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ISSN: 2362-0080

Category: Case Report

A Case of Neonatal Anaemia due to Feto-maternal Hemorrhage

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ARTICLE DETAILS

Article History

Published Online:

30th December 2020

Keywords

Neonatal anaemia, Feto maternal haemorrhage

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ABSTRACT

Anemia in neonates is defined as central or venous hemoglobin < 13 g/dl or capillary hemoglobin < 14.5 g/dl in infants >34 weeks and 0-28 days old. Hemorrhagic anemia can happen during ante partum period, intra partum period, or post-partum period. Fetalmaternal hemorrhage (FMH) was responsible for nearly 14% of unexplained fetal deaths and 3% of all fetal deaths. We present a rare case of neonatal anaemia due to FMH, who survived with careful management.

1. Introduction

Anemia in neonates is defined as hemoglobin less than 13 g/dl in central or venous blood or less than 14.5 g/dl in capillary blood of an infant (aged >34 weeks of gestation and 0-28 days old). Anemia in the newborn results from three processes. They are hemorrhagic anemia (the loss of RBC) which is the commonest, hemolytic anemia (increased destruction of RBC) and hypoplasticanemia (underproduction of RBC) [1]. Hemorrhagic anemia can occur either during ante-partum (eg. twin-twin transfusion, placenta previa), or intra-partum (eg. Feto Maternal Hemorrhage FMH, traumatic rupture of the cord) or during postpartum period (eg. enclosed hemorrhage, defects in hemostasis) [1]. Nearly in all pregnancies, some fetal red cells cross into the maternal circulation. The estimated incidence of FMH varies from 2 to 75 % of pregnancies [2] with the hemorrhage in some cases being large enough to compromise the fetus, resulting in loss of fetus, stillbirth or delivery of a severely anemic infant. FMH is known to be responsible for nearly 14% of unexplained fetal deaths and 3% of all fetal deaths [3]. However, owing to its relative rarity, there is a paucity of cases of neonatal anemia due to FMH in medical literature. Here we report a case of neonatal anemia due to massive FMH.

2. Case Report

A female infant was born to non-consanguineous parents at term via an assisted vaginal delivery (vacuum delivery) due to fetal distress with the birth weight of 2.8 kg. There was no history of placental abruption or ante/ perinatal bleeding or other antenatal complications. APGAR was good. The placenta was delivered completely and no evidence of hemorrhage. No trauma to umbilical cord or to placenta during delivery. This is the fourth pregnancy with three healthy children. Past obstetric history was not significant, and no family history of hemoglobinopathies other or hematological conditions were present.

Only abnormality detected was the pallor. As initial management baby was kept under the warmer and observed for 1 hour. But the color was not improved and had persistent pallor. On further examination baby did not have dysmorphic features/ skeletal anomalies, cephal/ caput/ subgaleal hematoma, skin rashes, evidence of frank bleeding manifestations or icterius. Cardiovascular system examination showed HR of 160/min, with soft systolic murmur, normal femoral pulses with no evidence of acute heart failure. Hepatosplenomegaly was absent.

Basic investigations revealed Hb- 4.8, MCV-112.5, MCH-30.1, MCHC-26.9, RDW-24.8% and RBC-1.59 X 10⁶. Blood picture showed normochromic cells with microcytic, hypochromic

cells with spherocyes, tear drop cells, target cells, acanthocytes and polychromatic cells with normal WBC and platelets.

Retic count was 4.8%. Both mother's and baby's blood groups were B+ and DAT was negative. Initial septic screening, serum bilirubin and clotting profiles were normal. USS brain and abdomen were normal.

Kleihauer–Betke (KB) test performed on the mother's blood shortly after delivery showed 2.05% (7500 cells in 50 low power field) fetal red cells in the maternal circulation, a volume larger than the expected total blood volume for an infant of this size.

Baby was transfused with group compatible blood slowly and while monitoring evidence of heart failure.

3. Discussion:

Manifestations of FMH depend on the magnitude and the acuity of blood loss [4]. If the fetus can compensate for the blood loss, the pregnancy may continue to delivery of an infant with varying degrees of anemia [5]. In the present case, pregnancy continued, and the baby had no evidence of complications such as heart failure. The only abnormality detected was pallor which, however, did not improve with initial management. Clinical suspicion of the possibility of FMH giving rise to neonatal anemia, in the absence of other common causes, has led to the early and appropriate management of this patient, giving rise to a favorable outcome. As in this case, the etiology of FMH is unclear and it is difficult to predict the risk in pregnancies. Furthermore, planning of preventive measures is also impractical. Early diagnosis of anemia due to possible FMH, and prompt management with volume replacement and correction of anemia is required to save the lives of affected newborns as was done in this case.

Acknowledgement

The parents of the neonate who gave consent for reporting this case as an anonymous report and the staff of Lady Ridgway Hospital, Colombo, Sri Lanka are acknowledged.

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