia was identified in 12/18 fetuses (66.7%). In one case in which MCA-PSV measurement was not feasible at initial evaluation due to early gestational age, subsequent assessment at a later gestational age demonstrated elevated MCA-PSV consistent with fetal anemia. Among anemic fetuses, the median MCA-PSV was 2.1 MoM (range: 1.61-2.6).

Maternal B19V serology was available for all cases. Serological profiles showed IgM+/IgG+ in 14/18 (77.8%), IgM+/IgG– in 1/18 (5.6%), IgM–/IgG+ in 2/18 (11.1%), and borderline IgM with IgG+ in 1/18 (5.6%).

Quantitative B19V DNA PCR testing in maternal plasma, when performed, revealed viral load values ranging from 2.27 to 11.67 log10 copies/ml. In cases in which PCR was performed on amniotic fluid or cord blood, viral load values ranged from 5.4 to 10.9 log10 copies/ml.

Fetal anemia was detected in 12/18 cases (66.7%), and IUT was planned accordingly. In two cases, intrauterine fetal demise occurred prior to the planned procedure (case 11 and 12). The clinical characteristics and pregnancy outcomes of the 10 fetuses who underwent IUT are presented in **Table 2**. IUT was performed in 10/18 cases (55.6%). The median gestational age at first IUT was 20 weeks (range: 18-26 weeks), and the median initial hematocrit before transfusion was 9.1% (range: 6-20%). In cases requiring repeated procedures, the maximum number of transfusion sessions was three.

Overall, intrauterine fetal demise (IUFD) occurred in 9/18 cases (50%). Notably, all IUFD cases occurred in fetuses with both severe anemia and hydrops, whereas all fetuses without anemia or hydrops resulted in live birth. Among these IUFD cases, 2 occurred prior to the planned IUT and 7 occurred following IUT. Post-transfusion fetal losses developed early after the procedure, with the interval between transfusion and fetal demise ranging from 1 to 14 days. In two cases, fetal demise occurred after the third transfusion session.

The overall live birth rate was 9/18 (50%). Among cases undergoing IUT, the live birth rate was 3/10 (30%). All cases without the development of fetal anemia or hydrops resulted in live birth. Among live-born infants, 6/9 (66.7%) were delivered at term and 3/9 (33.3%) were delivered preterm. The median gestational age at delivery was 38 weeks (range: 31-39), and the median birth weight was 3100 g (range: 1000-3470 g). Apgar scores ranged from 4/6 to 8/9.

DISCUSSION

In this study, we evaluated perinatal outcomes in a high-risk pregnancy cohort in which maternal serological testing was performed due to suspicious sonographic findings or suspected B19V exposure. Among 387 pregnant women screened for maternal IgG and IgM, IgG positivity was detected in 265 cases (68.5%). Previous reports indicate that 26-43.5% of women are susceptible to B19V infection at the beginning of pregnancy, corresponding to an overall immunity rate of approximately 56-74% among women of reproductive age.[4,18] The IgG seropositivity rate observed in our cohort is therefore consistent with the reported seroepidemiological range. However, our population represents a clinically selected high-risk group rather than an unselected obstetric population.

All cases were diagnosed during the second trimester, and hydrops as well as intrauterine fetal losses were predominantly observed in early to mid-gestation. These findings are in line with previous studies demonstrating that the risk of vertical transmission and fetal complications is highest within the first 20 weeks of pregnancy.[7,19] Meta-analytic data further confirm that fetal loss and hydrops occur more frequently in early gestation.[8]

Fetal anemia and hydrops were the most prominent clinical findings in our series, consistent with the existing literature.[20]

Table 1. Maternal characteristics, prenatal ultrasound findings and virological results in pregnancies with acute Parvovirus B19 infection

	Maternal age (years)	Gestational age at admission (weeks)	Prenatal ultrasound findings	MCA-PSV at admission (MoM)	Maternal Parvovirus B19 serology (IgM/IgG)	Parvovirus B19 DNA PCR (maternal plasma, quantitative log value)	Parvovirus B19 DNA PCR (amniotic fluid or cord blood, quantitative log value)
Case 1	30	24	Ascites Skin edema Pleural effusion Pericardial effusion Polyhydramnios cardiomegaly Tricuspid regurgitation	2.4	IgM (-) IgG (+)	3.1	10.9
Case 2	30	19	Ascites Skin edema Pleural effusion	2.2	IgM (+) IgG (+)	11.67	7.18
Case 3	18	17	Ascites Skin edema Pleural effusion Tricuspid regurgitation	2.6	IgM (+) IgG (+)	5.3	9.4
Case 4	30	17	Ascites Skin edema Cardiomegaly	1.61	IgM (+) IgG (+)	4.59	9.9
Case 5	31	20	Ascites Skin edema Pleural effusion Pericardial effusion	2.1	IgM (+) IgG (+)	Not performed	9.37
Case 6	28	15	Hepatic calcifications Ascites Tricuspid regurgitation	Not applicable*	IgM (+) IgG (-)	Not performed	5.7
Case 7	32	23	Ascites Skin edema Pleural effusion Pericardial effusion Placentomegaly	2	IgM borderline** (0.99) IgG (+)	4.12	8.7
Case 8	28	19	Ascites Skin edema Pleural effusion Pericardial effusion Tricuspid regurgitation Placentomegaly	2.1	IgM (-) IgG (+)	2.27	8.6
Case 9	25	18	Ascites Skin edema Pericardial effusion	1.9	IgM (+) IgG (+)	Not performed	6.1
Case 10	39	18	Ascites Skin edema Pleural effusion Pericardial effusion Echogenic bowel	1.8	IgM (+) IgG (+)	Not performed	5.4
Case 11	23	18	Ascites Skin edema Pleural effusion Pericardial effusion	2.1	IgM (+) IgG (+)	Not performed	6.14
Case 12	31	23	Ascites Skin edema Pericardial effusion Hepatic calcifications	2.2	IgM (+) IgG (+)	5.18	7.32
Case 13	29	17	Echogenic bowel pericardial effusion	0.85	IgM (+) IgG (+)	4.46	Not performed
Case 14	27	23	Unilateral mild ventriculomegaly	1.38	IgM (+) IgG (+)	5.24	Not performed
Case 15	48	21	Early onset FGR Echogenic bowel Mild ascites	0.88	IgM (+) IgG (+)	Not performed	8.2
Case 16	26	24	Bilateral mild ventriculomegaly Periventricular echogenic halo	1.32	IgM (+) IgG (+)	4.7	Negative***

The table continues

Table 1. Maternal characteristics, prenatal ultrasound findings and virological results in pregnancies with acute Parvovirus B19 infection (The table continues)

Case 17	22	29	Bilateral mild ventriculomegaly Mild long bone shortening	1.3	IgM (+) IgG (+)	Not performed	5.7
Case 18	30	23	Early onset FGR Echogenic bowel	0.9	IgM (+) IgG (+)	3.38	Not performed

MCA-PSV: Middle cerebral artery peak systolic velocity, MoM: Multiples of the median, PCR: Polymerase chain reaction, IgM: Immunoglobulin M, IgG: Immunoglobulin G, FGR: Fetal growth restriction
Maternal plasma Parvovirus B19 DNA PCR was not performed in all cases and was obtained based on clinical availability and physician discretion.
*MCA-PSV measurement was not performed due to early gestational age at diagnosis.
**IgM value of 0.99 was interpreted as borderline based on institutional laboratory cut-off values.
***Negative indicates undetectable Parvovirus B19 DNA by PCR.

Table 2. Intrauterine transfusion characteristics and pregnancy outcomes

	Intrauterine transfusion sessions (n)	Gestational age at IUT(s) (weeks)	Initial fetal hematocrit at first IUT (%)	Transfusion volume at each IUT (ml)	Pregnancy outcome
Case 1	1	25	8	15	Intrauterine fetal demise (1 day after IUT)
Case 2	3	20 21 22	8.9	10 13 15	Term live birth (38 weeks, 3100 g; Apgar 7/8)
Case 3	1	18	10.2	6	Intrauterine fetal demise (1 day after IUT)
Case 4	3	18 19 21	11	15 10 15	Intrauterine fetal demise (3 days after third IUT)
Case 5	3	20 21 23	9.2	10 14 15	Intrauterine fetal demise (2 weeks after third IUT)
Case 6*	1	23	20	25	Intrauterine fetal demise (1 day after IUT)
Case 7	1	26	6	15	Intrauterine fetal demise (1 day after IUT)
Case 8	1	20	13	10	Intrauterine fetal demise (1 day after IUT)
Case 9	2	19 21	9	15 15	Term live birth (37 weeks, 2650 g; Apgar 6/7)
Case 10	2	19 21	10	14 18	Spontaneous preterm live birth (32 weeks, 1700 g; Apgar 6/8)

IUT: Intrauterine transfusion
Values are presented in chronological order corresponding to each IUT session.
*Case 6: Fetal B19V infection was confirmed by amniotic fluid PCR at 15 weeks. Early ascites regressed spontaneously. Hydrops with MCA-PSV of 2.25 MoM developed at 23 weeks, leading to IUT. IUFD occurred 1 day after the procedure.

The overall IUFD rate was 50% (9/18), and notably, all fetal losses occurred in fetuses who developed both anemia and hydrops. In contrast, all six cases without anemia or hydrops resulted in live births. These findings reinforce the concept that the severity of hematologic impairment and the presence of hydrops—reflecting advanced hemodynamic compromise—are the principal determinants of fetal outcome.[21]

Recent evidence from a systematic review and meta-analysis has demonstrated that perinatal outcomes in B19V infection vary significantly according to the level of fetal involvement. While maternal infection alone is generally associated with a relatively low risk of intrauterine demise, the risk increases substantially in cases of confirmed fetal infection and is highest in fetuses requiring IUT. In addition, emerging data suggest that abnormal neuroimaging findings, particularly on fetal MRI, may be observed in some cases undergoing IUT, highlighting the potential role of advanced imaging in selected high-risk cases. These findings support the need for further large multicenter studies to better define the impact of prenatal interventions and the role of neuroimaging in clinical management.[22]

Cardiac findings such as cardiomegaly and tricuspid regurgitation were more prominent in cases resulting in fetal demise. Although these findings do not necessarily indicate direct myocardial infection, they may reflect advanced cardiovascular decompensation secondary to severe anemia, with or without additional viral myocardial involvement.[13,14]

In fetuses undergoing IUT, the median pre-transfusion hematocrit was 9.1%, indicating severe anemia. In B19V-related cases, pre-transfusion hemoglobin levels have been reported to be lower than those observed in other etiologies, and the need for transfusion often arises at earlier gestational ages.[23] Kosian et al.[24] reported a mean pre-transfusion hemoglobin level of 5.0 g/dl in infected fetuses. In our cohort, the median gestational age at first transfusion was 20 weeks (range 18-26 weeks), suggesting that infection may progress rapidly during the early to mid-second trimester.

Although the procedure-related fetal loss rate for IUT is generally reported to be approximately 1%, this risk varies depending on gestational age, operator experience, and particularly the presence of hydrops.[25,26] Bascietto et al.[21] demonstrated that post-transfusion loss rates are significantly

higher in hydropic fetuses. In our cohort, the high prevalence of hydrops and markedly low baseline hematocrit levels suggest that many fetuses were already in advanced stages of hemodynamic instability at the time of intervention. This may partially explain the elevated early post-transfusion loss rate observed in our series. These findings suggest that the relatively low survival rate observed in our cohort likely reflects the severity of the underlying disease rather than a limitation of the IUT procedure itself.

Our findings demonstrate that IgM negativity does not exclude acute infection. In two cases, fetal infection was confirmed despite negative maternal IgM results. These observations support previous reports emphasizing the importance of PCR testing when clinical suspicion persists.[15]

In our diagnostic strategy, amniocentesis with amniotic fluid B19V DNA PCR was performed in cases without fetal anemia or hydrops when invasive testing was accepted. In cases with documented fetal anemia, cordocentesis was undertaken for hematocrit measurement and simultaneous PCR confirmation, followed by IUT when indicated. PCR can be performed on both amniotic fluid and fetal blood samples. However, cordocentesis carries an approximately 1% procedure-related fetal loss risk, whereas amniotic fluid sampling is less invasive, associated with lower complication rates, and demonstrates higher detection rates. For this reason, amniocentesis is generally recommended as the preferred diagnostic modality.[9,27] In contrast, fetal serological testing is limited by immune immaturity and unpredictable antibody responses, resulting in low diagnostic sensitivity and lack of routine recommendation.[15]

Beyond hydrops, additional findings such as hepatic calcifications, echogenic bowel, mild ventriculomegaly, and fetal growth restriction were observed. Although these findings have been sporadically reported, B19V is not considered a classical teratogen.[13] The association between specific structural anomalies and maternal B19 infection is generally weak and primarily based on isolated case reports or small series. Central nervous system findings—including hydrocephalus, cerebellar hemorrhage, polymicrogyria, and neuronal heterotopia—have been described; however, a definitive causal relationship has not been established. Additionally, chromosomal abnormalities have been identified in some hydropic fetuses, and infection has been suggested to have a more severe course in genetically predisposed fetuses.[14] Therefore, the additional findings observed in our cohort are more likely attributable to secondary hemodynamic compromise, tissue hypoxia, or coincidental association rather than direct teratogenic effects.

Limitations

The strengths of our study include the detailed evaluation of maternal serological patterns in a clinically selected high-risk pregnancy cohort and the systematic demonstration of serological heterogeneity. The identification of IgM-negative but PCR-positive cases supports the notion that acute infection cannot be defined solely on the basis of IgM positivity. Furthermore, the comprehensive assessment of baseline hematocrit levels, gestational age at first intervention, and early post-transfusion losses provides valuable real-world data regarding the prognosis of severe B19V -related fetal anemia. However, our findings should be interpreted with caution, as the retrospective design, single-center setting, and relatively small sample size may limit the generalizability of the results. The lack of long-term neurodevelopmental follow-up also represents an important limitation.

CONCLUSION

B19V infection during pregnancy may present with variable clinical and serological patterns, and accurate diagnosis requires integrated interpretation of clinical findings, maternal serology, and, when indicated, molecular testing. Fetal infection is most reliably confirmed by detection of viral DNA in amniotic fluid or cord blood using PCR. Fetal prognosis is primarily determined by the development of anemia and nonimmune hydrops. In cases complicated by severe anemia and hydrops, particularly when accompanied by signs of cardiac dysfunction, outcomes may be significantly compromised, and IUT does not invariably ensure survival, as outcomes are largely influenced by the severity of fetal involvement.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study was approved by Ethics Committee No. 1 of Başakşehir Çam and Sakura City Hospital, İstanbul Provincial Health Directorate (Date: 09.07.2025, Decision No: KAEC/09.07.2025.193).

Informed Consent

This retrospective study used pre-existing anonymized patient data. No additional intervention was performed, and there was no direct patient contact. The study was approved by the Ethics Committee, and the requirement for written informed consent was waived by the ethics committee.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

The authors received no financial support for the conduct or publication of this research.

Author Contributions

Concept: KKB; Design: KKB; Control: KKB, CY; Resources: KKB, SAŞ, CY; Materials: KKB, SAŞ; Data Collection and/or Processing: KKB, SAŞ; Analysis and/or Interpretation: KKB, CY; Literature Review: KKB, SAŞ; Writing the Article: KKB, BB, CY; Critical Review: KKB, SAŞ, CY, BB.

REFERENCES

1. Dittmer FP, Guimarães CM, Peixoto AB, et al. Parvovirus B19 infection and pregnancy: review of the current knowledge. *J Pers Med*. 2024;14(2):139. doi:10.3390/jpm14020139

2. Hunter LA, Ayala NK. Parvovirus B19 in pregnancy: a case review. *J Midwifery Womens Health*. 2021;66(3):385-390. doi:10.1111/jmwh.13254
3. Gigi CE, Anumba DOC. Parvovirus B19 infection in pregnancy: a review. *Eur J Obstet Gynecol Reprod Biol*. 2021;264:358-362. doi:10.1016/j.ejogrb.2021.07.046
4. Bonvicini F, Bua G, Gallinella G. Parvovirus B19 infection in pregnancy: awareness and opportunities. *Curr Opin Virol*. 2017;27:8-14. doi:10.1016/j.coviro.2017.10.003
5. Jensen IP, Thorsen P, Jeune B, et al. An epidemic of Parvovirus B19 in a population of 3,596 pregnant women: a study of sociodemographic and medical risk factors. *BJOG*. 2000;107(5):637-643. doi:10.1111/j.1471-0528.2000.tb13306.x
6. Prasad S, Khalil A, Yinon Y, et al. Is Parvovirus B19 infection upsurge in 2023-2024 associated with adverse pregnancy outcome? *Ultrasound Obstet Gynecol*. 2025;66(3):307-313. doi:10.1002/uog.29301
7. Kagan KO, Hoopmann M, Geipel A, et al. Prenatal Parvovirus B19 infection. *Arch Gynecol Obstet*. 2024;310(5):2363-2371. doi:10.1007/s00404-024-07644-6
8. Xiong YQ, Tan J, Liu YM, et al. The risk of maternal Parvovirus B19 infection during pregnancy on fetal loss and fetal hydrops: a systematic review and meta-analysis. *J Clin Virol*. 2019;114:12-20. doi:10.1016/j.jcv.2019.03.004
9. Attwood LO, Holmes NE, Hui L. Identification and management of congenital Parvovirus B19 infection. *Prenat Diagn*. 2020;40(13):1722-1731. doi:10.1002/pd.5819
10. Malutan AM, Ormindean CM, Diculescu D, et al. Parvovirus B19 in pregnancy: do we screen for fifth disease or not? *Life (Basel)*. 2024;14(12):1667. doi:10.3390/life14121667
11. Society for Maternal-Fetal Medicine (SMFM), Norton ME, Chauhan SP, et al. Society for maternal-fetal medicine (SMFM) clinical guideline #7: nonimmune hydrops fetalis. *Am J Obstet Gynecol*. 2015;212(2):127-139. doi:10.1016/j.ajog.2014.12.018
12. Bouraddane M, Warda K, Zouhair S. Parvovirus B19 and pregnant women: a bibliographic review. *Open J Obstet Gynecol*. 2021;11(11):1543-1564. doi:10.4236/ojog.2021.1111145
13. Ergaz Z, Ornoy A. Parvovirus B19 in pregnancy. *Reprod Toxicol*. 2006;21(4):421-435. doi:10.1016/j.reprotox.2005.01.006
14. Ornoy A, Ergaz Z. Parvovirus B19 infection during pregnancy and risks to the fetus. *Birth Defects Res*. 2017;109(5):311-323. doi:10.1002/bdra.23588
15. Dijkmans AC, de Jong EP, Dijkmans BA, et al. Parvovirus B19 in pregnancy: prenatal diagnosis and management of fetal complications. *Curr Opin Obstet Gynecol*. 2012;24(2):95-101. doi:10.1097/GCO.0b013e3283505a9d
16. Kielaite D, Paliulyte V. Parvovirus (B19) infection during pregnancy: possible effect on the course of pregnancy and rare fetal outcomes. *Medicina (Kaunas)*. 2022;58(5):664. doi:10.3390/medicina58050664
17. Hartge DR, Weichert J, Gembicki M, et al. Confirmation of etiology in fetal hydrops by sonographic evaluation of fluid allocation patterns. *Eur J Obstet Gynecol Reprod Biol*. 2015;195:128-132. doi:10.1016/j.ejogrb.2015.09.006
18. Mossong J, Hens N, Friederichs V, et al. Parvovirus B19 infection in five European countries: seroepidemiology, force of infection and maternal risk of infection. *Epidemiol Infect*. 2008;136(8):1059-1068. doi:10.1017/S0950268807009661
19. Enders M, Weidner A, Zoellner I, et al. Fetal morbidity and mortality after acute human parvovirus B19 infection in pregnancy: prospective evaluation of 1018 cases. *Prenat Diagn*. 2004;24(7):513-518. doi:10.1002/pd.940
20. de Jong EP, Walther FJ, Kroes AC, et al. Parvovirus B19 infection in pregnancy: new insights and management. *Prenat Diagn*. 2011;31(5):419-425. doi:10.1002/pd.2714
21. Bascietto F, Liberati M, Murgano D, et al. Outcome of fetuses with congenital parvovirus B19 infection: systematic review and meta-analysis. *Ultrasound Obstet Gynecol*. 2018;52(5):569-576. doi:10.1002/uog.19092
22. Sorrenti S, Deuster E, D'Antonio F, Mascio DDI, Khalil A. Parvovirus B19 infection in pregnancy: perinatal outcomes derived from a systematic review and meta-analysis. *Am J Obstet Gynecol*. 2026:S0002-9378(26)00133-X. doi:10.1016/j.ajog.2026.03.002
23. Sánchez-Durán MÁ, Higueras MT, Avilés-García M, et al. Perinatal outcomes and central nervous system abnormalities following intrauterine fetal transfusion: 17 years' experience in a tertiary center. *Transfusion*. 2020;60(11):2557-2564. doi:10.1111/trf.16087
24. Kosian P, Hellmund A, Geipel A, et al. Intrauterine transfusion in 103 fetuses with severe anemia caused by parvovirus infection: a multicenter retrospective study. *Arch Gynecol Obstet*. 2023;308(1):117-125. doi:10.1007/s00404-022-06712-z
25. Lindenburg IT, van Kamp IL, Oepkes D. Intrauterine blood transfusion: current indications and associated risks. *Fetal Diagn Ther*. 2014;36(4):263-271. doi:10.1159/000362812
26. Zwiers C, Lindenburg ITM, Klumper FJ, et al. Complications of intrauterine intravascular blood transfusion: lessons learned after 1678 procedures. *Ultrasound Obstet Gynecol*. 2017;50(2):180-186. doi:10.1002/uog.17319
27. Crane J, Mundle W, Boucoiran I; Maternal Fetal Medicine Committee. Parvovirus B19 infection in pregnancy. *J Obstet Gynaecol Can*. 2014;36(12):1107-1116. doi:10.1016/S1701-2163(15)30390-X