

Case Report of Anemia Following Fetal–Maternal Hemorrhage

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ABSTRACT

Background: Any maternal history of blood loss, ABO or Rh incompatibility, and hydrops fetalis often leads to suspicion of neonatal anemia postnatally. When maternal history consists only of decreased fetal movement, recognition of neonatal anemia can be problematic.

Clinical Findings: This case was a transported late preterm neonate who presented initially with persistent hypoxia unresponsive to usual respiratory support. On examination, mild paleness was noted.

Primary Diagnosis: Anemia caused by fetal–maternal hemorrhage was the ultimate diagnosis confirmed by a Kleihauer-Betke test on maternal serum examining fetal cells.

Interventions: Neonatal resuscitation included positive pressure ventilation, oxygen, and intubation. However, oxygenation did not improve prompting consultation with the neonatologist. Sedation and a paralytic were given. A chest radiograph ruled out pneumothoraces and pleural effusions as causative. Initiation of inhaled nitric oxide produced a mild response. Eventually, the transport nurse obtained a complete blood count indicating severe anemia, which prompted an emergent blood transfusion. The accepting neonatology team consulted with the obstetrician and a Kleihauer-Betke test was performed on mother's blood confirming a large fetal–maternal hemorrhage.

Outcomes: This neonate responded well to blood transfusions, a pressor, and respiratory support and was discharged home at 7 days of life.

Practice Recommendations: Recognition of postnatal anemia is vital to sustaining life and this can occur in the transport environment. When maternal history is nonspecific and a neonate is hypoxic, uncommon causes of hypoxia can be identified with consultation and a complete blood count.

Key Words: anemia, fetal–maternal hemorrhage, hypoxia, Kleihauer-Betke test

Anemia is a below average red cell mass most often diagnosed by hemoglobin (Hgb) or hematocrit (Hct) levels. Hemoglobin is an essential component of circulating red cells responsible for carrying and delivering oxygen from the lungs to tissues. When levels of Hgb are considerably low, inadequate or poor oxygenation to tissues can occur. Anemia and polycythemia (excessive red cell volume) are the most common hematological problems diagnosed at birth.¹ Causes of anemia at birth are often grouped into 3 categories: hemolytic, acute or chronic hemorrhage, and impaired red cell production.^{1–3} Iron deficiency is by far the most common cause of anemia worldwide and in most cases is due to loss of blood.⁴ Blood loss of as little as 15 to 20 mL may result in anemia since total blood volume in neonates ranges from 78 mL/kg in term

infants to 105 mL/kg in extremely preterm infants.¹ This article will primarily discuss hemorrhage (or loss of blood) as the cause of anemia at birth. The most common causes of anemia are listed in Table 1.

When anemia is present at birth, a history of perinatal blood loss such as abruption (acute loss), placenta previa (chronic loss), suspected fetal–maternal hemorrhage (FMH), and twin-to-twin transfusion is important to note in order to stabilize an unstable neonate. The body does not presently have a mechanism for replacing iron loss.⁴ An important component of Hgb, iron enters the bone marrow daily and is quickly and efficiently incorporated into Hgb production.⁴ When blood volume is low, newly born neonates may fail to respond adequately to resuscitation.⁵ Administration of fluid or transfusions of blood are often necessary to replace acute or severe losses present at birth.

The exact value denoting anemia in neonates depends upon several factors including gestational and chronological age. Anemia is defined as an Hgb or Hct level 2 or more standard deviations below the mean for age.^{1,2} The expected hemoglobin at birth in a term neonate is 16.5 g/dL and expected hematocrit is 51%.¹ For a 32-week neonate, Hgb is expected to be 15 g/dL and hematocrit 47%.¹ For late preterm neonates, the gestation of the index case being discussed, an expected hemoglobin level at birth is

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TABLE 1. Common Causes of Neonatal Anemia by Category

<p>Hemolytic/destruction of red cells</p> <p>Immune: Rh, ABO incompatibilities, hydrops fetalis²</p> <p>Shorter life span (<120 d) of red cells: 30-50 d in preterm^{1,2} and 60-80 d in term¹</p> <p>Infection²</p> <p>Nonimmune: spherocytosis²</p> <p>Hemoglobinopathy/variants: sickle cell²</p>
<p>Hemorrhage/blood loss²</p> <p>Placental-abruption (acute), previa (chronic)</p> <p>Twin-to-twin transfusion</p> <p>Umbilical cord compression/rupture</p> <p>Fetal-maternal hemorrhage</p> <p>Coagulopathies</p> <p>Subgaleal, intra-/extracranial, or intraventricular hemorrhage</p>
<p>Impaired red cell production²</p> <p>Iron deficiency anemia</p> <p>Anemia of prematurity</p> <p>Acquired: parvovirus B19, human immunodeficiency virus, syphilis, infection</p> <p>Congenital: Diamond-Blackfan, Fanconi anemias</p>

around 16 g/dL. Hemoglobin and Hct values normally increase over the first 3 days of life and then gradually decline to a stable level by 2 weeks.¹ While some units prefer reporting Hct levels, a 2011 Neonatal Cochrane Systematic Review recommended using Hgb levels to guide the need for transfusion until better indicators of anemia are developed.⁶ For the remainder of this discussion, Hgb level will be discussed for clarity. (To calculate Hct, multiply the Hgb level by 3.1 [from the expected values listed previously]).

PRESENTING CONCERNS

A pediatric critical care transport team consisting of a nurse, respiratory therapist, and emergency medical technician was dispatched for an imminent delivery at a local community hospital with diagnoses of 34-week neonate and “fluid around the heart.” Initial concerns by the transport team were birth of a compromised neonate in a small hospital with inexperienced staff and limited equipment for a resuscitation. The “fluid around the heart” diagnosis concerned the nurse and the respiratory therapist that the neonate may have a pericardial effusion compromising circulation or pleural effusions that may need drainage. The team was realistically concerned that this neonate could die, and the nurse felt that the situation may be very challenging for patient, family, unit staff, and transport members.

Upon arrival to the referral facility and unit, an appropriate-sized male infant was born and intubated with a 2.5-cm endotracheal tube with an

audible air leak and occasional crying. When a larger endotracheal tube was successfully inserted by the pediatrician but the neonate’s pulse oximeter reading remained low, the nurse and respiratory therapist transport members knew that additional information was needed to explain the neonate’s hypoxia. Maternal history consisted of a normal pregnancy with recent report of decreased fetal movement for a few hours prompting the mother to come to the hospital for monitoring, which occurred for nearly 24 hours before a cesarean delivery was performed for decreased variability.

CLINICAL FINDINGS

Initial impression by the transport nurse was that this was a slightly pale, appropriately-sized late preterm male orally intubated with moderate respiratory distress and lethargy. Initial vital signs included a normal heart rate but unreliable oxygen saturation readings due to an unsteady waveform; blood pressure and glucose readings had not yet been obtained. After successful reintubation, oxygen saturations were 20% to 40% with a steady, reliable waveform present on the pulse oximeter monitor, concerning for hypoxia.

On physical examination, fontanelles were soft and flat; eyes were closed and did not open with examination; nares appeared patent; orally intubated; chest size was appropriate for age with symmetric movements; breath sounds were equal bilaterally and clear in all lobes; heart sounds had a regular rate and rhythm and no murmur was audible; pulses were diminished bilaterally and perfusion was delayed at 4 to 5 seconds centrally and 6 seconds peripherally; abdomen was soft and flat without active bowel sounds; hepatosplenomegaly not appreciated; extremities appeared normal; tone was diminished; and infant responded only to deep palpation or pain. The examination was consistent with a lethargic infant who was hypovolemic and hypoxic from an unknown etiology.

Timeline

See Table 2. The infant was 28 minutes old upon arrival of transport team.

DIAGNOSTIC FOCUS AND ASSESSMENT

After the chest radiograph failed to reveal a cause for the infant’s hypoxia, a posterior tibial arterial puncture was performed and an adequate amount of blood was obtained for a blood gas, blood culture, and complete blood count after previous attempts by referral hospital staff were unsuccessful. The blood gas revealed uncompensated metabolic acidosis and confirmed the hypoxia. The accepting

TABLE 2. Timeline for Case Study

Relevant Past Medical History and Interventions		
Date/Time	Summaries From Initial and Subsequent Contact/ Clinical Findings	Interventions
12/4/2014	G1P0 mother admitted to local community hospital because of decreased fetal movement. Ultrasonography revealed fetal pericardial effusion and fluid surrounding the liver.	
12/5/2014 at 1447	34-wk male born by cesarean section for fetal decelerations. Apgars were 2, 3, and 7 at 1, 5, and 10 min prompting positive pressure ventilation (PPV) and intubation.	
Date/Time	Summaries From Initial and Subsequent Contact/ Clinical Findings	Interventions
12/5/xx 1515	Transport team arrived to bedside. Infant slightly pale; occasional faint crying noted; orally intubated with 2.5-cm endotracheal tube.	PPV being performed by RT/MD Transport RN resumes PPV; extubates infant when changing to flow-inflating bag with pressure manometer. PPV with mask initiated.
1520	Obtained history of mother admitted previous day with decreased fetal movement. Cesarean section today for decelerations.	Infant reintubated with 3.0-cm endotracheal tube by pediatrician on first attempt.
1525		Chest radiograph obtained for endotracheal tube placement and to assess for pleural effusions
1530	Transport nurse consulted with neonatologist about hypoxia despite delivering 100% oxygen. Recommended sedation, then paralytic if no change in saturations. If these do not help, attempt needle aspiration for pleural effusions. Can also start nitric oxide.	Respiratory therapist (RT) placed infant on transport ventilator: Rate of 40 breaths per minute; PEEP of 5; PIP of 25; FiO_2 100% Endotracheal tube advanced 2 cm
1555	Infant asleep after sedation and paralytic. Pulses diminished $\times 4$.	Removed from ventilator; given PPV until saturations to 40 Given fluid bolus of normal saline
~1610		Respiratory therapist in process of starting inhaled nitric oxide Transport nurse performs right posterior tibial arterial puncture and obtains adequate blood for arterial blood gas (ABG), blood culture, and complete blood count (CBC) Transport nurse initiates antibiotics

(continues)

TABLE 2. Timeline for Case Study (Continued)

Date/Time	Summaries From Initial and Subsequent Contact/ Clinical Findings	Diagnostic Testing/Findings	Interventions
1618	Transport nurse called neonatologist with update on blood gas and CBC results. Ordered second fluid bolus and emergent blood transfusion.	Complete blood count results called to nurse from laboratory: white blood cells 45.9, hemoglobin 4.9 g/dL, hematocrit 18.3%, platelets 197,000	Transport nurse requests emergency release blood from hospital's blood bank Normal saline fluid bolus given.
1630			Nitric oxide started by RT
1636		Pulse oximeter: 50 Heart rate: 100	Transport nurse starts transfusion of packed red blood cells over 1 h.
1640	Infant loaded into transport Isolette. Discussed infant's serious condition with family and given brochure with phone number, security code for calls, and directions to hospital.	Pulse oximeter: 60-74	
1721	Arrived at children's hospital and transferred care to neonatal intensive care unit staff. Updated new neonatologist-night coverage.	Pulse oximeter: 90 Heart rate: 159	

Abbreviations: BP, blood pressure; MAP, mean arterial pressure; MD, medical doctor; PEEP, positive end expiratory pressure; PIP, positive inspiratory pressure; RN, registered nurse; RT, respiratory therapist.

neonatologist was notified with interpretation of the chest radiograph and arterial blood gas results and a fluid bolus was ordered and initiated and inhaled nitric oxide was started with mild improvement in oxygen saturations to 50%. Although there was slight improvement with suspected pulmonary vasodilatation and increased intravascular volume, the infant remained hypoxic.

THERAPEUTIC FOCUS AND ASSESSMENT

The results of the complete blood count revealed an elevated white blood cell count, a low hemoglobin of 4.9 g/dL, and a normal platelet count. The neonatologist was updated and ordered emergency release type O negative blood to run over 1 hour to correct the severe anemia, acidosis, and over time, the hypoxia by replacing iron and hemoglobin. Ampicillin and gentamicin were also ordered to start when in route to the children's hospital. Within 9 minutes of starting the blood transfusion at the referring hospital, the oxygen saturations had improved considerably to 74% indicating that anemia was likely the primary cause of hypoxia.

At almost 2 hours of life, the infant was placed in a transport Isolette and transfer to the local children's hospital ensued. The family was updated immediately before leaving the referral hospital regarding the infant's critical condition and the possible need for escalation of care depending on his response to the blood transfusion. In the 30-minute ambulance transport, the infant's heart rate remained stable, and upon arrival to the children's hospital an oxygen saturation of 90% was present. The neonate's father and grandparents were again updated upon arrival to the children's hospital and the neonate's mother was called with a condition update.

Shortly after admission, a repeat arterial blood gas revealed a pH of 7.01, a carbon dioxide level of 40, oxygen level of 53, and bicarbonate level of 10.1 with a base deficit of 19. A repeat complete blood count revealed a white count of 12,400, hemoglobin of 6.1 g/dL, and platelet count of 183,000, with a significant left shift on the differential. A second blood transfusion and later a third were given along with a dopamine infusion to increase blood volume and perfusion. The attending neonatologist consulted with the mother's obstetrician and a Kleihauer-Betke test was sent with mother's serum a few hours after admission. This test estimated 97 mL or approximately one-half the neonate's total blood volume present in the mother's circulatory system confirming a significant FMH.

In addition to the identification of severe anemia during transport, this neonate was later diagnosed with hydrops fetalis of unknown origin and pulmonary hypertension during the hospitalization. This neonate was successfully extubated the day after

admission and subsequently placed on room air on day 5. He quickly advanced to full volume nipple feedings and his hemoglobin before discharge had increased to 13.6 g/dL. Although a pericardial effusion was suspected on prenatal ultrasonography, this was not confirmed postnatally on echocardiogram. At discharge on day 7, this infant had the appearance of a healthy, late preterm infant.

DISCUSSION

In an FMH, fetal blood loss to the placenta and maternal circulation can sometimes be substantial and even result in fetal death. Prenatally, the placenta is normally the gatekeeper between maternal and fetal circulations and usually does well at maintaining these as separate, preventing transfer of cells between fetus and mother or vice versa. In about half of pregnancies though, fetal cells can be found in the maternal circulation,² but this usually does not affect the neonate after birth. Although fetal blood loss into maternal circulation rarely exceeds 1 mL, in term infants it can exceed 30 mL, considered severe by some sources, which is seen in about 0.3% of pregnancies⁷ or 1 in 3000 to 10,000 women.⁸ Since FMH is most often clinically silent in the mother and thus can be unrecognized during pregnancy, another method of assessing FMH incidence is by cases of FMH per number of anemic neonates shortly at birth. When this method was utilized in a large cohort study, an incidence of 182 cases of FMH per 1000 anemic neonates was found after investigators provided an educational intervention to physicians and thus increasing recognition and testing for FMH.⁹ Before the intervention, an incidence of 22 cases per 1000 anemic neonates was found with an unchanged incidence of neonatal anemia.⁹ This suggests that FMH causing anemia in the fetus/neonate likely occurs more often than previously reported, and the investigators felt that diagnosis is largely dependent upon physician recognition and thus, timely maternal testing for fetal hemoglobin before these cells are cleared from mother's system.⁹

Maternal trauma, version or external turning of the fetus, and conditions leading to placental anomalies such as preeclampsia can contribute to FMH.¹⁰ Beyond this, little is known about the etiology of FMH including when in gestation the transfer of cells occurs and whether the transfer is chronic or acute. Fetal-maternal hemorrhage is difficult to study since it is mostly asymptomatic except for nonspecific symptoms. Signs of fetal anemia in utero include decreased fetal movement, a sinusoidal fetal heart rate pattern, or devastating illness or outcome including fetal/neonatal death.⁹ Postnatally, pallor may be the most common or only symptom present in anemic neonates.³ If anemia is suspected in a newborn and

the cause is unknown, it is important to notify obstetrical staff to order testing for fetal cells in maternal serum before these are eliminated, which can be at an unpredictable rate. In one study, fetal cells could be found in maternal circulation up to 43 days after delivery.⁸ Most centers utilize the Kleihauer-Betke stain test^{8,9} in which staining is applied to the sample that is later absorbed by fetal cells but not adult cells and requires a manual count of these darker-stained cells by laboratory personnel. A milliliter amount is then calculated to determine the degree of FMH. In the case presented, 97 mL of fetal cells were noted in maternal serum indicating a severe FMH.

Severe hypoxia can be seen with anemia and present as a neonate who is unresponsive to typical resuscitation measures including oxygen administration and mechanical ventilation. Lack of adequate amounts of oxygen can result in cell and tissue death in as little as 2 minutes.¹¹ Hemoglobin, a protein, is responsible for providing cells with oxygen. Fetal hemoglobin (Hgb F) is the major Hgb type present in the fetus and the neonate. After umbilical cord excision through 6 months of age, adult hemoglobin (Hgb A) production increases significantly to become the major type, while Hgb F declines to less than 1% of total Hgb.² Several factors determine Hgb's oxygen-carrying capacity. Fetal hemoglobin has a higher oxygen attraction than does Hgb A allowing prenatal transport of oxygen across the placenta from maternal Hgb A to fetal red cells.² Temperature, pH, levels of carbon dioxide, and the concentration of organic phosphates in red blood cells also influence affinity of Hgb molecules for oxygen.²

Measurement of oxygenation is necessary for early recognition of hypoxia and to initiate interventions that restore adequate oxygen levels. Use of pulse oximetry for continuous noninvasive oxygen monitoring has been a standard for this purpose for almost 40 years. Before this pulse oximetry was refined for widespread use, dentists and anesthesiologists determined tolerance to anesthesia by the presence of cyanosis in patients.¹¹ This method of color perception is riddled with uncertainty and is no longer considered an accurate estimate of oxygenation. Humans are not well equipped to determine subtle changes in color as with determining cyanosis or presence of bilirubin. In newly born infants, even neonatal staff demonstrate considerable variation in color perception.¹² In addition, hemoglobin concentration can have a huge impact on detecting cyanosis. When hemoglobin concentrations in the red blood cell are abundant as in polycythemia, an infant is more likely to look cyanotic when oxygen levels are low. In the anemic infant with less hemoglobin, cyanosis may not be visible until oxygen levels are extremely low. To ensure safe estimation of hypoxia in infants, use of pulse oximetry monitoring

Summary of Recommendations for Practice and Research

What we know:

- Decreased fetal movement is one of the few prenatal signs of fetal–maternal hemorrhage (FMH).
- In half of pregnancies, fetal cells can be found in maternal circulation² but these rarely affect the fetus/neonate.
- Fetal blood loss rarely exceeds 1 mL in the maternal circulation but can be 30 mL or more in term infants, considered severe.
- Incidence of severe FMH is 0.3% of pregnancies⁷ or 1 in 3000-10,000 women.⁸

What needs to be studied:

- FMH is poorly understood and underrecognized.
- The proper technique for educating staff about rare but deadly topics such as FMH should be examined.
- Noninvasive methods of assessing fetal hemoglobin are desperately needed so that Hgb can be assessed quickly in pregnant women with decreased fetal movement.

What can we do today:

- Recognize signs and causes of anemia in newly born infants.
- Educate ourselves and others about FMH to increase awareness and save lives.
- Disseminate case studies of FMH and other diagnoses.
- When a newly born infant's response to resuscitation is inadequate, consider other, more rare causes such as FMH.

is now recommended by the neonatal resuscitation program guidelines.⁵

Use of anesthesia for surgical procedures led to the study and testing of several methods of assessing oxygenation in the mid-1900s.¹¹ In 1954, transcutaneous oxygen monitoring was created by Leland Clark as a noninvasive method of assessing systemic oxygen levels from skin as it “breathes.”¹¹ This method was later used in premature infants in 1972 to prevent infant blindness from hyperoxia.¹¹ Additional monitoring of oxygen levels was unfeasible, very costly, or plagued with artifact and not widely used in the clinical setting. In 1973, Takuo Aoyagi, a Japanese bioengineer, uncovered an equation to measure the percentage of oxygen saturation in arterial blood (Sao₂) using the ratio of red and infrared light absorbed or spread through tissues as it changed with arterial blood flow.¹¹ Saturation of the hemoglobin molecule is now widely used and is the preferred noninvasive method of assessing oxygenation.

Limitations of using pulse oximetry include inability in obtaining readings during states of low perfusion and inaccuracy of readings in low ranges. Unfortunately, most pulse oximeter systems are unable to assess oxygen saturation without a strong, reliable pulse. This could be due to lack of adequate red and infrared light. Also, when the hemoglobin molecule becomes less than 70% saturated, the pulse oximeter system is unable to reliably estimate saturation.¹³ Newer technology, although not universally adopted by all hospitals likely due to cost, has resulted in pulse oximeter probes capable of reading at low perfusion states.

According to neonatal resuscitation program, by 10 minutes of life, the expected oxygen saturations should be at least 85%.⁵ This case's saturations were well below this level despite delivering 100% oxygen through an appropriately-sized endotracheal tube. Some degree of pulmonary hypertension was suspected in this infant since the low saturations

were reported since birth and were mildly responsive to the pulmonary vasodilator nitric oxide. Assessing the degree of shunt by placing 2 simultaneous pulse oximeters in pre- and postductal positions can be done but was not in this case.

CONCLUSION

Since an adequate red cell mass and blood volume are crucial to deliver oxygen to tissues, anemia should be considered in any newly born neonate that does not respond to conventional resuscitation measures. While pallor in a neonate may be associated with anemia and should prompt verification with a complete blood count, it is a nonspecific sign and dependent upon many factors. This case demonstrates that a prenatal history of decreased fetal movement, mild paleness, and hypoxia unresponsive to typical measures can indicate severe anemia. More education is needed for neonatal staff to increase recognition of neonatal anemia from such causes as FMH to improve diagnosis and ultimately, outcome. Also, more research is needed to determine causes of FMH and early recognition before anemia is severe to improve fetal outcomes.

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