The Effects of Parvovirus B19 Infection in Pregnancy and the Fetus

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This poster discusses the significance and clinical impact of Parvovirus B19 infection in pregnancy, and the antepartum management required to improve fetal outcomes. Parvovirus B19 infection in pregnancy can lead to fetal anemia, non-immune fetal hydrops and even fetal death (1). There are no prophylaxis and targeted treatments available against Parvovirus, however increased surveillance of confirmed maternal infections with potential intervention to reverse fetal anemia may reduce the overall mortality rate (1).

CASE

We present a case of a 29-year-old, gravida 2 para 0 who was initially referred to our Maternal Fetal Medicine service at 20 weeks for suspicion of fetal hydrops on her anatomy scan (around 19+4 weeks). Her first ultrasound with MFM showed significant ascites (AC > 99%), pericardial effusion, scalp and skin oedema, with an elevated MCA PSV. Given her ultrasound findings, the patient was counselled and advised regarding her options, and she consented for an amniocentesis and maternal serology testing.

Maternal serology showed a positive IgG, IgM and PCR for Parvovirus B19 infection. Given the significance of fetal ascites and anemia, the patient consented for a fetal intrauterine transfusion at 22+3 weeks. The pre-transfusion fetal Hb was 34 (MCA PSV 33cm/s, post transfusion fetal Hb was 54 (MCA PSC 17cm/s). She received a total of 18mls of intrauterine transfusion and a further 15mls of RBC into the fetal peritoneal cavity, with a follow up ultrasound in 1 week.

Her follow up ultrasound did not show a significant improvement in fetal ascites and anemia, hence the decision was made for a second intrauterine transfusion at 23+3 weeks. The pre-transfusion fetal Hb was now 70 (MCA PSV 34cm/s), and post transfusion fetal Hb was 94 (MCA PSV 32cm/s). She received 20mls of intrauterine transfusion.

The patient's subsequent ultrasounds showed a significant improvement in the fetal hydrops and ascites, with eventually a complete resolution of fetal hydrops. A fetal brain MRI was also organized around 30 weeks gestation which was normal. At 36 weeks, ultrasound showed an active fetus with a normally grown AC, EFW 2.14kg, MVP 4.1cm, normal Dopplers and MCA PSV 53cm/s.

She was induced for labour at 37+1 weeks and delivered the baby via non-elective caesarean section for suspected fetal distress at 6cm , with a postpartum hemorrhage of 1L. Both mother and baby have since fully recovered post operatively with no ongoing complications.

DISCUSSION

Parvovirus is a single-stranded DNA virus that selectively lyses human erythroblasts, causing erythema infectiosum (fifth disease). In immunocompromised individuals like pregnant women, there is high risk of mother-to-fetus transmission causing fetal anemia, fetal hydrops, spontaneous miscarriage, stillbirth (2). Chronic congenital anemia may also persist following an intrauterine transfusion for fetal hydrops (3). Maternal parvovirus infection has not shown to cause any specific increase incidence in teratogenicity or developmental abnormalities, nor any long-term consequences in otherwise normal fetuses (3); however severe anemia may potentially be a stand-alone risk factor for long-term neurologic sequealae (1).

Diagnosis of Parvovirus in pregnancy is determined by testing Parvovirus-specific IgG and IgM. In acute Parvovirus infections, viral DNA may be identified via PCR of amniotic fluid through amniocentesis or fetal blood through cordocentesis (1).

Management of fetal hydrops and Parvovirus infection in pregnancy should be undertaken in a tertiary care setting. Cordocentesis may be indicated to determine fetal hemoglobin and reticulocyte count, where intrauterine transfusion might be necessary to improve fetal anemia and outcomes (1).

REFERENCES

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