Postpartum haemorrhage and haematological management

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Abstract

Postpartum haemorrhage (PPH) is a leading cause of maternal morbidity and mortality worldwide. Recent report of the Centre for Maternal and Child Enquiries (CMACE) in the United Kingdom, which was previously called Confidential Enquiries into Maternal and Child Health (CEMACH), confirmed a reduction in maternal deaths during the last Triennium (2006–2008). This is attributed to improvements in timely diagnosis and prompt and aggressive treatment. PPH is now the sixth most common direct cause of maternal deaths in the U.K. World Health Organization (WHO) estimates that postpartum haemorrhage accounts for 25% of maternal deaths worldwide. Substandard care and 'too little being done too late' remain a significant contributor of maternal deaths.

Primary PPH refers to a blood loss from the genital tract of 500 ml or more within 24 h of delivery (or >1000 ml during caesarean section). Secondary PPH refers to an excessive blood loss between 24 h and 6 weeks after birth. Massive PPH refers to a blood loss of over 2000 ml (or >30% of blood volume) and is associated with increased maternal morbidity and mortality. A timely, multi-disciplinary and systematic approach to restore the volume, clotting system and the oxygen carrying capacity of blood, whilst steps are taken to arrest bleeding, is essential to save life.

Primary postpartum haemorrhage is caused by uterine atony, genital tract trauma, retained placental tissue and membranes after birth or coagulopathy. The latter may not only be a cause of PPH, but also could be an effect of massive haemorrhage due to a 'washout phenomenon'. Rapid and profuse bleeding results in loss of platelets and clotting factors, that get 'washed out'. This may lead to a depletion of coagulation factors and resultant bleeding.

Keywords coagulopathy; communication; massive obstetric haemorrhage; placenta percreta; resuscitation; shock index

Introduction

Postpartum haemorrhage (PPH) is a potential killer around the world. Increasing caesarean section rates, maternal obesity, increasing childbearing age with associated obstetric and medical conditions are likely to cause an increase in the rate of PPH. Royal College of Obstetricians and Gynaecologists (RCOG) guidelines on PPH, refer to a blood loss of more than 1000 ml as PPH. This is further sub-classified into 'moderate PPH' (1000–2000 ml) and 'severe PPH' (>2000 ml). These arbitrary values are important to make a diagnosis. However, clinicians should appreciate that even a smaller blood loss (i.e. <1000 ml) may result in haemodynamic instability in a patient who is anaemic prior to delivery. Similarly, a woman with a low body mass index (BMI) may not be able to withstand even a moderate blood loss (1000–2000 ml) in view of her smaller circulating blood volume.

More importantly, it is well recognized that visual estimation of blood loss by clinicians is notoriously inaccurate. In addition to the visually estimated 'amount' of blood loss, clinicians should also assess the rate of bleeding and associated haemodynamic response of the woman, so as to institute appropriate management. According to the Scottish Confidential Audit into Severe Maternal Morbidity, the incidence of massive obstetric haemorrhage (2500 ml) or women who received more than 5 L of blood is estimated at 3.7/1000 maternities. Emergency caesarean sections were associated with an approximately three fold increase in postpartum haemorrhage as compared to elective caesarean section or spontaneous vaginal births.

Various risk matrices have been proposed to predict the occurrence of massive PPH. However, up to 40% of cases of PPH occur in women are initially classified as 'low risk'. This highlights the need for clinicians to be vigilant and anticipate, recognize and promptly institute timely and appropriate management to reduce morbidity and to avoid mortality due to massive postpartum haemorrhage. The role of multi-disciplinary simulation training, labour ward fire drills and development of local protocols for systematic, multidisciplinary management of massive obstetric haemorrhage (e.g. 'Code Blue') cannot be overemphasized.

Rule of 30 (Table 1) and Shock Index (SI) are useful tools that could be used by clinicians in an emergency to understand the amount of blood loss and the degree of haemodynamic compromise. Shock Index refers to pulse rate divided by systolic blood pressure (PR/SBP) and its normal value is 0.5–0.7. As a woman bleeds, the heart rate increases to compensate for the blood loss, much before any changes in systolic blood pressure are observed. Hence, the shock index (SI) increases. In severe haemorrhage, SI increases to 0.9–11.1 and it has been reported that a shock index of >0.9 was associated with a need for intensive therapy on admission.

Haematological management involves recognition of the amount and rapidity of blood loss and replacing the circulating blood volume and restoring 'coagulability' of blood. Massive postpartum haemorrhage resulting in a 'washout phenomenon' and depletion of coagulation factors is likely once 80% of the blood volume has been lost. This equates to approximately a blood loss of 4.5 L in an 'average size' woman. However, coagulopathy may set in earlier, especially if there is rapid bleeding or if there was an existing predisposing factor such as pre-eclampsia.

Case history

Background

A 34 year old woman was referred to our Regional Referral Service for Morbidly Adherent Placenta at 28 weeks of gestation. She had two previous caesarean sections and was diagnosed to

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'Rule of 30' for massive obstetric haemorrhage

Systolic blood pressure	Falls by 30 mmHg
Pulse	Increases by 30 beats/min
Haemoglobin	Falls by 30% (approx. 3 g/dl)
Haematocrit	Falls by 30%
Estimated blood loss	30% of normal (70 ml/kg in adults) (100 ml/kg during pregnancy)

Table 1

have a major degree placenta praevia at 22 weeks. She presented with unprovoked vaginal bleeding at 28 weeks to her local hospital. Antenatal imaging confirmed major degree placenta praevia, which was antero-lateral with percreta (infiltration into the posterior bladder wall).

Diagnosis was reconfirmed at the tertiary hospital by repeat imaging and the patient was counselled regarding management options. She declined a hysterectomy and hence, a decision was made for conservative surgical treatment with bilateral tubal ligation, as per the request of the patient. In view of a significant blood loss (1 L) at 28 weeks that necessitated a blood transfusion and an episode of bleeding (150 ml) at 33 weeks, a decision was made to deliver at 35 weeks by an elective caesarean section. 4 units of blood were cross-matched daily and the patient was managed as an inpatient until birth. Antenatal corticosteroids were administered on admission. The opinion of our haematology team was sought regarding thromboprophylaxis and a joint multi-disciplinary decision was made not to institute pharmacological thrombo-prophylaxis, in view of her increased risk of bleeding. An individualized local joint multi-disciplinary care plan was made.

Pre-operative care

Patient was transferred to the interventional radiology suite for prophylactic placement of pelvic artery balloon catheters, after insertion of epidural catheters. Balloon occlusion catheters were placed within the anterior division of the internal iliac artery. Pre-filled syringes containing a mixture of saline and contrast medium were attached to the catheter tube for inflation after the delivery of the fetus.

Prior to caesarean section, an image intensifier was used by the attending consultant interventional radiologist to exclude balloon migration. Cell saver and 6 units of cross matched blood were kept ready in the theatre in view of anticipated massive obstetric haemorrhage. Two consultant obstetricians and two consultant anaesthetists were involved with the procedure and the haematology team and the blood bank were alerted. A bed was reserved in the intensive treatment unit. The neonatal team was in attendance.

A peri-operative ultrasound examination was performed on the theatre table to delineate the upper border of the placenta, prior to caesarean section.

Intra-operative management

A supra-pubic transverse incision was made. Perforation of the placenta through the previous uterine scar as well as laterally into the left uterine wall was noted. A lower segment caesarean section was performed and a live baby boy, weighing 2.4 kg was delivered in good condition. Profuse and rapid bleeding was noted from the friable placental tissue that was perforating through the serosa.

Pelvic artery balloon catheters were inflated by the interventional radiologist immediately after the delivery of the baby. In addition to a bolus dose of 5 units of oxytocin, an infusion of 40 units of oxytocin in 500 ml normal saline was commenced at 10 units/h (125 ml/h). In view of the lateral perforation of the placenta (Figure 1), a complete myometrial excision was not performed as there was a risk of injury to ureters. An anterior myometrial excision with attached placenta was carried out (Figure 2). In view of profuse bleeding from the area of infiltration on the lateral uterine wall, placental tissue was removed and multiple compression sutures were placed. Low vertical compression sutures were inserted to control bleeding from the placental site. The placental tissue infiltrating the posterior bladder wall was not separated as there was no bleeding. The myometrial defect was closed in two layers and routine closure of the anterior abdominal wall was carried out. Post-operative uterine artery embolization was carried out once the patient was haemodynamically stable and the patient was managed in the ITU for 24 hours.

Haematological management

In view of profuse and rapid haemorrhage and the location of placental invasion (i.e. lateral uterine wall), the patient lost approximately 4 L of blood within 5 min of uterine incision. Our Major Obstetric Haemorrhage Protocol 'Code Blue' was activated when the blood loss exceeded 2 L.

Resuscitation with intravenous (i.v.) fluids (crystalloids and colloids) was commenced and blood was sent for urgent full blood count (FBC) and serum electrolytes and clotting screen. Immediate blood transfusion was commenced and autologous blood transfusion was also carried out using the blood salvaged through the cell saver. Haemabate (Prostaglandin F2alpha) 250 mcg was administered intramuscularly and two doses were administered to optimize uterine contraction and to minimize bleeding due to a co-existing atonic myometrium.

The following blood products and clotting factors were administered:

- 8 units of cross matched blood
- 8 units of Fresh Frozen Plasma (FFP)

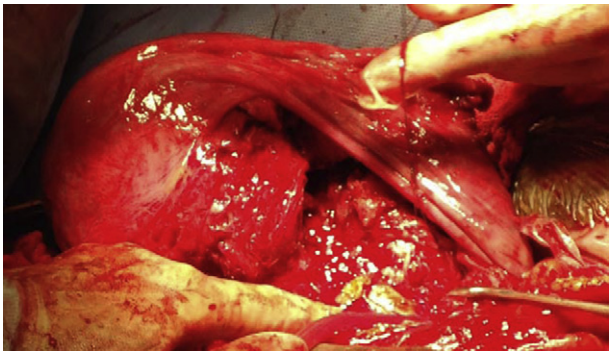

Figure 1 Placenta percreta infiltrating the left uterine wall after delivery of the baby.