

Case Report

A Neonate with Severe Anemia

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Abstract

We presented a case of male neonate, term baby 39+5 weeks of gestation with birth weight 2970g and Apgar scores were 2, 5 and 6 at 1, 5 and 10 minute respectively. His early day of life was complicated with severe anemia, perinatal asphyxia and multiple organ failure. Following C-section delivery, he presented with hypovolemic shock, congestive heart failure and transient myocardial injury, hypoxic-ischaemic encephalopathy, acute hypoxic injury to the liver and kidneys. The clinical diagnosis was massive fetomaternal hemorrhage (FMH). After intensive medical care, he was discharged without significant complication and major short-term neurological sequelae.

Introduction

Nearly all pregnancies in late gestation have fetal red cells in the maternal circulation. Fortunately, the volume lost is too small. However, in some cases massive fetomaternal hemorrhage is enough to compromise the fetus, resulting in fetal demise, stillbirth, severe anemia causing early neonatal death, despite an uneventful normal pregnancy. We report a newborn with severe anemia who was delivered in our hospital.

Case Report

A male neonate weighing 2970g was born by urgent cesarean section at 39+5 weeks of gestation. The mother, a 30-year-old woman, presented to our hospital obstetric emergency unit with markedly decreased fetal movement. There was no history of pain, trauma or vaginal bleeding. She had her regular prenatal care in health center since first trimester and later by obstetrician since 3rd trimester. She was found to have gestational diabetes that was well controlled without medication treatment. Her blood analysis was unremarkable without significant anemia during the whole pregnancy. Her blood group was O RhD positive. Current pregnancy was quite uneventful, except that the placenta was low lying with one episode of threatened premature labor in 29+6 weeks gestation, with hospitalization for several days then discharge uneventfully. The third trimester ultrasound at 33 weeks of gestation showed normal fetus and placenta position. The last ultrasound at 39 weeks of gestation, showed body weight 3061g, with normal amniotic fluid index and placenta, Doppler of UTA within normal range. Routine cardiotocography (CTG) showed reactive fetal heart rhythm at 38 and 39 weeks of gestation. One day before delivery she started to feel reduced fetal movement.

On arrival at the obstetric emergency unit, mother vital signs were stable. Physical examination was unremarkable. No vaginal

bleeding was found. CTG showed continuous sinusoidal heart rate pattern and flattening of the tracing even after multiple stimulations. Rupture of membranes occurred at delivery, with thick meconium stained amniotic fluid, stage II. The placenta and the cord were normal appearance. Outcome was a baby boy requiring aggressive resuscitation immediately after birth due to floppiness appearance, marked pallor without respiratory movement and no heart beat. Meconium stained liquid inside oral cavity, nasal cavity and near whole body were noted. After immediate resuscitation, Apgar scores were 2, 5 and 6 at 1, 5 and 10 minute respectively.

The baby was transferred to neonatal intensive care unit for further evaluation and connected to mechanical ventilation. On admission, his vital signs were: blood pressure 43/17mmHg, heart rate 149 bpm, temperature 36 degrees Celsius, respiratory rate 40 bpm on conventional mechanical ventilation with around 40% oxygen, SPO₂ 100%. Physical examination revealed very pale infant with little spontaneous movement and respiratory effort. Poor perfusion was noted with delayed capillary refill, equal but weak peripheral pulses. The liver was palpated 4cm below the right costal margin (suggestive of congestive cardiac failure). The spleen was not palpable, and no petechiae noted. No evidence of peripheral edema or hydrops noted. Peripheral venous catheters were placed and a bolus of normal saline was administered.

Initial blood gas at around 30 minutes of life revealed severe metabolic acidosis, pH 7.019; PCO₂ 40.4mmHg; base excess was -21mmol/L; HCO₃ 10.4mmol/L; hemoglobin beyond the limit of the point-of-care analysis equipment, blood glucose 0.9mmol/L. Immediate glucose administration and infusion 8.4% sodium bicarbonate two doses in 7 hours interval then eventually the pH increased to 7.29 at 10 hours of age and 7.31 at 17 hours of age. Blood glucose remained stable.

A complete blood count revealed that hemoglobin was 2.7g/dL, hematocrit 10.1% at around 1 hour of age. An immediate

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transfusion of two packs O-Positive red blood cells (15ml/kg) was given. Repeat blood test shown hemoglobin 10g/dL and hematocrit 32.4% at around 20 hours of age. Later hemoglobin was stable around 14g/dL without further blood transfusion. Direct coombs test was negative. There was no significant jaundice and not need phototherapy. Urine test for urobilinogen and bilirubin were normal.

The white blood cell count was $19.2 \times 10^9/L$ and platelet was 16400/uL. Sepsis evaluation and empiric antibiotics were started. TORCH (toxoplasmosis, rubella, cytomegalovirus and herpes) titers were obtained and were later noted to be normal. Blood culture showed no growth. The placenta was sent for pathological evaluation and no significant abnormalities were detected.

Brain ultrasound on 1 day of age found no intraventricular hemorrhage (IVH) but signs suspected hypoxic-ischaemic encephalopathy (HIE) in early stage. At 2 day of age, the neonate demonstrated abnormal movement, which were suggestive of seizures, and was started Phenobarbital and midazolam. Seizure under controlled and keep on low dose of oral Phenobarbital. Performed brain MRI on 10 day of age showed bilateral and symmetrical neonatal hypoxic ischaemic injury probably related with watershed predominant pattern of injury. Diffuse abnormality of the sub cortical white matter was noted in the cerebral hemispheres bilaterally. Restricted diffusion was noted posterior in the splenium of corpus callosum and in the parietal and occipital regions. HIE was evident without IVH and hydrocephalus.

There were biochemical markers of hypoxic injury to the liver and kidneys. Abnormal aspartate aminotransferase and alanine aminotransferase increase to 10 to 40 folds of normal upper limit respectively. Under supportive management, liver function gradually returns back to normal range. Acute renal functional failure was noted as increased creatinine with general subcutaneous edema, urine output decrease, microscopic hematuria, which responded well to fluid restriction and furosemide administration.

Congestive heart failure and transient myocardial injury were evident as tachypnea, tachycardia, hepatomegaly, as well as Troponin T elevated to 136 to 184.2pg/ml in first 4 days of life with significant CK and CK-MB elevation over 500 hundreds to thousands. Supportive with mechanical ventilation, as well as correction of metabolic abnormalities and fluid restriction and diuretics were effective without inotropic agent needed in our case.

The neonate was weaned from respiratory support and exubated at 46 hours of age. Follow extubation, he required continuous positive airway pressure (CPAP) ventilation support until 5 days of age. Since then, his respiratory condition was stable in room air. He started nasogastric tube feeding on 3 days of age and initially some feeding difficulties in first week of life, later good sucking by mouth. Over the course of his stay in the neonatal intensive care unit, his body temperature was stable and normal. He was discharge to home on day 15 with oral Phenobarbital.

Discussion

The ability of fetal red cells to cross into maternal circulation was first hypothesized by Wiener in 1948 [1] and was later confirmed as a cause of neonatal anemia by Chown in 1954 [2]. There is no universally accepted definition of the volume of fetal erythrocytes in the maternal circulation that constitutes a massive fetomaternal hemorrhage (FMH). Katiyar R. reported that involves fetal blood loss into the maternal circulation of more than 150 ml or otherwise more than half the fetal blood volume [3]. At this cut point, FMH occurs in 0.12 to 0.5% of pregnancies [4].

The initial symptoms of an acute FMH are often subtle and nonspecific. Most are diagnosed retrospectively after an infant is stillborn, experiences unexplained fetal distress or is born with symptoms consistent with a hemorrhage [5]. Prenatally, the mother may present with a history of decreased or absent fetal movement [6]. The sinusoidal heart rate pattern, with or without decreased fetal movement, is a common presenting sign in published reports of FMH [4, 7, 8, 9].

In our case, the initial impression was severe fetal anemia leading to perinatal asphyxia. The cause of fetal anemia was most likely due to fetomaternal hemorrhage, which was evident by the significant decrease fetal movement that happened only the day before delivery in the previous healthy pregnancy, and fetal compromise in CTG showing sinusoidal heart rate pattern and flattening of the tracing, also excluded other identified neonate anemia diagnosis such as hemolytic anemia, underproduction anemia, obstetrical causes or internal hemorrhage in the baby.

The Kleihauer-Betke (KB) test is the current standard quantitative method of detecting fetal-maternal hemorrhage. Unfortunately, it is not available in our hospital at present so that had not been tested in our case. Although no confirmation KB test, the clinical picture highly represented massive FMH. Spontaneous FMH is considered in our case without identified cause, such as direct trauma to the abdomen, abruptio placentae, vasa previa with membranous insertion, choriocarcinoma, amniocentesis, chorionic villous sampling and external cephalic version.

The exact time of FMH in our case cannot be estimated but more suggestive of acute onset and the fetal compromise just at the end of pregnancy because no significant cardiomegaly and fetal hydrops.

Fetomaternal hemorrhage is not a rare condition. Therefore, it is important to pool our experiences and to alert us the possible diagnosis of FMH for those having similar presentations or neonate severe anemic cases, especially those of severe neonatal outcome. Moreover, we recommended the launching of diagnostic test of FMH in our hospital in the near future. The short-term and long-term prognosis for fetuses that experience a fetomaternal hemorrhage is variable. In our case, short-term prognosis is quite well but we need long-term follow up him for the long-term neurodevelopment consequences.

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