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Case Report

A rare case of maternal anti-m antibody causing autoimmune hemolytic anaemia in newborn

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ABSTRACT

Maternal antibodies against fetal red blood cells can cause hemolytic disease of the fetus and newborn (HDFN), leading to fetal anemia, fetal ascites (hydrops), and postnatal complications. While anti-D antibodies are the most common cause, other antibodies such as anti-M, anti-c, and anti-K1 can also be clinically significant. A 34-year-old woman (G2A1) with a prior second-trimester medical termination of pregnancy was found to have anti-M antibodies in her current pregnancy. Serial monitoring with blood tests and fetal Doppler scans indicated a risk of fetal anemia. Antenatal monitoring using ICT titers and MCA Doppler was performed for early detection of fetal complications. She delivered a preterm neonate (2.68 kg) who required NICU care, ventilation, phototherapy, and antigen-negative blood transfusions. Both mother and baby recovered well. Early detection of rare maternal antibodies, careful fetal surveillance, and planning for safe transfusions are essential for managing alloimmunized pregnancies. A multidisciplinary approach improves outcomes for both mother and newborn.

Keywords: Anti-M antibody, Maternal alloimmunization, Hemolytic disease of the fetus and newborn, Fetal anemia, Antigen-negative transfusion, NICU

INTRODUCTION

Autoimmune hemolytic anemia (AIHA) is a rare condition in which autoantibodies target red blood cell membrane antigens, causing premature destruction of red cells. The estimated incidence is approximately 1 in 80,000 live births.[1] AIHA and other immune-mediated hemolytic diseases can result in severe neonatal complications such as fetal anemia, hydrops fetalis, ascites, and perinatal morbidity. Recent reports also describe neonatal AIHA presenting with rapid hemoglobin decline, significant jaundice, and the need for early transfusion support, underscoring the severity of this condition when it occurs.[2]

Hemolytic disease of the fetus and newborn (HDFN) most commonly results from Rh incompatibility, particularly anti-D antibodies, which continue to be the leading cause of severe fetal anemia despite preventive immunoprophylaxis.[3] However, HDFN remains a significant contributor to perinatal morbidity even today, especially when caused by antibodies outside the Rh system.[4] An increasing number of cases are now being attributed to non-Rh antibodies, including Kell, Duffy, Kidd, and MNS blood group antibodies.[5]

Among these, anti-M antibodies are an uncommon but clinically significant cause of HDFN. Although often naturally occurring and IgM in nature, anti-M can sometimes include an IgG component capable of crossing the placenta, resulting in suppressed erythropoiesis, severe fetal anemia, and late-onset neonatal anemia.[6,7] Several reports have highlighted that anti-M–related HDFN may be more severe than previously assumed and may require transfusion support or early delivery.[6]

Given these risks, early antenatal screening, appropriate antibody identification, and serial monitoring—including ICT titers and Doppler assessment of the middle cerebral artery peak systolic velocity (MCA-PSV)—are essential for timely diagnosis and prevention of adverse fetal outcomes.[3]

CASE REPORT

A 34-year-old woman, gravida 2, abortion 1 (G2A1), with a history of second-trimester medical termination of pregnancy due to fetal anomaly, presented for routine antenatal evaluation.[2] Her blood group was A positive, while her husband's was B positive. Antenatal screening revealed a positive indirect Coombs test (ICT) with an initial titer of 1:32, which later decreased to 1:4. Peripheral smear showed macrocytic anemia with a normal reticulocyte count.

Serial fetal monitoring was performed using middle cerebral artery (MCA) Doppler and cerebroplacental ratio (CPR) to assess fetal anemia. Prophylactic antenatal corticosteroids were administered at 32 weeks. Due to abnormal MCA Doppler findings at 32 weeks, an emergency lower-segment cesarean section (LSCS) was performed.

She delivered a preterm neonate weighing 2.68 kg at 32 weeks. The infant required resuscitation for apnea and NICU admission. Laboratory evaluation showed anemia (Hb 7.6–9.8 g/dl), reticulocytosis (8.2%), and a peripheral smear revealing macrocytic and nucleated red blood cells with anisocytosis. Total bilirubin was initially elevated (2.3 mg/dl) but improved after phototherapy. The direct Coombs test was negative.

Management included mechanical ventilation, non-invasive positive pressure ventilation (NIPPV), phototherapy, transfusion of antigen-negative packed red blood cells (PRBCs), feeding support, and vitamin supplementation. The neonate gradually improved and was discharged in stable condition.

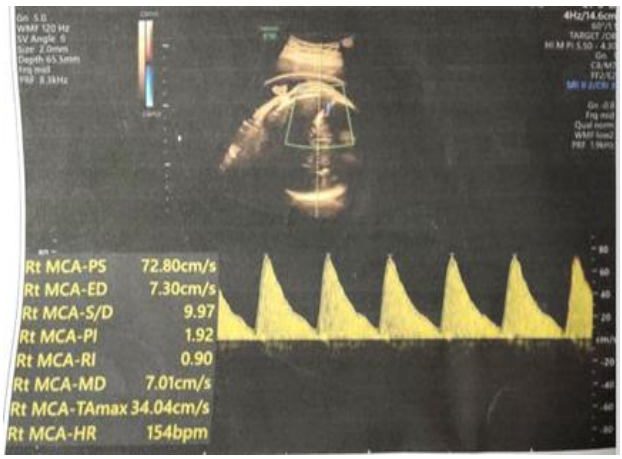

Figure 1: Abnormal MCA Doppler in the fetus.

DISCUSSION

This case highlights the clinical significance of maternal anti-M antibodies in causing hemolytic disease of the fetus and newborn (HDFN). Although anti-M is usually weakly clinically significant, its detection at 37°C warranted close antenatal monitoring in our patient. Fetal surveillance using MCA-PSV and cerebroplacental ratio (CPR) helped predict fetal anemia and guided the timing of delivery, consistent with established guidelines for alloimmunized pregnancies.[3,8] Neonatal management with antigen-negative PRBC transfusions prevented further hemolysis, in line with recommended transfusion practices for rare antibody-mediated HDFN.[5]

A case study conducted in Japan reported 34 babies with M/N-incompatible HDFN.[6] Among these, 21 of 29 survivors had severe hemolytic anemia and/or hydrops. Five infants were stillborn or died. Late-onset anemia requiring M-negative transfusion occurred in 14 infants.

Another study found that maternal anti-M antibodies caused HDFN and prolonged anemia in two siblings due to erythropoietic suppression.[7]

Similarly, a retrospective study from China on 17 infants with anti-M HDFN found that 14 required transfusion and one required exchange transfusion. Sixteen improved; one died.[8]

CONCLUSION

Early detection of rare blood groups which causes hemolytic disease of fetus and newborn using ICT test is necessary for early identification and detection of complications. Maternal anti-M alloimmunization can lead to significant fetal and neonatal morbidity. Multidisciplinary management ensures timely intervention and improves prognosis. Similar cases and clinical experiences have also been reported in the literature, including the studies referenced in items 8, further supporting the clinical significance of anti-M–mediated HDFN.

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