Journal of Perinatology (2013) 33, 429–434
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www.nature.com/jp

ORIGINAL ARTICLE

Severe neonatal anemia from fetomaternal hemorrhage: report from a multihospital health-care system

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OBJECTIVE: The incidence of fetomaternal hemorrhage that is severe enough to cause neonatal anemia is not known. Owing to its relative rarity, much of the literature describing this condition is in the form of case reports and small case series. We performed a large, muiticentered, sequential, case series to determine the incidence, antecedents and outcomes.

STUDY DESIGN: From the multicentered databases of Intermountain Healthcare, we obtained records of all neonates with hematocrit (Hct) $<30\%$ or hemoglobin (Hgb) $<10\,g\,dl^{-1}$ on the day of birth, who had Kleihauer–Betke staining or flow cytometric evidence of fetomaternal hemorrhage.

RESULT: Among 219 853 live births, 24 had anemia with evidence of fetomaternal hemorrhage (incidence estimate, 1 per 9160 live births). The initial Hgb ranged from 1.4 to 10.2 $g\,dl^{-1}$ (Hct 29.8%). The initial Hgb was $<7\,g\,dl^{-1}$ in 18 (67%), $<5\,g\,dl^{-1}$ in 12 (50%) and was $<3\,g\,dl^{-1}$ in 7 (29%). All 7 mothers in whom neonatal Hgb was $<3\,g\,dl^{-1}$ had reported absent fetal movement, as did 13 of 18 mothers when the initial Hgb was $<7\,g\,dl^{-1}$. Outcomes were poorer in those with the lowest initial Hgb; in the two lowest, one died on day 1, and the other developed a grade 4 intraventricular hemorrhage (IVH). The adverse outcomes of death, IVH, periventricular leukomalacia, bronchopulmonary dysplasia or hypoxic-ischemic encephalopathy were common; occurring in 71% (17 of the 24), including all with an initial Hgb $<5\,g\,dl^{-1}$ and all born at $\leqslant 35$ weeks of gestation.

CONCLUSION: Fetomaternal hemorrhage is a rare but sometimes devastating condition. Those with fetomaternal hemorrhage and an initial Hgb of $<5\,g\,dl^{-1}$ are expected to need resuscitation at birth, to receive emergent transfusion support and to be at risk for death and major morbidities. Antenatal suspicion of this diagnosis should occur when absent fetal movement is reported. Improvements in rapid diagnosis are needed to prepare first responders and transfusion services.

Journal of Perinatology (2013) **33,** 429–434; doi:10.1038/jp.2012.142; published online 29 November 2012

Keywords: anemia; hemorrhage; fetomaternal

INTRODUCTION

Very small quantities of fetal blood enter the maternal circulation in essentially all pregnancies,[1,2] but the incidence of fetomaternal hemorrhage that is severe enough to cause fetal/neonatal anemia is not known. Using Kleihauer–Betke testing, Sebring *et al.*,[3] Pollack *et al.*[4] and Rubod *et al.*[5] reported that fetomaternal hemorrhage $\geqslant 30$ ml occurs once per 330 births. De Almeida and Bowman[6] reported that hemorrhages exceeding 150 ml of fetal blood occur once per 5000 births.

Fetomaternal hemorrhage is important to obstetrical practice as a cause of fetal death and stillbirth.[2,7] It is also important to pediatricians and neonatologists, because such hemorrhage can be the source of neonatal hypovolemic shock, anemia, hydrops and respiratory distress. Best practice models for caring for these neonates should be based on data and evidence. However, most of the pertinent literature is in the form of single case reports and small case series,[8–14] owing to the relative rarity of the condition. We reasoned that information relevant to neonatal care might be obtained by identifying all recognized liveborn neonates with anemia and laboratory evidence of fetomaternal hemorrhage in the Intermountain Healthcare multihospital system.

METHODS

This was a retrospective analysis of clinical and laboratory data existing in Intermountain Healthcare data repositories. The Intermountain Healthcare Institutional Review Board approved the study as a deidentified data-only investigation not requiring written consent.

The experimental approach was to cohort the records of all live births cared for in any Intermountain Healthcare hospital with a date of birth between 1 January 2005 and 31 December 2011, and then to use two criteria to identify the study group from this cohort; (1) an initial hematocrit (Hct) on the day of birth of $<30\%$ or hemoglobin (Hgb) $<10\,g\,dl^{-1}$, and (2) laboratory evidence of fetomaternal hemorrhage obtained after delivery. The latter consisted of a positive Kleihauer–Betke stain of maternal blood or flow cytometric quantification of fetal blood in the maternal blood. Acid elution techniques such as Kleihauer–Betke stain testing have been shown to have a 96% sensitivity of detecting a >10-ml fetal maternal hemorrhage.[15] The electronic data sources were ICD9, Case Mix (the billing, coding and financial data mart used by Intermountain Healthcare) and EVOX (the extended Vermont-Oxford database).

Gestational age was determined by obstetrical assignment unless this was changed by the neonatologist on the basis of gestational age assessment (physical examination and neurological/neurodevelopmental findings). The Vermont/Oxford definitions[16] were used for severe intraventricular hemorrhage (IVH grade $\geqslant 3$), periventricular leukomalacia and bronchopulmonary dysplasia.

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Received 20 August 2012; revised 24 October 2012; accepted 25 October 2012; published online 29 November 2012

The program used for data collection was a modified subsystem of 'clinical workstation'. Clinical workstation is a web-based electronic medical record application that stores demographic and clinical information, such as history, physical examination results, laboratory data, radiographic findings, problem lists and discharge summaries. The 3M Company (Minneapolis, MN, USA) approved the structure and definitions of all data points for use within the program.

RESULTS

Of the 219 853 live births in 20 hospitals during this period, 24 were identified as having anemia at birth (Hct <30% or Hgb <10 g dl^{-1}) with laboratory evidence of fetomaternal hemorrhage. Thus, the entity was recognized in 1 per 9160 live births. The annual number of cases recognized in our health system did not change appreciably over this period (three to five cases per year).

All 24 cases had a positive Kleihauer–Betke stain of mother's blood; 4 also had cytometric quantification of the volume of fetal blood in the maternal circulation. Laboratory reports in 10 read 'positive for fetal maternal hemorrhage' with no estimate of the percentage of fetal erythrocytes in the mother's blood. The 14 others were reported as 'positive for fetal maternal hemorrhage' and listed a percentage of fetal cells ranging from 0.9 and 6.2%. Flow cytometric estimates of the volume of fetal blood in the maternal circulation ranged from 92 to 370 ml.

Demographic features of the 24 affected neonates are shown in Table 1, listed in order of the initial Hgb, from lowest (case 1, 1.4 g dl^{-1}) to highest (case 24, 10.2 g dl^{-1} (Hct 29.8%)). Of the 24 birth, 10 were in hospitals with level III neonatal intensive care units, 12 in hospitals with level II care units and 2 in hospitals with level 1 (well-baby) nurseries only. The two neonates with the lowest Hgb were the two earliest gestational age and lightest birth weight neonates. Mothers reported absent or decreased fetal movements in all 7 with an initial Hgb <3 g dl^{-1}, in 12 of 14 with Hgb <5.8 g dl^{-1} and in 13 of 18 with Hgb <7 g dl^{-1}. One of the neonates was hydropic at birth (case 19); the other 23 had no ascites, pleural fluid or hydrops noted.

Of the 24 neonates, 19 were delivered by emergent cesarean section due to non-reassuring fetal heart tracings, and all of these had an initial Hgb <8 g dl^{-1}. Three (cases 3, 7 and 13) had sinusoidal fetal heart tracings charted. None of the 24 neonates was detected antenatally; none had cordocentesis or intrauterine transfusion. The pregnancy, before the day of delivery, was reported as uncomplicated in all but six cases; two of these had an amniocentesis, one (case 19) to relieve pleura fluid (nonimmune hydrops) and the other (case 22) to assess fetal lung maturity before delivery. One (case 12) had a car accident at 19 weeks of gestation; one (case 13) reported vaginal bleeding from 12 to 20 weeks of gestation; one (case 24) was taking one baby aspirin daily and was diagnosed with subchorionic hemorrhage in the first trimester.

The initial laboratory tests on the first day of life are shown in Table 2. Nucleated red blood cell counts were above the reference range in 21 cases.[17] Thrombocytopenia was recognized on the first day in seven cases. All 24 cases had an initial pH >7.00. Weak positive correlations were identified between the initial Hgb and the first base excess ($R^2 = 0.089$) and between initial Hgb and first pH ($R^2 = 0.051$). Negative correlations were identified between initial Hgb and nucleated red blood cell count ($R^2 = 0.272$), and between initial Hgb and reticulocyte count ($R^2 = 0.153$).

Clinical presentation and interventions during the first day are shown in Table 3 in association with the initial Hgb. Low Apgar scores were common among those with an initial Hgb <5 g dl^{-1}. A correlation was identified between initial Hgb and 1 min Apgar score ($R^2 = 0.229$) and between initial Hgb and 5 min Apgar score ($R^2 = 0.297$). Five of the seven cases with initial Hgb <3 g dl^{-1} received chest compressions for resuscitation after delivery. In addition, 14 cases received mechanical ventilation and 7 cases received vasopressors.

A total of 21 neonates received red blood cell transfusions on the first day; 14 of these were emergent uncrossmatched

Table 1. Demographic features of 24 neonates with anemia at birth (hematocrit <30% or Hgb <10 g dl^{-1}) and laboratory evidence of fetomaternal hemorrhage

Case no.	*Initial Hgb*	*GA*	*BW*	*Gender*	*Race*	*Mat Age*	*Gravida*	*Decreased fetal movement*	*Sinusoidal FHT*
1	1.4	29.0	1465	F	White	29	4	Yes	No
2	1.4	28.5	1045	F	White	20	1	Yes	No
3	1.7	37.4	3653	M	Am Indian	26	3	Yes	Yes
4	2.3	32.1	1810	F	White	24	1	Yes	No
5	2.6	37.2	2514	F	White	25	1	Yes	No
6	2.7	40.0	3550	M	White	25	1	Yes	No
7	2.8	34.6	2235	F	White	24	1	Yes	Yes
8	3.1	35.0	2013	F	White	24	1	No	No
9	3.5	33.2	2280	M	White	37	9	Yes	No
10	3.8	37.4	3653	M	White	27	4	Yes	No
11	4.4	39.0	4598	M	White	27	2	Yes	No
12	4.8	36.5	2615	M	White	29	4	No	No
13	5.4	39.2	2840	F	White	37	6	Yes	Yes
14	5.6	35.2	2548	F	White	29	1	Yes	No
15	5.8	37.0	3309	M	Hispanic	26	3	No	No
16	5.8	40.1	2930	M	Hispanic	17	1	No	No
17	6.1	38.0	3574	F	White	27	1	No	No
18	6.8	39.0	3009	F	White	32	5	Yes	No
19	7.9	29.6	1812	F	White	23	1	No	No
20	8.3	40.4	3765	M	Hispanic	29	3	No	No
21	8.6	40.3	3758	M	White	24	1	No	No
22	9.5	37.6	2855	F	White	39	1	No	No
23	9.5	34.5	2011	M	White	30	4	No	No
24	10.2	35.6	2839	M	White	24	2	No	No

Abbreviations: BW, birth weight (in grams); F, female; FHT, fetal heart tracing; GA, gestational age (weeks.days); Hgb, hemoglobin (g dl^{-1}); M, male; Mat, maternal.

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Table 2. Initial Hgb value with other laboratory values obtained on the first day of life

Case no.	*Hgb, g dl^{-1}*	*Hct, %*	*MCV, fl*	*NRBC/ 100 WBCs*	*WBC, K μl^{-1}*	*Platelet count, K μl^{-1}*	*1st pH*	*1st base excess*	*Retic, %*	*ARC, K μl^{-1}*	*Fib, mg dl^{-1}*	*PT, s*	*aPTT, s*	*ALT, U l^{-1}*	*AST, U l^{-1}*
1	1.4			270	8.8	42	7.05	−12.1			114	24.1	64		
2	1.4			83	19.9	39	7.15	−16.9			73	33	119		
3	1.7	7.9	117.9	130		213	7.16	−9.9							
4	2.3	7.4	131.6	477	8.3	96	7.06	−11.2			132	32.8	46	1144	233
5	2.6	9.7	132.6	81	28	227	7.18	−18.5	1.5	73.3				189	116
6	2.7		107	59	28	185	7.03	−22			154	15.7	42	281	403
7	2.8	9	98.6	39	27.1	101	7.5	−9.5							
8	3.1			97	13.5	129	7.23	−5.8						95	35
9	3.5	10.9	114	79	28.9	183	7.09	−10	1.8	79.5	189	15.7		99	57
10	3.8	15.6	115	16.6	25.5	293	7.3	−2.8	28.1	366					
11	4.4	16.3	118	157	43.2	175	7.04	−12			154	19.4	28	387	196
12	4.8	14.2	122	90	29	90	7.22	−10			116	14.4	35	71	223
13	5.4	16.6	111.2	44	49.6	215	7.21	−9	5.6	257				110	124
14	5.6	17.2	113.1	47	24.6	215	7.25	−7.8							
15	5.8	16.6	120.3	172	29.3	172	7.21	−8	7.9	250				395	307
16	5.8		117.2	72.9	15.2	293	7.15	−5.5							
17	6.1	22.9	105.9	56	21.4	240	7.35	−5						73	18
18	6.8	21.6	111.8	26	27	476	7.31	−6.9	9.2	157				24	30
19	7.9	28	110	38	43	221	7.01	−7.2	5.5	282	180	12.9	25	170	40
20	8.3	20.4	106.4	16	20.6	258	7.24	−5.8	18.7	18.7					
21	8.6	26.3	100.4	2	14.2	115									
22	9.5	27.9	109.6	6.4	12.9		7.01	−24.1							
23	9.5			33			7.42	−1.4	2.7	72.7				54	15
24	10.2	29.8	107.9	19	9	200	7.32	−6.2							

Abbreviations: ALT, alanine transaminase; aPTT, activated partial thromboplastin; ARC, absolute reticulocyte count; AST, aspartate transaminase; Fib, fibrinogen; Hct, hematocrit; Hgb, hemoglobin; MCV, mean corpuscular volume; NRBC, nucleated red blood cell; PT, prothrombin time; Retic, reticulocyte count; WBC, white blood cell.
Blank cells indicate that the test was not performed in the first 24 h.

type O (−) erythrocytes. All four neonates born at <32 weeks of gestation also received platelets and fresh frozen plasma on the first day. Of the 24 neonates, 5 were not admitted to the neonatal intensive care unit immediately after birth. These five neonates, all with Hgb ⩾5.8 g dl^{-1}, were initially cared for in a well-baby unit until 4 to 12 h after birth, when respiratory distress was observed and anemia was diagnosed.

Neonatal morbidities are shown in Table 4. Of the two neonates with the lowest initial Hgb, one died on the first day and the other developed a severe IVH with posthemorrhagic hydrocephalus, resulting in ventriculoperitoneal shunting and poor neurodevelopmental outcome. Death, IVH, periventricular leukomalacia, bronchopulmonary dysplasia or hypoxic-ischemic encephalopathy occurred in 17 of the 24 neonates, including all of 8 born at ⩽35 weeks of gestation.

DISCUSSION

Anemia at birth is an important problem, but one that is incompletely understood. The complexity of the condition is derived from the various etiologies, mechanisms, kinetics and genetics involved.[18] In an attempt to improve our understanding of anemia at birth, and thereby improve outcomes, we have engaged in a step-wise approach, including first defining the reference ranges for Hct/Hgb, red blood cell indices and nucleated red blood cell count according to gestational and postnatal age.[17,19,20] The present study was undertaken as another step; focusing on the subgroup of neonates with anemia at birth and evidence of fetomaternal hemorrhage.

Fetomaternal hemorrhage can require the rapid, intensive and coordinated efforts of Obstetrics, Pediatrics, Neonatology, the transfusion service and the clinical laboratory.[2] Fortunately, the condition is rare. However if, as our present data suggest, 1 case occurs in each 9000 live births, ~450 to 500 liveborn neonates are affected in the United States each year. We recognize that our calculations underestimate the actual incidence. First, our catchment criteria for this study miss those with anemia but no Kleihauer–Betke testing, and those with anemia too mild to produce signs prompting neonatal intensive care unit admission, and those with a hemorrhage occurring only early in pregnancy. Second, fetomaternal hemorrhage can cause stillbirth and we made no attempt to study stillbirths. However, using Intermountain Healthcare and University of Utah data, Silver *et al.*[7] estimated that 3 to 14% of stillbirths follow fetomaternal hemorrhage.

Outcomes of liveborn neonates after fetomaternal hemorrhage include neonatal death and various neonatal morbidities.[2,5] Perhaps 20 to 30% of affected neonates have one or more of these outcomes, with higher odds of a poor outcome if born prematurely or with Hgb <3 g dl^{-1}.[2,5] Means of improving the care of these patients, and increasing the odds of a good outcome, are surely needed.

Many important issues relevant to fetomaternal hemorrhage are unresolved. For instance, it is unclear how much fetal blood must be lost before the fetus is seriously endangered. Studies by Brace,[21] using fetal sheep, indicate that if the rate of blood loss is slow, a loss of up to 30% of the intravascular volume can be tolerated. However, with acute blood loss the chance for fetal damage is much higher. With hyperacute hemorrhage (occurring within minutes to an hour before birth), the Hgb and Hct of a neonate might be normal at birth. Only after the intravascular volume increases by ingressing fluid will the Hgb fall from dilution. As all 16 of our patients with Hgb <6 g dl^{-1} were first measured within minutes to an hour after birth, and all were low, we assume that hyperacute hemorrhage was not present in these patients.

The lack of hydrops at birth in 23 of our 24 patients, despite a very low Hgb, may give some understanding of the timing of the hemorrhage. Hydrops from anemia takes a period of time to develop. Observational reports suggest that hydrops can develop

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Table 3. Initial Hgb, clinical presentations and interventions during the first day of life

Case no.	*Hgb*	*Apgar*		*Resuscitation*		*NICU admission*	*Resp support*	*Vasopressor*	*Uncrossmatched O neg*	*Transfusions (ml kg^{-1}) during 1st DOL*		
		1 min	*5 min*	*PPV*	*Chest Compr*					*RBC*	*Platelets*	*FFP*
1	1.4	0	1	Yes	Yes	Immediate	Vent	Yes	Yes	20[a]	20	15
2	1.4	0	0	Yes	Yes	Immediate	Vent	No	No	60	20	20
3	1.7	0	ND	Yes	Yes	Immediate	Vent	No	Yes	120	0	0
4	2.3	1	3	Yes	Yes	Immediate	Vent	Yes	Yes	30	10	15
5	2.6	2	5	Yes	No	Immediate	Vent	Yes	Yes	40[a]	0	0
6	2.7	0	3	Yes	Yes	Immediate	CPAP	Yes	Yes	45	0	15
7	2.8	4	5	Yes	No	Immediate	Vent	No	Yes	55	20	0
8	3.1	7	8	Yes	No	Immediate	Vent	No	Yes	2[b]	0	0
9	3.5	4	6	Yes	No	Immediate	HFNC	No	Yes	2[b]	0	0
10	3.8	8	8	BB O_2	No	Immediate	HFNC	No	Yes	15	0	0
11	4.4	2	3	Yes	No	Immediate	Vent	No	Yes	20	0	0
12	4.8	1	3	Yes	No	Immediate	Vent	No	Yes	45	15	0
13	5.4	4	7	CPAP	No	Immediate	NC	No	Yes	30	0	0
14	5.6	8	8	BB O_2	No	Immediate	Vent	No	Yes	40	0	0
15	5.8	8	9	BB	No	Immediate	HFNC	No	No	15	0	0
16	5.8	8	9	No	No	8 h	RA	No	No	0	0	0
17	6.1	6	8	No	No	4 h	NC	No	No	20	0	0
18	6.8	7	8	No	No	7 h	NC	No	No	20	0	0
19	7.9	1	7	Yes	No	Immediate	Vent	Yes	No	1[b]	3[b]	1[b]
20	8.3	3	9	No	No	Immediate	NC	No	No	0	0	0
21	8.6	8	9	BB O_2	No	10 h	NC	No	No	20	0	0
22	9.5	6	8	BB O_2	No	12 h	NC	No	No	0	0	0
23	9.5	2	2	Yes	No	Immediate	Vent	Yes	Yes	20	0	0
24	10.2	8	8	Yes	No	Immediate	Vent	Yes	No	10	0	0

Abbreviations: BB O_2, blowby oxygen; CPAP, continuous positive airway pressure; DOL, day of life; FFP, fresh frozen plasma; h, hours of life; Hgb, hemoglobin; HFNC, high flow nasal cannula; NICU, neonatal intensive care unit; ND, not done; PPV, positive pressure ventilation; RA, room air; RBC, red blood cell; Resp, respiratory; Vent, mechanical ventilation.
[a]A portion was given as exchange transfusion.
[b]Indicates number of transfusions (ml kg^{-1} not available in electronic records).

if the fetal Hgb falls below $\sim$6 g dl^{-1} for several days (4 to 6 days).[22] Of our 24 cases, 16 had an initial Hgb $<$6 g dl^{-1} but none of these were hydropic. Thus, perhaps the hemorrhages occurred within only a day or two before birth, not giving time for hydrops to develop. We speculate that the hemorrhages in our cases were neither hyperacute nor did they occur many days before birth, but generally occurred in a period intermediate between these two.

Another important unknown aspect is the relationship between hemorrhage volume and odds of brain injury. Rubod *et al.*[5] concluded that when fetal blood loss exceeds 20 ml kg^{-1}, the neonate generally requires neonatal intensive care unit care, when it exceeds 40 ml kg^{-1}, the chance of stillbirth or major morbidity is significant and at $>$80 ml kg^{-1}, adverse outcomes are invariable In our study, 8 of 23 (35%) surviving infants had evidence of brain injury. In contrast, neurologic injury was found in only 0 to 4% of surviving infants in smaller series.[3,5] We suspect than many neonates in previous reports were not as severely affected as most of the neonates in the present report. Accurate data on brain injury from fetomaternal hemorrhage are elusive because some fetuses might sustain brain damage from hemorrhage months before delivery, yet not be anemic at birth.

If adverse consequences of fetomaternal hemorrhage are to be avoided, it is important to diagnose this process and intervene as early as possible. However, we had no cases where an antenatal diagnosis was made. Obstetricians generally perform testing for fetomaternal hemorrhage in a limited number of situations; abdominal trauma, abruption, fetal hydrops and stillbirth. Gacoia[23] found no case where maternal trauma or abruption were antecedent to fetomaternal hemorrhage. In our series, only one case had trauma during pregnancy (a car accident), and two had vaginal bleeding. Neither of the vaginal bleeding case seemed to be from an acute abruption, and thus they would not likely have had a test for fetomaternal hemorrhage. Similarly, testing was not ordered on the woman after the car accident. The American College of Obstetrics and Gynecology does not recommend testing for fetomaternal hemorrhage in women undergoing trauma except in cases in which the dose of Rhogam must be calculated.[24]

Decreased or absent fetal movement was reported by mothers in 54% of our cases and in 27% of those reviewed by Gacoia.[23] In both series, this was by far the most common presenting sign or symptom. These reports raise the question as to whether maternal perception of fetal movement could be a useful screening method for severe fetomaternal hemorrhage. However, in obstetric practice it is quite common for patients to report decreased movement, and it is not clear whether this should always prompt a consideration of fetomaternal hemorrhage. When decreased fetal movement is reported, a typical response is to perform a nonstress test or biophysical profile. In our study, 19 of the 24 had a 'non-reassuring' fetal heart rate pattern prompting an emergent cesarean section. Thus, perhaps a normal nonstress test or biophysical profile might indeed be reassuring that a significant hemorrhage has not occurred. Our database did not allow us to precisely define the type of 'non-reassuring' fetal heart rate pattern present. The classic pattern with severe fetal anemia resembles a sine wave (hence the term 'sinusoidal'). However, this

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Table 4. Initial Hgb, morbidities and clinical outcomes

Case no.	*Initial Hgb*	*BPD*	*IVH*	*PVL*	*HIE*	*LOS (days)*	*D/C disp*
1	1.4	Yes	IVH[a]	No	No	96	Home
2	1.4	[b]	[b]	[b]	[b]	1	Expired[b]
3	1.7	No	No	No	Yes	10	Home1
4	2.3	Yes	No	No	No	32	Home
5	2.6	Yes	No	No	Yes	22	Home
6	2.7	No	No	No	Yes	31	Home
7	2.8	No	No	Yes	No	11	Home
8	3.1	Yes	No	No	No	25	Home
9	3.5	Yes	No	No	No	22	Home
10	3.8	Yes			No	6	Home
11	4.4	No	No	Yes	Yes[c]	9	Home
12	4.8	Yes	No	No	No	13	Home
13	5.4	No	No	No	No	7	Home
14	5.6	No			No	10	Home
15	5.8	No	No	No	Yes	6	Home
16	5.8	No			No	3	Home
17	6.1	No	No	No	No	13	Home
18	6.8	No			No	5	Home
19	7.9	Yes	No	No	No	73	Home
20	8.3	No			No	5	Home
21	8.6	Yes			No	5	Home
22	9.5	No			No	7	Home
23	9.5	Yes	No	No	Yes	15	Home
24	10.2	Yes			No	9	Home

Abbreviations: BPD, bronchopulmonary dysplasia; D/C Disp, discharge disposition; Hgb, hemoglobin; HIE, hypoxic-ischemic encephalopathy; IVH, intraventricular hemorrhage; LOS, length of neonatal intensive care unit (NICU) stay; PVL, periventricular leukomalacia.
[a]Posthemorrhagic hydrocephalus requiring ventriculoperitoneal shunt placement.
[b]Support withdrawn at 18 h of age (encephalopathic with fixed pupils).
[c]Therapeutic hypothermia. Blank cells indicate that no cranial imaging (head ultrasound, computer axial tomography (CAT) or magnetic resonance imaging (MRI)) was performed.

Table 5. Recommendations for improving care and outcomes of anemic neonates born after FMH

Area of improvement	*Potential means of improvement*
Heightened suspicion of FMH by the obstetrical staff	Consider FMH when mother reports lack of fetal movement. Perform a nonstress test or biophysical profile when mother reports lack of fetal movement. Consider FMH in cases with 'non-reassuring' fetal heart rate pattern after mother reports lack of fetal movement.
Improved availability of KB or flow cytometric testing for FMH	Advances in flow cytometric or other means of identifying and quantifying FMH. Availability of best test for FHM to all hospitals with labor and delivery services. More rapid turnaround/reporting of results.
Rapid and consistent communication that FMH is being considered by the obstetrical staff. Rapid communication of positive test results.	Communication between clinical laboratory and obstetrics, labor and delivery staff, pediatrics, neonatal resuscitation team, neonatology and blood bank.

Abbreviations: FMH, fetomaternal hemorrhage; KB, Kleihauer–Betke test.

pattern was seen in only 16% of the patients reviewed by Gacoia[23] and in only 3 (12.5%) of ours.

It is unclear whether flow cytometry should be performed on all women reporting absent fetal movement.[25,26] Laboratory confirmation of a hemorrhage could help prepare the pediatric providers and the transfusion service. However, on a practical level, many hospital laboratories do not have a rapid flow cytometric test for this purpose, and sending the specimen to a reference laboratory would require a delay. Moreover, the majority of women with a severe fetomaternal hemorrhage are delivered by emergent cesarean for 'non-reassuring' fetal heart rate patterns. Thus, in many cases there would not be time to perform flow cytometry and receive the result before the delivery. However, laboratory confirmation within a few hours after birth could be of value; providing clear evidence of the cause of the hypovolemia and estimating the volume of fetal blood lost.

We recognize weaknesses in our study, primarily the pitfalls prevalent in all retrospective analyses, including incomplete data, errors in initial recording and unintended introduction of bias based on the data elements selected for review. Strengths include the relatively large number of cases included, all within one health system, and an electronic database of integrated maternal and neonatal information. Surely, much discovery is needed before the best ways to diagnose and treat this condition are defined. Table 5 is our attempt to summarize needed areas of improvement, based on our study of the present 24 cases. We speculate that better outcomes could occur with heightened antenatal suspicion, more rapid diagnosis and better preparation of first responders including labor and delivery and pediatric nurses, pediatricians and neonatologists and blood banks.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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