Sri Lanka Journal of Medicine **SLJM**

Case Report

Citation: Arunath V, Mohamed MYN & Thanshayan B, et al., 2021. Fetomaternal hemorrhage causing severe anemia in a neonate. Sri Lanka Journal of Medicine, pp 112-116.
DOI: http://doi.org/10.4038/sljm.v30i2.270

Foetomaternal haemorrhage causing severe anaemia in a neonate

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Abstract

Case report: A baby girl, born at term with a birth weight of 2165 grams by emergency caesarian section was pale and developed grunting at birth with oxygen saturation of 90% at 10 minutes. Haemoglobin on day 1 was 7.7g/dL. Blood film showed evidence of acute blood loss. Kleihauer–Betke test performed on mother's blood was positive. Baby was transfused with packed red cells and started on haematinics.

Discussion: Foetomaternal haemmorhage occurs when fetal blood enters maternal circulation. It may be large enough to cause severe anemia in the neonatal period and should be considered while evaluating a neonate with anemia.

Keywords: Foetomaternal hemorrhage, Anemia, Kleihauer–Betke test

INTRODUCTION

Fetomaternal hemorrhage (FMH) occurs when fetal blood enters maternal circulation before or during delivery. An insignificant hemorrhage of fetal blood into maternal circulation is a common obstetrical event in nearly all pregnancies (1,2). However, very rarely, FMH may be large enough to compromise the fetus, resulting in fetal demise, stillbirth or severe anemia in the neonatal period (1). We report a baby girl who developed severe anemia on day one of life following FMH.

CASE REPORT

A baby girl, 1st born child of non-consanguineous parents, was born at 40 weeks of gestation by emergency lower segment caesarian section due to fetal distress. Her birth weight was 2165grams. APGAR scores were 10 at 1, 5 and 10 minutes of age. Liquor was meconium stained.

Mother had an uncomplicated antenatal history except for the fundus less than dates, which was identified at 34 weeks of gestation. Unfortunately, mother defaulted the clinic follow up due to the pandemic and got admitted at 40 weeks of gestation, due to reduced fetal movements for 2 days. On admission, fetal cardiotocography was

Received: 2021-08-21 **Accepted revised version: 2021-11-11** **Published: 2022-01-25**

pathological (Figure 1) and the middle cerebral artery pulsatility index was 0.82. Emergency caesarian section was performed immediately. Macroscopic examination of placenta did not reveal a retroplacental clot or any other abnormality.

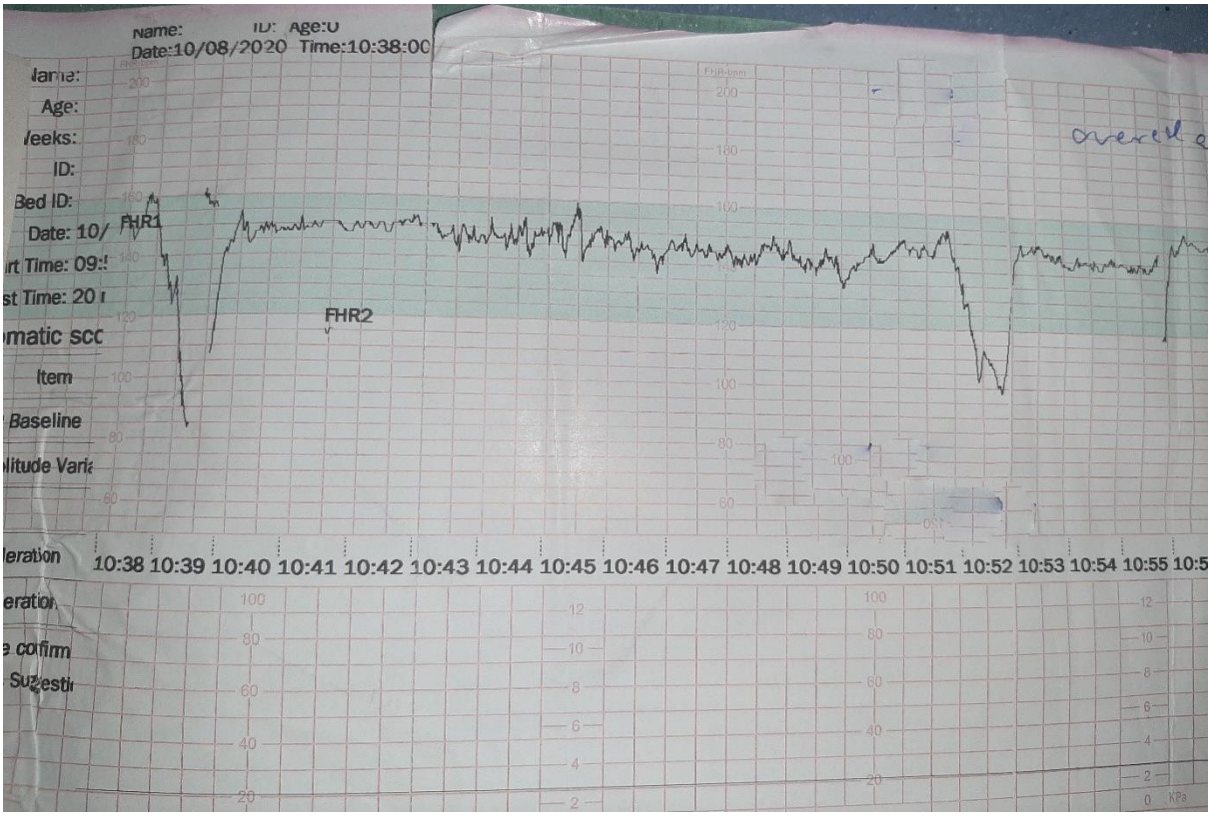

Figure 1: Fetal cardiotocography of the mother at 40 weeks of gestation showed decelerations

The baby developed grunting at the time of birth and respiratory rate was 58 breaths per minute. She was pale with a heart rate of 148/min and oxygen saturation at 10 minutes of age was 90%. A grade 3 systolic murmur was heard, best in the pulmonary area. Clinically there was no cardiomegaly. Blood pressure was 68/41mmHg with a mean of 41mmHg. Rest of the neonatal examination was unremarkable.

Complete blood count of the baby on day 1 revealed; haemoglobin - 7.7g/dL (14-22), white cell count- 17,300/mm^3 (N-70%, L-22%), platelets- 123,000/mm^3 and 70% of nucleated red cells. Blood film showed evidence of blood loss without evidence of hemolysis. Reticulocyte count was 30.5%. Venous blood gas analysis revealed pH 7.24 (7.31–7.41), pCO2 34 mmHg (30–40), HCO^{3-} 15.8 mmol/L (22–29), and lactate 10 mmol/L (8–16), suggestive of metabolic acidosis. Coagulation screen was unremarkable. Aspartate transaminase, alanine transaminase and serum creatinine were 177U/L (35-140), 44.2U/L (4 – 41) and 100.9µmol/L (26.5-106) respectively. Blood groups of both the mother and the baby was AB positive and direct antiglobulin (Coombs) test was negative.

Kleihauer–Betke(KB) test performed on the mother's blood shortly after the delivery was positive, suggestive of a FMH of around 75ml (Figure 2). The baby was transfused with 10ml/kg of packed red cells on day 1. Haemoglobin of the neonate was 11g/dl on Day 6 of life. The baby was discharged on Day 6 of life with oral iron and folate supplementation and is being followed up in the neonatal clinic.

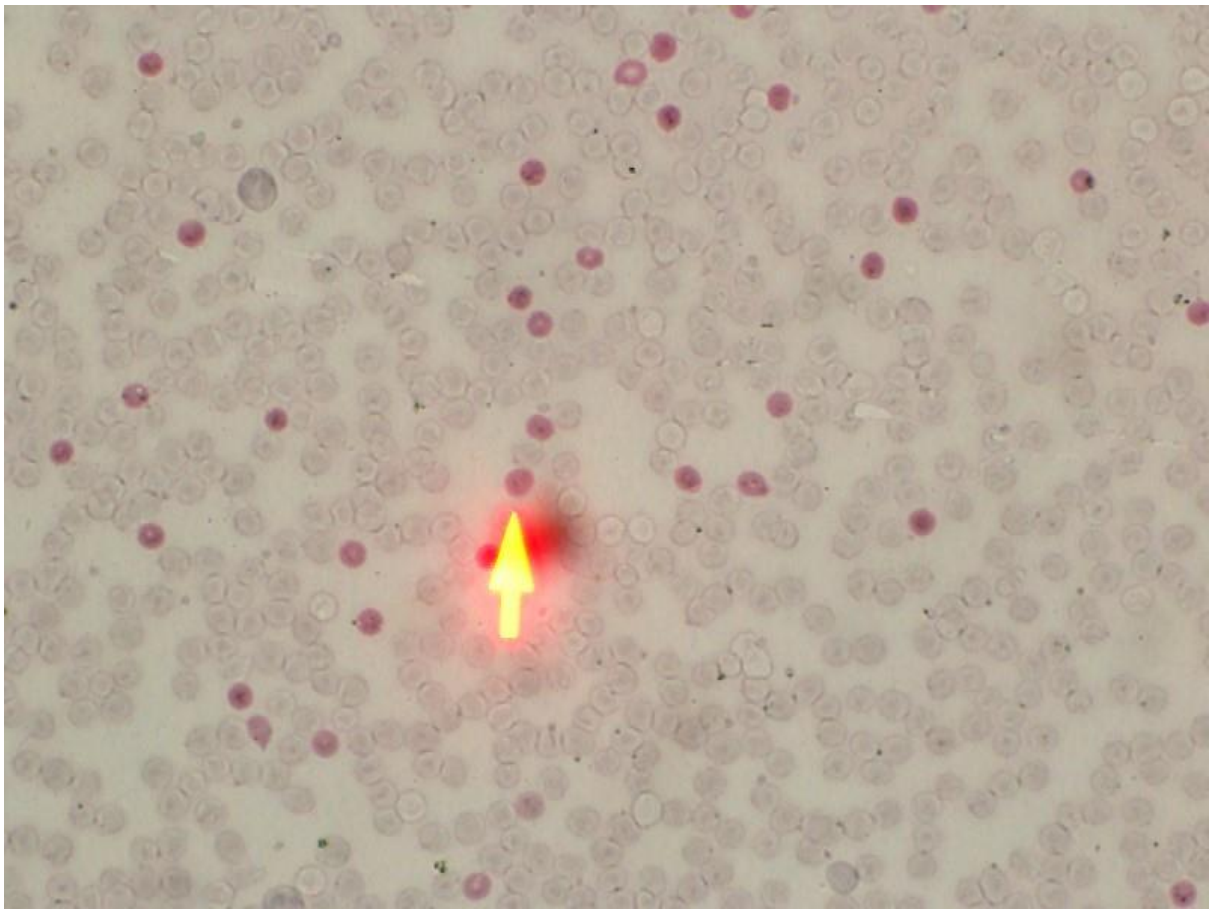

Figure 2: The microscopical Kleihauer-Betke acid elution test performed in mother showed cherry-red fetal cells (yellow arrow) and in the background maternal cells appear as uncolored "ghost cells".

DISCUSSION

FMH physiologically occurs as early as 4 weeks of gestation if there is disruption of the feto-maternal barrier (2-4). A FMH volume >30ml is infrequent and a massive FMH (>150 ml) is rare (1-3). However, the minimum volume which can cause clinically significant FMH is controversial (1,2,5).

Significant FMH is usually associated with antenatal abdominal trauma, obstetric procedures (amniocentesis, cesarean section) and fetal or placental complications (fetal demise, abruptio placentae). In many cases (82%), the cause of FMH is idiopathic without identifiable inciting event (1-4,7-10). Etiology in our patient was unclear. It may be related to silent placental abruption or idiopathic.

The initial symptoms and signs of FMH are often nonspecific. Most are diagnosed retrospectively after an infant is stillborn or is born with symptoms of hemorrhage. The initial and most significant warning sign is the reduction in fetal movement. Fetal heart rate monitoring may show a sinusoidal pattern, a lack of acceleration, and recurrent late decelerations. Fetal growth restriction may be the presenting symptom in a few where FMH has taken place chronically. Neonatal anemia has been reported to be the presenting sign in one third of the cases (2,4,7-10). In cases of FMH, placental pathology can show erythroblastosis in villi, which is a sign of significant fetal anemia and placental clots due to the activation of maternal clotting which limits the effect of the hemorrhage (2,7). Unfortunately, we were unable to perform the histopathological examination of placenta as FMH was suspected few hours after the delivery and at which point the placenta was already discarded.

When a FMH is suspected, maternal blood can be checked for the presence of fetal red blood cells by KB test or flow cytometry. The KB acid elution test is used to quantify fetal erythrocytes in the circulation of the mother. Adult hemoglobin in maternal red cells is eluted in acidic solution, and maternal erythrocytes appear pale, whereas fetal red cells stain bright pink as fetal hemoglobin is relatively resistant to acid (1-7). Flow cytometry is an alternative quantitative test and less labor intensive but requires specific equipment. It has a better precision than KB test but it may not be widely available. Therefore, KB test is still the standard test to diagnose this condition (1,2,7-10). Although our case is a typical example of how in-utero events can lead to a series of devastating effects, FMH was not immediately suspected at the time of admission, because the pregnancy had been normal except for fundus less than the dates until she presented with decreased fetal movements. The fetal heart tracing showed decelerations which was a sign of a distressed fetus and which led to an emergency cesarean section. The infant was recognized to be pale immediately after birth and showed evidence of hypoxic injury as evident by high lactate, deranged liver function tests and metabolic acidosis which improved with blood transfusion. There was an elevated nucleated red blood cell count and reticulocyte count, consistent with increased hematopoiesis for a prolonged period and in utero bone marrow activation caused by chronic anemia. The exact time of FMH, in our case cannot be estimated but hematological parameters were suggestive of a relatively subacute or chronic onset, which caused fetal compromise just at the end of pregnancy.

FMH is a rare condition but its actual incidence is probably underreported unless routine Hb estimation of cord blood is carried out. In majority of the cases no risk factors are found and unfortunately the clinical findings are also nonspecific and not enough to make the diagnosis before delivery. A high degree of suspicion of FMH in hypoxic neonates should be a routine in neonatal care.

Author declaration

Acknowledgements

We acknowledge all the staff who contributed in the management of this child including Professorial Obstetrics & Neonatology Units of De Soysa Hospital for Women and Department of Haematology, De Soysa Hospital for Women.

Authors' contributions

All authors (VA, MYNM, BT, VGQ, AKPR, AW and MNL) contributed in the management of the patient. VA involved in writing the manuscript. Final editing was done by AKPR, AW and MNL. All authors read and approved the final manuscript.

Conflict of Interests: The authors declare that they have no conflict of interests.

Funding: Not applicable

Availability of data and materials: Not applicable

Ethical approval and consent to participate: Not applicable

Consent for publication: Written informed consent was obtained from the patient's parents for publication of this case report.

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