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A CASE REPORT ON FETOMATERNAL HEMORRHAGE INCITING AUTOIMMUNE HEMOLYTIC ANEMIA -FACTUAL OR NOT

KEY WORDS:

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ABSTRACT

Introduction: Non-D Rh alloimmunization is a relatively uncommon pregnancy complication, but it can cause mild to moderate Hemolytic Disease of Fetus and Newborn (HDFN). Red cell transfusion is the most important risk factor for non-D Rh alloimmunization, followed by parity, major surgeries, and hematological diseases. It is crucial to identify and address non-Rh D alloimmunization in all maternity care provision. **Case presentation:** A 20-year primigravida with 35 weeks 5 days of gestation married for 2 years in a 3rd degree consanguineous marriage k/c/o hypothyroidism with IUGR and oligohydramnios. She went into spontaneous labor at 37 weeks 2 days POG, and was taken up for emergency LSCS I/V/O Abruptio placenta. The patient presented as vertex and delivered an alive girl child of B.wt 2.04 kg with APGAR-5,7,9 for the first 1,5,10 minutes. Estimated blood loss was 1300 ml. **Discussion:** Massive fetomaternal hemorrhage is a rare and serious event that occurs in 15-20% of all pregnancies. Implicated antibodies may be naturally occurring or immune antibodies developed following sensitization. This hemolytic process results in fetal anemia and hyperbilirubinemia in 10% of the cases. An ABO incompatibility occurs more commonly in O group than group A or B individuals, and total pregnancy wastage is higher in ABO incompatible mating (24.59%) than compatible mating (8.45%).The placenta is thought to demonstrate gross and microscopic changes associated with FMH. Acute FMH may lead to hemodynamic changes in the placenta, such as thrombosis and vasoconstriction. Chronic FMH is often less visible on placental examination, and the precise roles of various placental lesions in FMH remain uncertain. **Conclusion:** Compound antibody screening positive (Anti CcE +ve) is a rare non-D Rh alloimmunization (11%), and there may be a chance of adverse perinatal, fetal, and neonatal outcomes in consecutive pregnancies. Antibody screening (IDCT) must be done as an essential test in all pregnant women, especially those who are Rh(D)positive and are erroneously regarded as not being at risk of isoimmuninzation and HDFN.

INTRODUCTION:

- Non-D Rh alloimmunization is relatively uncommon complication of pregnancy
- How ever non –D Rh antibodies causes mild to moderate Hemolytic Disease of Fetus and Newborn(HDFN)
- Red cell transfusion is most important risk factor for non –D Rh alloimmunization followed by parity, major surgeries and hematological diseases
- Non-Rh D alloimmunization therefore must be identified and if present acted upon by all involved in maternity care provision

Case Report:

A 20 yr primigravida with 35 weeks 5 days period of gestation married for 2 years in a 3rd degree consanguineous marriage. Her blood group was B positive. She is a K/C/O hypothiroidism with IUGR and oligohydramnios admitted in Dr.PSIMS & RF for safe institutional delivery. .At 32 weeks period of gestation symmetrical IUGR noted .No H/O blood transfusions prior.

Patient went into spontaneous labor at 37 weeks 2 days POG spontaneous rupture of membranes occurred immediately bloody amniotic fluid noted , liquor adequate, no cord prolapse, CTG reassuring.

Patient taken up for emergency LSCS I/V/O Abruptio placenta.

Preoperative Events:

Preoperatively coagulation profile, cross mating were done.Her Blood group was B+VE.Cross matching incompatibility noted with B+VE,O+VE,B-VE,O-VE, DCT-VE,IDCT+VE, Coagulation profile , BT,CT reports were normal. Hb 9.7gms, peripheral smear showed normocytic normochromic anemia Platelets were 2.6 lakhs, LFT, RFT, TC, DC, CUE reports are normal.

IOF: Blood stained liquor noted.No retro placental clots indicating reveled type of abruption. Baby presented as vertex and delivered an alive girl child of B.wt 2.04 kg with APGAR -5,7,9 for the first 1,5,10 minutes. Atonic PPH noted managed by medical and surgical methods. Estimated blood loss was 1300 ml.

Postoperative Events:

a) 1 Pint saline washed O-VE PRBC and 2 Pints B-VE FFP transfusion done I/V/O Hb 6.7 gm% On POD-1
b) Transfusion reaction(Fever,tachycardia) noted with 2nd time transfusion with saline washed O-VE PRBC .
c) Patient started on inj.Dexamethasone 4mg iv BD x5days from POD-1 changed to T.Prednisolone 15 mg BD for 1 wk
d) ANA profile negative, Anti TPO-38.24 IU/ml, reticount 2%,complement levels C3-126mg/dl,C4-62.9mg/dl. Patient blood sent for antibody screening test and showed DAT+VE,IgG (4+) reactive ,PAN positive for antibody screening Anti CcE +ve
e) Patient discharged on POD-10 in a stable condition.

f) Patient on follow up after 1 wk :DCT +ve ,IDCT + ve ,reticulocyte count 5% diagnosed as autoimmune hemolytic anemia and steroid dose increased T.Prednisolone 20mg ODx7days. Suggested review after 1wk

g) Baby BGT was B+VE DCT -VE ,Hb-13.4 gm%,and had normal blood picture

HPE Of Placenta: Tertiaryvilli with choriangiosis, multiple syncytial knots, focal fibrin degeneration all these features suggestive of ischemic changes of placenta

DISCUSSION:

- As a consequence of pregnancy, blood transfusion, or both, or other hematological conditions women will continue to make antibodies against blood group antigens other than D within the Rh system, and to antigens of many other blood group systems.[1]
- These other antibodies though rare, have a relatively increasing importance for the pregnant patient, her fetus, and the attending clinician.
- Massive fetomaternal hemorrhage is a rare and serious event.[2,3]
- Implicated Ab could be naturally occurring or immune antibodies developed following sensitization[4].
- This occurs in 15-20% of all pregnancies. This hemolytic process results in fetal anemia and hyperbilirubinemia in 10% of the cases[5].
- An ABO incompatibility occurs more commonly in O group than group A or B individuals. A fetal ABO incompatibility may lead to fetal hemolysis in the first pregnancy because of preexisting antibodies in mother from infancy[6].
- Most spontaneous massive FMHs occur unexpectedly and are clinically silent.
- The prenatal diagnosis of FMH is difficult, and studies have proved that the morbidity of FMH is highly underestimated because of the under utilization of confirmatory FMH test[7,8].
- Studies have reported a link between intrauterine growth restriction and massive FMH as in this case[9,10,11].
- The placenta is thought to demonstrate gross and microscopic changes that are associated with FMH[12,13].
- Acute FMH may lead to hemodynamics changes in the placenta, such as thrombosis and vasoconstriction[14,15].
- In contrast, chronic FMH is often less visible on placental examination, as the bleeding occurs over a longer period of time and may be more diffuse. In some cases, the placenta may become swollen and edematous, resulting in increased placental weight[15]
- Acute FMH was associated with chorangiosis, which is characterized by vascular proliferation in the placental villi[16]
- In contrast, chronic FMH was associated with villous fibrosis and decreased villous vascularity. the degree of villous fibrosis was significantly higher in cases of severe FMH compared to mild or moderate cases[17,18]
- In this case we saw increased syncytial knot density, a factor whose pathophysiological mechanism in FMH has not yet been specifically described.[19,20]
- The presence of syncytial knots was considered as a response to trophoblastic ischemia that in turn causes impaired maintenance of fetomaternal exchange and contributes to intrauterine fetal distress[19,20]
- The precise roles of the various placental lesions in FMH remain uncertain, and most of these lesions may not necessarily be considered pathognomonic of FMH[19,20].
- However, some studies have reported that most cases of massive FMH occur in the absence of any clinical risk factors. The placentas of women with massive FMH show no pathologic findings on either gross or microscopic examination[19,20].

CONCLUSION:

- This case has shown compound antibody screening positive (Anti CcE +ve) rare non-D Rh alloimmunisation (11%).
- There may be chance of adverse perinatal, fetal and neonatal outcome in consecutive pregnancies.
- Antibody screening (IDCT) must be done as an basic test in all pregnant women with this level of clinical alertness especially with those who are Rh(D)positive and who are erroneously regarded as not being at risk of isoimmuninzation and therefore of HDFN.

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