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Case Report

Massive blood transfusion in a post cesarean patient with placenta praevia

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KEYWORDS

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Abstract Massive blood transfusion is replacement of a patient's total blood volume in less than 24 h, or acute administration of more than half the patient's estimated blood volume per hour. This case report describes the clinical profile, laboratory parameters and management of a post operative patient who lost nearly double her blood volume in 36 h. A 27 yr old Indian woman with no history of any medical illness, or co-morbidities underwent elective cesarean section for placenta praevia totalis. The maximal allowable blood loss calculated for her weight with target haemoglobin of 10 g % was 450 ml. Though adequate haemostasis was achieved before closure, she had significantly increased bleeding PV 1 h post-operatively. Over the next 24 h she underwent a subtotal and then a total hysterectomy. Blood loss within this period amounted to 6500 ml. The following day she lost 1900 ml blood through the drains. As is the approach to a surgical patient with massive blood loss, it was ensured that the surgical cause of bleeding is controlled with simultaneous blood component therapy so as to maintain haemostasis, blood oxygen carrying capacity, oncotic pressure and plasma biochemistry. The patient received a total of 18 units Packed Red Blood Cells, 20 units Fresh Frozen Plasma and six platelets over 36 h. The patient showed good recovery. Key points in therapy include control and management of underlying cause, supportive treatment with appropriate blood components for maintaining tissue perfusion and oxygenation.

1. Introduction

Massive blood loss is usually defined as the loss of one blood volume within a 24 h period [1], normal blood volume being approximately 7% of ideal body weight in adults and 8–9% in children. Alternative definitions include 50% blood volume loss within 3 h or a rate of loss of 150 ml/min [2].

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It is imperative to recognise major blood loss early and institute effective action promptly if shock and its consequences are to be prevented.

The aim of transfusion is maintenance of tissue perfusion and oxygenation by restoration of blood volume and haemoglobin, arrest of bleeding by treating any traumatic, surgical or obstetric source judicious use of blood component therapy to correct coagulopathy [3].

2. Case presentation

A 27 yr old Indian woman with no past history of any medical illness, or co-morbidities underwent elective cesarean section for placenta praevia totalis. She had history of previous CS (for breech) under spinal anaesthesia and ovarian cystectomy under general anaesthesia. Peri-operative period for both surgeries were uneventful. Pre-anaesthetic evaluation did not reveal any abnormal findings. Pre-operative haemoglobin was 11.0 g %, blood group O +ve, and coagulation profile was normal. Written informed consent for surgery and anaesthesia was taken and the possibility of hysterectomy/placenta accreta was explained.

LSCS was performed under subarachnoid block. Estimated Blood Loss (EBL) was 750 ml; maximal allowable blood loss (MABL) was 440 ml for target Hb of 10 g %. One unit PRBC was given during the surgery. Good haemostasis was achieved, uterus well contracted, patient was shifted to ICU on syntocinon infusion. Intra-operative period was uneventful.

An hour after completion of surgery, she developed bleeding per vaginum. The bleeding was transiently reduced following carboprost injection, but recurred after 2 h.

Decision to perform subtotal hysterectomy was taken. At that time the patient had tachycardia (HR – 130/min, regular), but was hemodynamically stable (BP – 110/80).

Surgery revealed friable and bleeding lower uterine segment with adherent placental tissue. Subtotal hysterectomy was done, during which the patient was transfused with three units packed cells. Two wide bore peripheral lines (14 G) and a radial arterial line were inserted. Estimated blood loss during surgery was 1500 ml, urine output was 200 ml. Post-operatively drains continued to fill and four units PRBC were given, along with two units of Fresh Frozen Plasma (FFP) in the immediate post operative period. Differential diagnoses considered were surgical bleeding or disseminated intravascular coagulation (DIC).

Surgical and medical consultations sought were of the opinion that conservative management be continued as the clinical picture favoured DIC. After about 11 h postoperative, it was decided to electively intubate & ventilate in view of continued bleeding and severe anaemia, so as to reduce the work of breathing. By next morning, the patient had lost 4500 ml blood via the drains and had received 12 units PRBC, 10 units FFP, four units platelets. Urine output continued to decline and drains continued to fill. Emergency exploratory laparotomy was planned to rule out surgical cause of bleeding. Another three units FFP were given prior to shifting to the operation theatre (OT). Intra-operative findings revealed haemoperitoneum with about 1250 ml blood and clots along with oozing from raw surface of the stump. Haemostatic sutures were inserted and haemostasis established. Two units PRBC, one unit FFP and one unit platelet concentrate were given inside the theatre. The patient was shifted to ICU and continued on ventilatory support. Bleeding continued from drains for another 2–3 h after which it gradually reduced and stopped.

Patient was extubated 4 h after laparotomy.

Post-operatively the patient received another four units PRBC, two units FFP and one unit platelet. She was also given 20 ml of calcium gluconate after documenting hypocalcemia. (Serum Calcium-6.5 mg %). Throughout the peri-operative period and ICU stay, the laboratory parameters (Table 1) and arterial blood gases (Table 2) were regularly monitored. Total blood loss over 36 h was 8400 ml while the patients estimated blood volume (66×70) was 4620 ml.

The patient received a total of 18 units Packed Red Blood Cells, 20 units Fresh Frozen Plasma and six platelets over 36 h (Table 1). She showed good recovery, and after another 3 days in the ICU and 2 days in the ward she was discharged. Her last report before discharge from the ICU showed Hb of 11.3 g %, haematocrit of 32.6, platelet count 74,000/cu mm, and PT/INR, APTT within normal limits. The patient was last seen 5 months after surgery and was doing well.

3. Discussion

Massive transfusion involves the selection of the appropriate amounts and types of blood components to be administered, and requires consideration of a number of issues including volume status, tissue oxygenation, management of bleeding and coagulation abnormalities, as well as changes in ionised calcium, potassium, and acid–base balance.

Table 1 Blood components transfused and laboratory parameters.

Date/Time	Hb	Platelets	PT/INR	APTT	Blood component
D1 Intraop (LSCS)	11	206	14/1.2	30.8	1 PRBC
16:30 Intraop (Subtotal hyst)	6.9	150	NA	NA	3 PRBC
19.00	NA	NA	22.5/2.1	43.8	2 PRBC, 2FFP
21.50	7.7	106	21/1.9	33.2	2 PRBC, 4 FFP
D2 – 00.30	5.2	30	NA	NA	2 PRBC, 4 Platelets
03.30	6.5	65	14.2/1.13	35.6	2 PRBC, 4 FFP
07.30	NA	NA	20.4/1.8	41	3 FFP
08.30 Intraop (Laparotomy)	6.2	85	13.9/1.11	44.6	2 PRBC, 1 FFP, 1 Platelet
12:15	4.6	50	17.1/1.45	50	2 PRBC, 2 FFP, 1 Platelet
15:30	10.5	60	16.5/1.28	39.5	2 PRBC
D3-08:00	11.3	74	NA	NA	

Table 2 Arterial blood gases on Day 1, Day 2 and Day 3.

Date/Time	PaO2	PCO2	pH	HCO3	BE	Hb	Comment
D1-14:12	351	34	7.31	17	−8.7	6.5	Post hysterectomy
16:00	107	26	7.35	16	−1.0	7.7	
20:30	171	23	NA	NA	NA	5.5	
D2-00:30	175	27	7.46	21	−4	7.3	
03:00	559	33	NA	NA	NA	7.5	Post intubation
06:30	172	30	7.45	22	−2.4	6.8	
10:15	185	35	NA	NA	NA	5.1	Post laparotomy
17:00	116	35	7.46	23	−1.2	10	Post extubation
D3-08:30	78	29	7.47	23	−2.0	10.8	On room air
D4-09.30	155	26	7.49	22	−2.5	10.8	

3.1. Therapeutic aims

The purpose of blood transfusion is to improve the oxygen-carrying capacity of the blood. During the operative and immediate postoperative period, intravascular volume and haemoglobin concentration may change rapidly. The over-riding first requirement is maintenance of tissue perfusion and oxygenation, which is critical in preventing the development of hypovolaemic shock and consequent high mortality from multi-organ failure [4].

3.2. Monitoring

Frequent assessment of changing intravascular volumes, oxygen delivery and patient physiology is required [4]. Blood samples should be sent to the laboratory at the earliest possible opportunity for blood grouping, antibody screening and compatibility testing, as well as for baseline haematology, coagulation screen and biochemistry investigations. When dealing with an evolving process it is important to check parameters frequently, (and after each therapeutic intervention) to monitor the need for and the efficacy of component therapy [4]. Temperature monitoring is important as hypothermia increases the risk of end organ failure and coagulopathy and may be prevented by pre-warming of resuscitation fluids, patient warming devices such as warm air blankets and the use of temperature controlled blood warmers (Grade C recommendation, Level IV evidence).

3.3. Component therapy

Allogenic blood from volunteer donors is a limited and valuable resource that must be used carefully, appropriately and safely.

3.4. Red blood cells

The function of red cells is oxygen delivery to tissues; they should not be used as a volume expander [5]. Red cells also contribute to haemostasis by their effect on platelet margination and function. The optimal haematocrit to prevent coagulopathy is unknown, but experimental evidence suggests that a relatively high haematocrit, possibly 35, may be required to sustain haemostasis in patients with massive blood loss [6,7]. Red cell transfusion is likely to be required when 30–40% blood volume is lost; over 40% blood volume loss is immediately life-threatening [8].

3.5. Platelets

Expert consensus advises that the platelet count should not be allowed to fall below the critical level of 50×10^9/l in the acutely bleeding patient [9] (Grade C recommendation, level IV evidence), and this is endorsed by the British Committee for Standards in Haematology (BCSH) guidelines for the use of platelet transfusions[10]. A platelet count of 50×10^9/l may be anticipated when approximately two blood volumes have been replaced by fluid or red cell components [11] but there is marked individual variation.

3.6. Fresh frozen plasma

Coagulation factor deficiency is the primary cause of coagulopathy in massive transfusion because of dilution of coagulation factors following volume replacement with crystalloid or colloid and transfusion of red cell components. The level of fibrinogen falls first; the critical level of 1.0 g/l is likely to be reached after 150% blood volume loss, followed by the fall of other labile coagulation factors to 25% activity after 200% blood loss [6]. Prolongation of the activated partial thromboplastin time (APTT) and prothrombin time (PT) to 1.5 times the mean normal value is correlated with an increased risk of clinical coagulopathy [12]. Although 'formula replacement' with fresh plasma is not recommended, it may be required in situations where rapid turnaround of coagulation tests cannot be guaranteed. Infusion of FFP should be considered after one blood volume is lost [13].

3.7. Complications of massive transfusion

Include coagulopathy, caused by a dilutional effect on the host's clotting factors and platelets, as well as the lack of platelets and clotting factors in packed red blood cells, volume overload, hypothermia, hyperkalemia, metabolic alkalosis and hypokalemia due to transfusion of a large amount of citrated cells and hypocalcemia. Complex metabolic changes may occur due to hypovolaemia, hypothermia and the infusion of large volumes of stored red cells and blood products, especially plasma. The commonest is ionised hypocalcaemia due to citrate toxicity [14]. Reduced ionised calcium reduces myocardial contractility, causes vasodilation and exacerbates further bleeding and shock. Hyperkalaemia may occur, due to the high extracellular potassium concentration in stored red cell units. This may be compounded by oliguria and the metabolic acidosis associated with shock [15].

Table 3 Key recommendations - British Committee for Standards in Haematology [3].

Goal	Procedure	Comments
Restore circulating volume	Wide bore IV/central access	Consider arterial line
	Pre warmed colloid/crystalloid	Monitor CVP
	Avoid hypotension	Warm patient
	Urine output > 0.5 ml/kg/h	Look for concealed bleeds
Contact key personnel	Attending clinician	ICU admission
	Anaesthetist	Designated person responsible for communication &
	Haematologist	Documentation
	Blood bank	
Arrest bleeding	Early surgical/OBG intervention	
	Interventional radiology	
Laboratory investigations	CBC, PT, APTT, TT, Fibrinogen,	Correct patient identity
	ABG, biochemical profile	Colloids may alter results
		May need to give components before results available
Maintain platelets > 75	Anticipate platelet < 50 after 2 x	Allow margin of safety
	Blood volume replacement	Aim for platelet > 100 for multi/CNS trauma
Maintain Hb > 8	Assess urgency	
	Blood salvage	
	Group O; Rh −ve	In emergency; O +ve acceptable
		In males/post menopause
	ABO group specific once known	
	Fully compatible blood	
	Use blood warmer/rapid infuser	Time permitting
		If flow rate > 50 ml/kg/h in adults
Maintain PT & APTT < 1.5 × control	FFP-12–15 ml/kg	PT/APTT > 1.5 × control correlates with increased micro-vascular bleeding
	Anticipate need for FFP after 1–1.5 × blood vol replaced	Keep ionised Ca > 1.13 mmol/l
Maintain fibrinogen > 1.0 g/l	If not corrected by FFP give 2 pools cryoprecipitate	Rarely needed except in DIC
Avoid DIC	Correct precipitating causes: shock, hypothermia, acidosis	Rare but has high mortality

Coagulopathy associated with massive transfusion remains an important clinical problem. It is an intricate, multifactorial and multicellular event. Disseminated intravascular coagulation is a feared complication in the acutely bleeding patient. The cardinal clinical sign of DIC is microvascular oozing, whilst microthrombi in small vessels can result in end-organ damage. A DIC-like syndrome can result from the activation of the coagulation cascade secondary to tissue trauma, resulting in excessive consumption of platelets and coagulation factors. Patients at particular risk are those with tissue damage due to prolonged hypoxia, hypovolaemia or hypothermia, and those with massive head injury or extensive muscle damage [7]. This syndrome carries a high mortality, and is difficult to reverse. Prolongation of the PT and APTT in excess of that expected by dilution, together with significant thrombocytopenia and fibrinogen of < 1.0 g/l are highly suggestive of a developing DIC-like state and hence frequent estimation of platelet count, fibrinogen, PT and APTT is strongly recommended. Measurement of D-dimer may also be useful in providing an early warning. Laboratory evidence of a consumption coagulopathy should be sought before microvascular bleeding becomes evident, so that appropriate and aggressive action can be taken to address the underlying cause [16]. Treatment strategies include the maintenance of adequate tissue perfusion, the correction of hypothermia and anaemia, and the use of haemostatic blood products including platelets, FFP and cryoprecipitate to correct microvascular bleeding, taking care to avoid circulatory overload [7].

Transfusion-related acute lung injury (TRALI) and other acute immunologically mediated reactions are uncommon, but occur 5–6 times more frequently following administration of platelets and FFP than red cells [15]. It is a severe acute reaction characterised by respiratory distress, hypoxia and pulmonary infiltrates soon after transfusion with no other apparent cause. The reported incidence varies from 0.001% to 0.16% per patient transfused [16].

Acute haemorrhage leading to acute hypovolaemic shock is a medical emergency associated with a high mortality, and requires prompt and effective treatment [16]. A methodological approach to a patient with massive blood loss is essential for proper management. British Committee for standards in Haematology key recommendations are given in Table 3. Key points in therapy include control and management of underlying cause, supportive treatment with appropriate blood components for maintaining tissue perfusion and oxygenation. In this case too surgical bleed was considered and the patient re-explored, while simultaneously providing blood component therapy as and when indicated by the laboratory parameters. Repeated monitoring of arterial blood gases and haematological tests helped guide us in ensuring a good outcome.

References

[1] Hewitt PE, Machin SJ. Massive blood transfusion. In: Contreras M, editor. ABC of transfusion. London: BMJ Publishing; 1992. p. 38–40.

[2] Fakhry SM, Sheldon GF. Massive transfusion in the surgical patient. In: Jeffries LC, Brecher ME, editors. Massive transfusion. Bethesda: American Association of Blood Banks; 1994. p. 17–38.
[3] Stainsby D, MacLennan S, Thomas D, et al. British committee for standards in haematology: writing group, British society for haematology. Br J Haematol 2006;13:634–41.
[4] Hogman CF, Andreen M, Rosen I, et al. Haemotherapy with red-cell concentrates and a new red-cell storage medium. Lancet 1983;1:269–71.
[5] Reiss RF. Hemostatic defects in massive transfusion: rapid diagnosis and management. AJCC 2000;9:158–65.
[6] Hardy JF, De Moerloose P, Samama M. Massive transfusion and coagulopathy: pathophysiology and implications for clinical management. Can J Anaesth 2004;51:293–310.
[7] Committee on Trauma. Advanced trauma life support manual. chicago, American College of Surgeons; 1997. p. 103–12.
[8] Contreras M. Consensus conference on platelet transfusion: final statement. Blood Rev 1998;12:239–40.
[9] Hiippala ST, Myllyla GJ, Vahtera EM. Hemostatic factors and replacement of major blood loss with plasma-poor red cell concentrates. Anesth Analg 1995;81:360–5.
[10] British committee for standards in haematology, blood transfusion task force. Guidelines for the use of platelet transfusions. Br J Haematol, 2003;122(1):10–23.
[11] Ciavarella D, Reed RL, Counts RB, et al. Clotting factor levels and the risk of diffuse microvascular bleeding in the massively transfused patient. Br J Haematol 1997;67:365–8.
[12] Hiippala S. Replacement of massive blood loss. Vox Sang 1998;74:399–407.
[13] Dzik WH, Kirkley SA. Citrate toxicity during massive blood transfusion. Transfus Med Rev 1998;2:76–94.
[14] Stainsby D, Cohen H, Jones H, et al. Serious hazards of transfusion (SHOT) annual report 2004. Manchester, serious hazards of transfusion steering group; 2005. p. 1–51.
[15] Stainsby D, MacLennon S, Hamilton PJ. Management of massive blood loss: a template guideline. Br J Anaesth 2000;85(3):487–91.
[16] Donaldson MDJ, Seaman MJ, Park GR. Massive blood transfusion. Br J Anaesth 1992;69(6):621–30.