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**Case Report**

# Non-immune hydrops fetalis: a case of parvovirus B19 infection in pregnancy

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## ABSTRACT

Parvovirus B19 infection during pregnancy is mostly asymptomatic, but in approximately 3% of infected women, it can cause hydrops fetalis, severe fetal anemia and even fetal demise. A 23 year old primigravida with 33+5 weeks period of gestation came to our hospital with threatened preterm labour and polyhydramnios. Ultrasound with Doppler showed features of fetal hydrops with fetal anemia. She was tested positive for parvovirus IgM and was planned for intra uterine transfusion. But patient went into preterm labour and delivered a single, stillborn, male of 2.75 kg with no visible gross anamolies. There is currently no approved vaccine or antiviral treatment for parvovirus B19 infection, but counselling of non-immune mothers and active management of confirmed maternal infections with intrauterine transfusion to correct fetal anemia is likely to improve the survival rates with significant reduction in neonatal morbidity and mortality.

**Keywords:** Parvovirus B19, Fetal hydrops, Intrauterine transfusion

## INTRODUCTION

Parvovirus B19 or the 5th disease infects 1-5% of pregnant women, generally with normal pregnancy outcome.[1] Risk of parvovirus B19 infection is higher in day care personnel and women of child bearing age with young children.[2] It can cause fetal loss, severe anemia, high output cardiac failure, fetal hydrops, neurological anamolies, intrauterine death.[3]

## CASE REPORT

A 23 year old primigravida with 33+5 weeks period of gestation came to our hospital with complains of threatened preterm labour and polyhydramnios. She was started on conservative management of preterm labour with tocolytics and steroids.

Her blood group was O positive and she had no co-morbidities.

On per abdomen examination, abdomen was grossly distended, uterus was 36 weeks size relaxed with free floating head, with FHR of 160 bpm.

Scan done at 28 weeks was normal.

Obstetric scan with Doppler done on admission showed gross polyhydramnios of 40.6 cm, bilateral pleural effusion, ascites, diffuse subcutaneous edema and Doppler showed increased MCA PSV values of 1.6 MoM which was suggestive of fetal hydrops with fetal anemia.

This indicated late onset fetal hydrops and the differential diagnosis included ABO incompatibility, TORCH infections and placental causes.

Relevant investigations were done and was tested positive for IgM parvovirus and she was planned for intrauterine transfusion.

Patient did not respond for conservative management of threatened preterm and went into preterm labour and delivered a single, still born male of 2.75 kg. No visible gross anamolies. All resuscitative efforts were unsuccessful. The postnatal period was uneventful.

**Table 1: Evaluation and management of parvovirus B19 infection in pregnancy.**

| Serological testing results | Interpretation | Management |
|---|---|---|
| **IgG+, IgM-** | Past infection, immune | Reassure patient that she is immune to infection |
| **IgG-, IgM-** | No past infection, non-immune | Repeat test in 2-4 weeks |
| **IgG+, IgM+** | Recent infection | Requires fetal evaluatiom |
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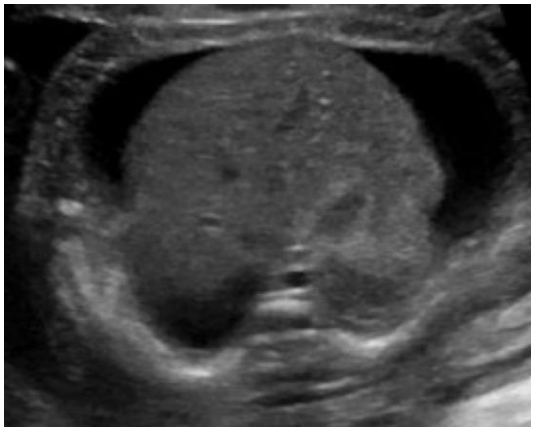

**Figure 1: Obstetric scan showing fetal ascites.**

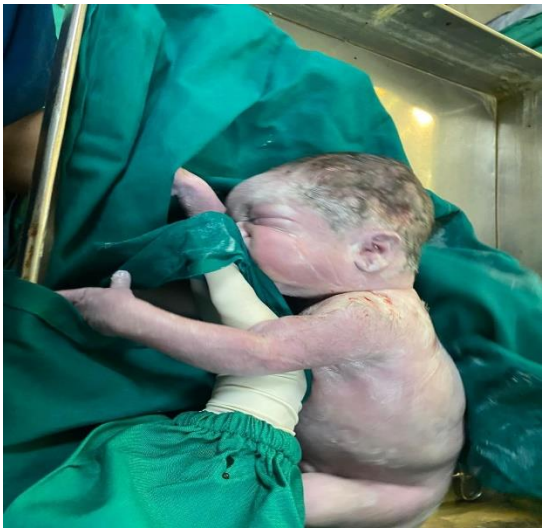

**Figure 2: Hydrops baby born at 33 wog.**

**Figure 3: Resuscitative efforts in still born hydrops baby.**

## DISCUSSION

Parvovirus B19 is a small, single stranded DNA virus belonging to *Parvoviridae* family.[2] It mainly replicates in erythroid precursors, myocardial and endothelial cells.[4]

Maternal infections usually occurs through respiratory droplets, hand to mouth contact.[5]

Maternal viremia reaches its peak 7 days after infection and IgM antibodies are detected in maternal blood between 7-14 days and IgG between 14-21 days.[4]

In 20-30% cases, infection is asymptomatic and some may develop flu like symptoms.

Transplacental transmission occurs in 15% cases before 15 weeks of gestation and 25% cases between 15-20 weeks of gestation which increases to 70% towards term. The risk of fetal hydrops is greatest during 1st half of pregnancy (i.e., 3% <26 weeks compared with 1% >20 weeks).

Diagnosis is generally made by maternal serological testing for specific IgG and IgM.[6]

If IgM is positive, it indicates recent infection and fetal surveillance to be done with ultrasound and MCA PSV Doppler every 2 weeks for 10 weeks after infection. If sonography shows hydrops fetalis, hepatomegaly, splenomegaly, placentomegaly and elevated MCA PSV, cordocentesis should be done for CBC, reticulocyte count and parvovirus B19 DNA PCR. If required, intrauterine transfusion should be considered.

In neonates, infection can be confirmed by detection parvovirus B19 IgM in neonatal blood and virus isolation by PCR.

It has been suggested that neurodevelopment and psychomotor disability in congenitally infected fetus are associated with infection per se rather than degree of anemia.[4]

## CONCLUSION

Parvovirus B19 accounts for 8-10% cases of non-immune fetal hydrops. Major congenital anomalies are rare and there is no evidence that parvovirus B19 is a significant teratogen. Fetal infection with parvovirus B19 can cause severe anemia, hydrops fetalis, myocarditis, IUD, neurological and neurodevelopment sequelae. As it can cause severe morbidity and mortality, it should be a part of routine work up in pregnant women who have been exposed to virus or with suspected fetal hydrops. Depending on gestational age, induction of labour versus fetal blood sampling with intrauterine transfusion should be considered by a high-risk perinatal team.

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