Fetomaternal Transfusion as a Cause Of Severe Fetal Anemia Causing Early Neonatal Death: A Case Report

Masood Ahmed, Mohammad Abdullatif

Abstract

Fetomaternal hemorrhage refers to the entry of fetal blood into the maternal circulation before or during delivery. Very small amount of fetal red cells are normally detectable in all pregnancies. Massive fetomaternal bleed is very rare and even rarer is the resultant severe anemia causing early neonatal death, despite an uneventful normal pregnancy until the end. Antenatal fetomaternal hemorrhage is a pathological condition with a wide spectrum of clinical variation. Secondary to the resultant anemia, fetomaternal hemorrhage may have devastating consequences for the fetus such as neurologic injury, stillbirth, or neonatal death. The Presentation is frequently without an evident precipitating factor. Recognition may become apparent only after injury has occurred, if at all. The most common antenatal presentation is decreased fetal activity and a heightened index of suspicion is warranted in cases of persistent maternal perception of decreased fetal movements.

Keywords: Fetomaternal hemorrhage (FMH); Fetal anemia; Hydrops fetalis; Kleihauer-Betke test (KBT); Cardiotocograph (CTG).

Introduction

Small amount (<0.1 ml) of fetal blood is commonly found in maternal circulation. Massive fetomaternal hemorrhage (FMH) involves fetal blood loss into the maternal circulation of more than 150 ml or otherwise more than half the fetal blood volume. Large bleeds can be a cause of intrauterine death in up to 0.04% of all births. Most cases of acute and chronic fetomaternal hemorrhage are idiopathic in origin, most often spontaneous and involve uncomplicated near term pregnancies. We report a case of severe and fatal fetomaternal transfusion, which was proved by Kleihauer-Betke test. The purpose of this report is to make others aware of this rare but potentially fatal condition.

Case Report

A 37-year-old female in her third pregnancy at 35 weeks gestation by menstrual dates presented to her obstetrician with markedly decreased fetal movements. There was no history of pain, trauma or vaginal bleeding. The patient was known to be Rhesus positive. Past obstetric history revealed a normal delivery at term in her first two pregnancies. Current pregnancy was uneventful until 2 days before delivery when she started to feel reduced fetal movements.

On arrival at the delivery unit, cardiotocograph (CTG) showed reduced beat to beat variability with variable deceleration for which the mother underwent emergency lower segment cesarean section (LSCS) at 35 weeks of gestation. Outcome was a baby girl. Apgar scores were 2 and 3 at 1 and 5 minute respectively.

On examination, the baby was markedly pale with respiratory depression not responding to bag and mask ventilation for which the baby was immediately intubated and connected to mechanical ventilation. The liver was palpable 4 cms below the right costal margin (suggestive of congestive cardiac failure). The spleen was not palpable, and no petechiae was noted. Birth weight was 2.8 kg. Subcutaneous edema was also noted. Cord Blood gas Ph: 7.063; HCO3: 14.2; base excess was -12.2; blood sugar: 2 mol; and lactate: 15 mmol.

Our initial impression was severe fetal anemia leading to birth asphyxia. The cause of fetal anemia was most likely to be due to fetomaternal hemorrhage. Hematological results were as follow: Anemia with higher reticulocyte counts was suggestive of chronicity of in utero bleed. (Table 1)

<table>
<thead>
<tr>
<th>Parameter</th>
<th>HB (g/dl)</th>
<th>Hematocrit (%)</th>
<th>Retics (%)</th>
<th>Platelets (10^9/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cord Blood</td>
<td>3.2</td>
<td>10</td>
<td>Not done</td>
<td>110</td>
</tr>
<tr>
<td>At 6 hrs.</td>
<td>7.3</td>
<td>22</td>
<td>8.6</td>
<td>113</td>
</tr>
<tr>
<td>At 24 hrs.</td>
<td>10.1</td>
<td>31</td>
<td>6.0</td>
<td>82</td>
</tr>
</tbody>
</table>

There was evidence of multiorgan involvement in the form of deranged coagulation (Table 2), markedly deranged liver function test (Table 3), and marked hematuria (+++ on urine dipstick examination), which were consistent with severe degree of birth asphyxia affecting more than one organ systems.

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Table 2: Coagulation profile.

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prothrombin Time</td>
<td>36.8 sec</td>
<td>9.9 – 12.4</td>
</tr>
<tr>
<td>Activated Partial Thromboplastin Time</td>
<td>61 sec</td>
<td>31 - 55</td>
</tr>
<tr>
<td>INR</td>
<td>3.31</td>
<td>0.91 – 1.08</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>0.9 g/L</td>
<td>1.7 – 3.6</td>
</tr>
</tbody>
</table>

Table 3: Liver function tests.

<table>
<thead>
<tr>
<th>Test</th>
<th>Value</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alanine aminotransferase</td>
<td>333 IU/L</td>
<td>0 - 31</td>
</tr>
<tr>
<td>Aspartate aminotransferase</td>
<td>1970 U/L</td>
<td>0 - 32</td>
</tr>
<tr>
<td>Albumin</td>
<td>16 g/L</td>
<td>28 - 44</td>
</tr>
<tr>
<td>Total protein</td>
<td>25 g/L</td>
<td>46 - 70</td>
</tr>
</tbody>
</table>

Cranial ultrasonography showed grade 1 Intraventricular hemorrhage (Figs. 1 & 2), which was most likely relate to severe birth asphyxia. Right sided pleural effusion and ascites were also confirmed by ultrasonography. (Fig. 3)

Figure 1: Cranial US, sagittal view showing Intraventricular hemorrhage (arrow).

Figure 2: Cranial US, coronal view showing bilateral Intraventricular hemorrhage (arrows).

Figure 3: Ultrasound showing mild peritoneal fluid (arrow above) and pleural fluid (arrow below).

The presence of subcutaneous edema and fluid in two body cavities (peritoneal and pleural) were suggestive of hydrops fetalis. The baby required high ventilatory settings and 100% oxygen with oxygen saturations around 80%. Blood pressure was irrecordable with refractory hypotension due to blood loss. Two boluses of saline, each 10 ml per kg were given as well as packed red blood transfusion and inotropic support with dopamine; dobutamine and adrenaline were administered without any improvement in clinical condition.

The Kleihauer-Betke test (KB test) was done on the mother’s blood, which revealed that maternal circulation had 6% of fetal blood, which is nearly 140 ml of fetal blood. This confirms a very large amount of fetomaternal hemorrhage, reaching up to 50 ml/kg of fetal blood. When the transfused volume exceeds or equals 20 ml/kg, massive fetomaternal hemorrhage may lead to severe prenatal or neonatal complications including early neonatal death.

Inspite of extensive supportive measures being taken, the baby’s condition continued to deteriorate and the baby expired at 28 hrs of life. The final impression in our case was chronic fetomaternal hemorrhage leading to severe anemia causing birth asphyxia and non-immune hydrops.

Discussion

Fetomaternal hemorrhage can begin any time from the mid-first trimester onwards. The causative factor is usually a breach in the integrity of the placental circulation. The presence of fetal red cells in the maternal circulation is not abnormal. By term, 50% of mothers will have detectable fetal red cells. Though 96-98% of pregnancies have very small leaks of up to 2 ml.6

There is no universally accepted definition of the volume of fetal erythrocytes in the maternal circulation that constitutes a massive FMH; volumes of 10 to 150 mL have been proposed. FMH greater than 80 mL and greater than 150 mL is estimated to occur in 1 in 1000 deliveries and 1 in 5000 deliveries, respectively. Various studies have described the incidence of FMH >20-30 ml at delivery to be about 1 in 200-300 deliveries.4,7,8

Massive fetomaternal hemorrhage (>150 ml) [in this case 140 ml] occurs in 0.12 to 0.5% of pregnancies.9 A standard approach
to assess the severity of FMH is to estimate the percentage of the fetal blood volume represented by the FMH. A FMH of 20 mL/kg, which represents 20% of the fetoplacental blood volume is considered massive because it is associated with significant fetal/neonatal morbidity or mortality.

Other associated factors include: direct trauma to the abdomen, motor vehicle accident, abruptio placentae, vasa previa with membranous insertion, choriocarcinoma, amniocentesis, chorionic villous sampling and external cephalic version.

In this case, placental analysis revealed umbilical cord with 3 blood vessels and no funisitis. The membranes have no chorioamnionitis. Placenta shows multiple chorionic villi with no blood vessels and no funisitis. The membranes have no chorionic villous sampling and external cephalic version.

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References