How we treat: management of life-threatening primary postpartum hemorrhage with a standardized massive transfusion protocol

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Management of massive, life-threatening primary postpartum hemorrhage in the labor and delivery service is a challenge for the clinical team and hospital transfusion service. Because severe postpartum obstetrical hemorrhage is uncommon, its occurrence can result in emergent but variable and nonstandard requests for blood products. The implementation of a standardized massive transfusion protocol for the labor and delivery department at our institution after a maternal death caused by amniotic fluid embolism is described. This guideline was modeled on a existing protocol used by the trauma service mandating emergency release of 6 units of group O D– red cells (RBCs), 4 units of fresh frozen or liquid plasma, and 1 apheresis unit of platelets (PLTs). The 6:4:1 fixed ratio of uncrossmatched RBCs, plasma, and PLTs allows the transfusion service to quickly provide blood products during the acute phase of resuscitation and allows the clinical team to anticipate and prevent dilutional coagulopathy. The successful management of three cases of massive primary postpartum hemorrhage after the implementation of our new massive transfusion protocol in the maternal and fetal medicine service is described.

ABBREVIATIONS: D&C = dilatation and curettage; ICU = intensive care unit; MTP = massive transfusion protocol; PT = prothrombin time; PTT = partial thromboplastin time.

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Postpartum hemorrhage is an important and often preventable cause of maternal mortality and morbidity worldwide.[1,2] Primary postpartum hemorrhage is defined as hemorrhage occurring within 24 hours of delivery. At term, the uteroplacental circulation receives an estimated 700 mL of blood per minute, so it is not surprising that failure of the normal hemostatic mechanisms after delivery can result in life-threatening hemorrhage.[3] Major causes of postpartum hemorrhage include uterine atony, pathologic placental implantation, retained products of conception, uterine rupture, birth trauma, and existing or acquired coagulopathy.

In the modern era of fractionated blood component therapy, optimal management of massive hemorrhage is an evolving concept. Resuscitation of a patient with postpartum hemorrhage is conceptually similar to resuscitation after traumatic injury, where the goals are to establish rapid control of bleeding and restore systemic oxygen delivery. The trauma literature defines two phases of resuscitation: an immediate phase directly after injury with ongoing hemorrhage and a maintenance phase after stabilization.[4] Modern trauma resuscitation protocols advocate sequential administration of therapeutic components, beginning with colloid-crystalloid solutions in fused to replace lost intravascular volume. Second, red blood cells (RBCs) are transfused to restore oxygen-carrying capacity. Third, clotting factors and platelets (PLTs) are delivered to restore physiologic hemostasis. Rapid restoration of the components of the blood is essential for ensuring adequate tissue perfusion and preventing acidosis, coagulopathy, and hypothermia. Adequate replacement of plasma components is particularly important for avoiding dilutional coagulopathy in the massively bleeding patient.

Massive transfusion protocols have been adopted by many hospitals with accredited trauma centers.[5] The trauma center certification process administered by the American College of Surgeons Committee on Trauma (ACS-COT) stipulates that individual institutions address

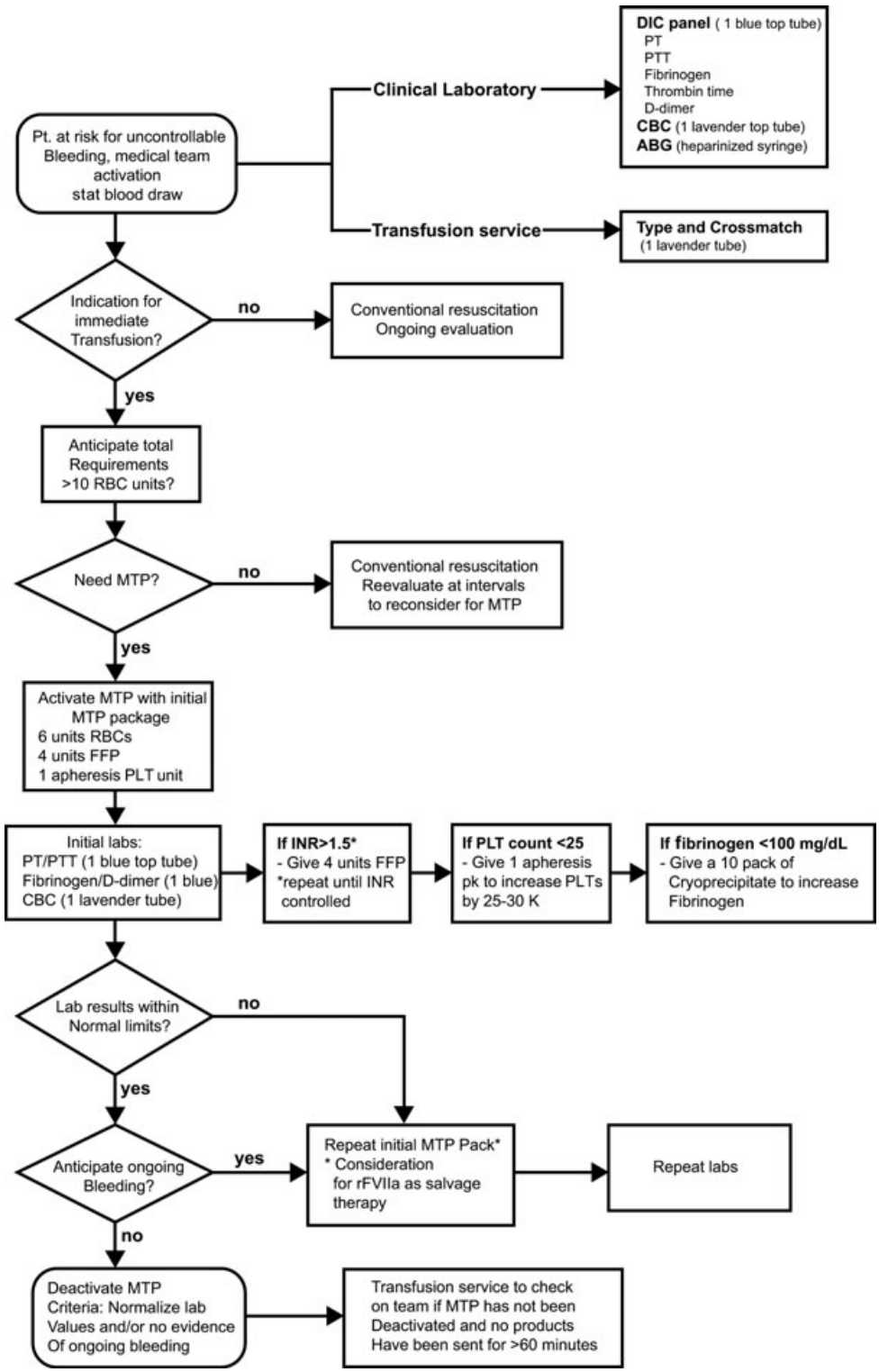

Fig. 1. Massive transfusion protocol algorithm. Pt. = patient; DIC = disseminated intravascular coagulation; CBC = complete blood count; ABG = arterial blood gas; INR = international normalized ratio.

the role of the transfusion service in support of the massively hemorrhaging patient.[6] The clinical team is responsible for activating the massive transfusion protocol when a trauma patient presents with life-threatening hemorrhage. Our massive transfusion protocol for the trauma service provides for emergency release of a "package" consisting of 6 units of RBCs, 4 units of plasma (liquid or fresh-frozen), and 1 apheresis PLT unit (Fig. 1). The entire

complement of blood products can be compiled, electronically issued, and delivered to the operating room, delivery room, or emergency department in less than 15 minutes.

The 6:4:1 ratio of RBCs, plasma, and PLTs was designed to replace approximately 70 percent of the total RBC volume and 60 percent of the total circulating plasma volume of a 70 kg individual. The 6:4:1 combination of RBCs, plasma, and apheresis PLTs approximates a 60:40 volume-to-volume ratio of plasma to RBCs. Whole blood is theoretically the ideal replacement therapy for massive hemorrhage. The 6:4:1 ratio of blood products approximates the composition of whole blood with a hematocrit level of 40 percent.

Blood draws for essential coagulation parameters are integrated into the massive transfusion protocol. Figure 1 illustrates the massive transfusion protocol (MTP) flowchart, where the obstetrical anesthesiologist sends initial laboratory tests to the clinical laboratory upon initiation of the massive transfusion protocol. The laboratories include prothrombin time (PT), partial thromboplastin time (PTT), fibrinogen, D-dimer, and a complete blood count. If a patient continues to hemorrhage or future hemorrhage is anticipated, additional 6:4:1 packages can be requested from the transfusion service provided that additional coagulation laboratories are sent with each request (Fig. 1). Importantly, the massive transfusion guideline does not preclude customized ordering of other blood products and pharmaceuticals. Later in resuscitation, abnormal laboratory values such as prolonged PT and/or PTT, low PLT count, and low fibrinogen values can be addressed individually as depicted in Fig. 1.

To provide rapid delivery of plasma in the MTP package, four units of thawed fresh-frozen plasma (FFP) for blood groups A and O are continuously in inventory. For patients who are blood group B or AB, or whose blood type is unknown, liquid plasma is initially substituted for FFP. The RBCs and plasma units earmarked for the massive transfusion protocol are checked daily for integrity and expiration dates.

We instituted our massive transfusion protocol in the labor and delivery service to expedite and optimize delivery of blood products during the acute, immediate phase of resuscitation for patients with uncontrolled, primary postpartum hemorrhage. This guideline was initiated after the case of a woman with severe hemorrhage and coagulopathy after cesarean delivery of twins.

CASE 1

Patient 1 presented as a 49-year-old Asian woman gravida 9, para 4044 (9 pregnancies, 4 live births, 0 preterm births, 4 miscarriages and/or therapeutic abortions, and 4 living children). She was pregnant with diamniotic and/or dichorionic twins and was initially hospitalized at 33 weeks' gestation for persistent vaginal bleeding secondary to a presumed chronic placental abruption. At 35 weeks 6 days' gestation she underwent induction of labor for nonreassuring fetal heart tracings and intermittent vaginal bleeding. She failed to progress and proceeded to low transverse cesarean section. She delivered twin A with Apgar scores of 7 and 8 at 1 and 5 minutes. Twin B was delivered with Apgar scores of 9 and 9. The patient tolerated the procedure well and was taken to the recovery room in good condition.

After 5 minutes in the postanesthesia recovery unit, she developed increasing vaginal bleeding that was unresponsive to administration of multiple uterotonics. She rapidly became hypotensive and was transferred to the operating room for emergent dilatation and curettage (D&C). The patient suddenly went into cardiac arrest and she was shocked into sinus rhythm. She was resuscitated with large volumes of crystalloid hetastarch solution, followed by infusion of uncrossmatched RBCs. Because the patient was blood group B, and no group B FFP was immediately available, FFP was delivered 45 minutes after the request due to the time required for thawing this product. After initial resuscitation, the obstetrical team performed a supracervical hysterectomy. Diffuse microvascular oozing from the uterus and pelvic side walls occurred throughout the procedure. Two sequential 4.8-mg bolus doses of recombinant factor (rF)VIIa were administered in succession approximately 30 minutes after her initial hemorrhage. Laboratory results showed florid disseminated intravascular coagulation (Table 1). The patient became acidotic and hypothermic during the hysterectomy procedure and suffered an estimated 10 L of blood loss. The patient went into ventricular fibrillation and was again successfully defibrillated.

After surgery, she was again returned to the intensive care unit (ICU) where her abdomen became tense as blood reaccumulated. She became increasingly acidotic and hypothermic. Nearly 13 hours after her cesarean section she developed asystole and was pronounced dead after prolonged resuscitation attempts. Seventy-seven units of RBCs, 37 units of FFP, 9 pools of cryoprecipitate (10 units each for each pool), and 10 apheresis PLT units were administered during her cesarean section, D&C, hysterectomy, and resuscitation. Table 1 shows that despite restoration of blood pressure and RBC volume in this patient, she developed severe coagulopathy, hypothermia, and tissue hypoxia that were refractory to replacement therapy. Postmortem examination revealed cytokeratin-positive fetal amniocytes within the pulmonary vasculature, consistent with amniotic fluid embolism.

Comment

The pathophysiology of amniotic fluid embolism is controversial.[7,8] Maternal-fetal barriers can be compromised

TABLE 1. Cumulative transfusion products, laboratory values, and vital signs for Patient 1

	Laboratory values												
Time	RBCs (total number of units)	Plasma (total number of units)	Cryo-precipitate (total number ×10)	PLTs (total number of units)	Hb (mg/dL)	PT (sec)	PTT (sec)	PLTs ($\times 10^9$/L)	Fibrinogen (mg/dL)	D-dimer (ng/mL)	MAP (mmHg)	pH	Temperature (°C)
12:10	0	0	0	0	10.7						105		37
13:05	0	0	0	0	9.9			131			60		
13:45	2	0	0	0							30		
14:15	13	0	0	0	8.9	>300	>300	27	<50	>20K	0		
14:23	13	0	0	1	6.5						70	6.77	
14:47	20	0	0	1	8.2						110	7.50	
15:04	23	4	0	1	7.1	>100	>300	26	<50	>20K	70	7.43	
15:50	29	8	3	4	7.5	13.2	66.4	119	157	>20K	80	7.37	
17:30	39	13	4	6	12.4	13.7	58.4		165		68	7.26	34.3
21:05	45	17	5	6	7	17.0				9218	71		34.3
21:25	49	17	7	6	7.7						83	7.07	34.5
22:30	51	19	7	6	12.1						80	7.18	
23:00	54	19	7	6	6.4						105	7.50	
23:55	58	23	8	8	7.2	26.1	107	16	112		90		34.7
00:30	69	27	8	9							80	7.30	34.4
01:50	77	37	9	10	3.7	24.5	124	21		949	84	7.13	33.5

MAP = mean arterial pressure.

at any time during labor, resulting in escape of amniotic fluid into the maternal venous circulation. Patients with severe amniotic fluid embolism experience a biphasic clinical course: immediate hyperacute cardiovascular collapse followed by onset of profound disseminated intravascular coagulation. If a patient survives initial cardiopulmonary collapse, the thromboplastinlike substances in amniotic fluid are thought to trigger widespread intravascular activation of FVII with subsequent uncontrolled activation of the extrinsic coagulation pathway culminating in disseminated intravascular coagulation.[9]

CASE 2

Patient 2 presented as an 18-year-old Caucasian woman gravida 1, para 0102. She was pregnant with monoamniotic-dichorionic twins and admitted for spontaneous onset of labor at 36 weeks' gestation. Spontaneous vaginal delivery of twin A with Apgar scores of 8 and 9 was followed by twin B with Apgar scores of 8 and 9. No cervical or vaginal lacerations were noted. After delivery she was transported to a close observation unit where brisk vaginal blood flow was noted and her uterus was found to be boggy and large. She immediately received multiple doses of uterotonic agents. Hemorrhage persisted despite this pharmacotherapy so the new MTP was initiated. The patient returned to the labor and delivery operating room to undergo emergent D&C. The patient was resuscitated with crystalloid solution and immediately received a 7.2-mg bolus of rFVIIa before the D&C procedure. Her total estimated blood loss was 2 L. All blood products in the massive transfusion package were not administered because she recovered rapidly. In all, she received 6 units of RBCs and a single apheresis PLT unit. Laboratory studies (Table 2) showed no evidence of disseminated intravascular coagulation. She was stabilized and transferred to the ICU in stable condition. She was discharged 3 days after delivery in good condition.

Comment

Case 2 described a woman with postpartum uterine atony and hemorrhage which may not technically qualify as massive and life-threatening. Nonetheless, a massive transfusion guideline was initiated by the obstetrician in this case and hemorrhage was controlled by administration of uterotonics, blood replacement therapy, and rFVIIa. Uterine atony is the most common cause of postpartum hemorrhage, occurring in approximately 1 in 20 singleton deliveries.[10,11] Most cases of postpartum uterine atony are mild and self-limited, responding to administration of the uterotonic agents oxytocin, prostaglandin, and ergot alkaloids. Some cases of atony, however, are severe and refractory to therapy, requiring radical intervention

TABLE 2. Cumulative blood products, laboratory values, and vital signs for Patient 2

	Laboratory values												
Time	RBCs (total number of units)	Plasma (total number of units)	Cryo-precipitate (total number ×10)	PLTs (total number of units)	Hb (mg/dL)	PT (sec)	PTT (sec)	PLTs ($\times 10^9$/L)	Fibrinogen (mg/dL)	D-dimer (ng/mL)	BP (mmHg)	pH	Temperature (°C)
5:00	0	0	0	0	13.5			189					
10:50	6	0	0	1	9.9			137			100/80		
11:00	6	0	0	1	12.1						155/100	7.32	37
13:30	6	0	0	1	13.5	8.7	31.9	140	357	7304	150/85		
20:25	6	0	0	1		10.9	32.4		311	5584			

BP = blood pressure.

including uterine artery embolization, and emergent hysterectomy.

CASE 3

Patient 3 presented as a 37-year-old Caucasian woman gravida 2, para 1001. She was admitted at 38 weeks 2 days for a scheduled singleton cesarean delivery. She underwent primary low transverse cesarean section and delivered a liveborn male with Apgar scores of 7 and 9. Approximately 1 hour after delivery, while in the observation unit, the patient developed increasing vaginal bleeding. Rectal and intramuscular uterotonic agents were administered but failed to control her bleeding. Coagulation studies (Table 3) showed evidence of disseminated intravascular coagulation with a PT of 16.0 seconds, PTT of 53.3 seconds, a fibrinogen level of 86 mg per dL, and D-dimer level greater than 20,000 ng per dL. Our massive transfusion protocol was initiated. The patient was taken to the operating room for emergent hysterectomy to control bleeding after she was deemed too unstable for transport to interventional radiology for embolization. A supracervical hysterectomy was completed without complications. The patient had 3 L of estimated blood loss during the procedure and she was brought to the ICU in stable condition. In total, 16 units of RBCs, 15 units of FFP, 1 pool of 10 units of cryoprecipitate, and 5 apheresis PLT units were transfused. The patient was discharged in stable condition on Postoperative Day 4. Pathologic examination of the hysterectomy specimen demonstrated focal invasion of the myometrium by chorionic villi consistent with placenta accreta.

Comment

Abnormal placental implantation, including placenta increta, accreta, and percreta, occurs in approximately 0.2 percent of pregnancies and the incidence is reported to be increasing, presumably as a result of increasing rates of cesarean section.[12] Both placenta accreta and retained products of conception may result in primary postpartum hemorrhage refractory to uterotonic therapy. Retained products of conception are often successfully treated with D&C. In contrast, placenta accreta with postpartum hemorrhage is most often managed by total hysterectomy.[13]

CASE 4

Patient 4 presented as a 30-year-old female gravida 2, para 1012. Her diamniotic-dichorionic twin pregnancy after in vitro fertilization was complicated by mild intrahepatic cholestasis of pregnancy. She was admitted at 37 weeks 1 day gestation for elective cesarean section. Twins A and B were liveborn males with excellent Apgar scores. During cesarean section a heart-shaped, bicornuate uterus was

TABLE 3. Cumulative transfusion products, laboratory values, and vital signs for Patient 3

	Laboratory values												
Time	RBCs (total number of units)	Plasma (total number of units)	Cryo-precipitate (total number ×10)	PLTs (total number of units)	Hb (mg/dL)	PT (sec)	PTT (sec)	PLTs ($\times 10^9$/L)	Fibrinogen (mg/dL)	D-dimer (ng/mL)	MAP (mmHg)	pH	Temperature (°C)
8:10	0	0	0	0	13.8	10.1	29.7	158	287	>20K	82		
10:15	0	0	0	0	8.9	15.9	53.3	121	86	>20K	86		37
11:39	7	8	0	1	8.5	16.0	62.3	100	93	>20K	65		
12:10	7	8	0	1	8.4						80	7.21	
12:30	11	12	0	3	7.6			79			75		
12:39	16	15	0	3	8.6						75	7.42	
13:18	16	15	0	5	10.5	14.0	39.0	150	192	>20K	90		35.2
14:30	16	15	0	5	13.5						112	7.26	36.2
15:50	16	15	1	5							130	7.36	
16:45	16	15	1	5	13.6	9.6	31.4	137			118		36.2
19:55	16	15	1	5							120	7.40	36.7
21:00	16	15	1	5	13.8	10.1	29.7	158	287	>20K	111		37.2

MAP = mean arterial pressure.

identified. After removal of the twin placenta there was marked atony of both uterine cornua. Administration of a series of uterotonic agents and continuous uterine massage failed to ameliorate uterine atony and resultant hemorrhage. Our massive transfusion guideline was activated. A bolus of 6 mg of rFVIIa was administered 40 minutes after her initial hemorrhage. Initial laboratories obtained after activation of the massive transfusion protocol showed disseminated intravascular coagulation with a D-dimer level greater than 20,000 ng per mL, a fibrinogen level of 78 mg per dL, PT of 16.6 seconds, PTT of 131 seconds, and a PLT count of 60×10^9 per L. She suffered an estimated 6 L of blood loss during her initial hemorrhage.

The patient underwent emergent cesarean supracervical hysterectomy. Moderate oozing from the pelvic sidewall was noted at the outset of the procedure and an additional 6 mg of rFVIIa was administered 95 minutes after the initial dose. The abdomen and vagina were packed and she was sent to the ICU in stable condition. In all, she received 16 units of RBCs, 7 units of FFP, 2 pools of cryoprecipitate 10-packs, and 3 apheresis PLT units during her cesarean section and hysterectomy. Pathologic examination of the uterus showed no abnormalities. She recovered without complications and was discharged on Postoperative Day 6.

Comment

Case 4 showed rapid development of a life-threatening, florid coagulopathy during postpartum hemorrhage caused by uterine atony. This patient developed intractable uterine atony necessitating emergent total abdominal hysterectomy. Adequate clotting factor replacement to control bleeding in the context of ongoing DIC during the hysterectomy procedure was critical for this case.

DISCUSSION

Our index case and subsequent three cases illustrate the clinical utility of a standardized MTP for life-threatening postpartum hemorrhage. Examination of the coagulation test results over time (Tables 3 and 4) indicate that empiric administration of RBCs, plasma, and PLTs ameliorated dilutional coagulopathy and disseminated intravascular coagulation in Cases 3 and 4. Resuscitation of massive blood loss requires plasma repletion to reverse dilutional coagulopathy after large volume infusion of colloids and/or crystalloids. Routine underutilization of plasma during massive hemorrhage is described in the trauma literature.[14] Moreover, a recently published mathematical model of whole-blood loss during hemorrhagic shock advocates unit-for-unit coadministration of plasma and RBCs during resuscitation to reverse dilutional coagulopathy.[15] Therefore, plasma and PLTs are delivered along-

TABLE 4. Cumulative transfusion products, laboratory values, and vital signs for Patient 4

	Laboratory values												
Time	RBCs (total number of units)	Plasma (total number of units)	Cryo-precipitate (total number ×10)	PLTs (total number of units)	Hb (mg/dL)	PT (sec)	PTT (sec)	PLTs ($\times 10^9$/L)	Fibrinogen (mg/dL)	D-dimer (ng/mL)	BP (mmHg)	pH	Temperature (°C)
16:45	16	15	1	5	13.6	9.6	31.4	137			118		36.2
14:00	0	0	0	0	11.4						125/70		
15:30	8	4	0	0							78/40		
16:50	12	4	0	1	10.5	16.6	131		78	>20K	100/40		
18:00	12	4	0	1	10.9	14.3	56.6	186	110	>20K	100/45		
18:10	13	7	0	1	10.5						95/60	7.14	33.3
19:10	13	7	2	1	9.4						100/70	7.34	34.3
20:00	13	7	2	3	7.7			145			100/70		35.1
21:00	14	7	2	3							110/75	7.39	35.3
02:00	16	7	2	3	9.0	10.4	29.9	117		5584			36.4

BP = blood pressure.

side RBCs in the MTP package to facilitate plasma repletion.

FFP requires up to 45 minutes to thaw, issue, and transport to the labor and delivery ward. This delay is problematic in the context of sudden, unexpected hemorrhage in an obstetrical patient. In contrast to trauma patients, who arrive at the hospital with advance warning, postpartum hemorrhage most often occurs suddenly and unexpectedly.[16,17] Risk factors for primary postpartum hemorrhage are known, but massive postpartum hemorrhage is unpredictable and many cases occur in women without risk factors. In our experience, liquid plasma functions as an excellent alternative to FFP in cases of massive postpartum hemorrhage.

Liquid plasma has distinct advantages over FFP.[18] Liquid plasma is refrigerated and never frozen. Therefore, it can be issued immediately for emergency release. A constant supply of group AB liquid plasma in our hospital transfusion service permits immediate issue of this product without the lengthy thawing process required for FFP. For whole blood collected in adenine saline preservative solution, liquid plasma preserved in citrate-phosphate-dextrose has a 26-day outdate compared to a 5-day shelf life for thawed plasma.[19] Never-frozen liquid plasma is reported to contain virtually normal activity for all coagulation factors with the exception of diminished levels of FV and FVIII. Plasma FVIII activity decreases by 50 percent in the first 24 hours of storage at 4°C and then remains stable for up to 1 week.[20] Thawed FFP is reported to retain 50 percent FV and 10 to 50 percent FVIII activity.[21] Because the process of freezing is reported to have little effect on the activity of plasma coagulation proteins, we predict that thawed FFP and liquid plasma contain comparable levels of the labile clotting FV and FVIII.[22] Although liquid plasma is not recommended for therapy in patients who are specifically deficient for FV or FVIII, this product can replace vital coagulation factors during acute hemorrhage. FVIII is an acute phase reactant that is frequently elevated in trauma.[23,24] Moreover, PLTs contain large amounts of membrane-bound FV and coadministration of PLTs may compensate for decreased concentrations of FV in liquid plasma.[25] Although no randomized clinical trial supports the equivalence of liquid plasma and FFP in resuscitation of hemorrhagic shock, we believe that the benefits of immediate plasma availability outweigh potential risks of diminished levels of coagulation FV and FVIII.

In our experience, the results of laboratory coagulation testing (PT, PTT, fibrinogen, and PLTs) are best used to guide replacement therapy during the maintenance phase of resuscitation. Threshold coagulation parameters, such as prolongation of the PT and PTT to above 1.5 times the normal range, PLT counts below 25×10^9 per L, and fibrinogen concentrations below 100 mg per dL, are reproducible and time-tested tools for guiding optimal

fractionated blood product therapy.[26,27] Laboratory data, however, are usually unavailable during the immediate phase of resuscitation because of the time required for the hospital laboratory to perform these tests. A stat blood sample if sent to the clinical laboratory when a massive transfusion protocol is activated. Coagulation (PT, APTT, fibrinogen, D-dimer) and hematology (complete blood count) testing at our institution require that a blood sample be sent to the laboratory via a pneumatic tube system where the specimen is accessioned, aliquoted, transported to instruments, and analyzed. The national standard for turnaround of a stat laboratory test is 60 minutes.[28] Even "super-stat" coagulation results are typically available in 15 to 30 minutes.[29] Thromboelastography, although useful for monitoring coagulopathic patients, is too slow to be useful during the immediate phase of resuscitation.[30] For these reasons, laboratory values are helpful only during the maintenance phase of resuscitation. At present, our massive transfusion guideline allows the anesthesiologist and obstetrician to guide resuscitation of the patient with clinical acumen and real-time monitoring data during the dynamic and unstable hyperacute phase of resuscitation.

The role of rFVIIa in primary postpartum hemorrhage is controversial. Although no prospective, randomized clinical trials have been conducted, several case series and retrospective case audits demonstrate efficacy and safety of rFVIIa in the maternal population.[31-35] In Case 1, rFVIIa was administered as rescue therapy 30 minutes after estimated blood loss of 10 L. In Case 2, rFVIIa was used as initial therapy at the discretion of the clinical team. rFVIIa was not used in Case 3. rFVIIa was given as rescue therapy in Case 4 approximately 40 minutes after 3 L of blood loss. These cases demonstrate that rFVIIa is currently being administered both as initial and as salvage therapy in women with life-threatening primary postpartum hemorrhage. The cases also illustrate the variable and nonstandard administration of pharmaceuticals even in the setting of a standardized massive transfusion protocol. Oversight of rFVIIa administration in the setting of massive transfusion remains an area of opportunity for transfusion medicine physicians.[36,37]

CONCLUSION

Severe postpartum hemorrhage is an unpredictable and potentially catastrophic complication of childbirth. Standardized massive transfusion protocols simplify and expedite delivery of life-saving blood products to an acutely hemorrhagic obstetrical patient. Optimal treatment and resuscitation of postpartum hemorrhage requires a multidisciplinary clinical approach and effective communication with the transfusion service. Advances in the treatment of hemorrhagic shock, including novel hemostatic technologies, are likely to improve survival in massive postpartum hemorrhage.[38,39] Rapid, early, and definitive intervention is required to optimize patient outcomes for primary postpartum hemorrhage, achieved in part by implementation of a massive transfusion protocol for the hospital labor and delivery service.

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