**Massive postpartum haemorrhage and management of coagulopathy**

Edwin Chandraraharan

Sabaratnam Arulkumaran

---

**Abstract**

Postpartum haemorrhage (PPH) is a leading cause of maternal morbidity and mortality in both the developed and developing world. By definition, PPH refers to a blood loss of > 500 ml or > 1000 ml during a caesarean section) after the delivery of the fetus. However, this is an arbitrary value as women who are 'small made' (low blood volume) or anaemic may not tolerate even a blood loss of 500 ml. Massive PPH refers to the loss of 30–40% (generally > 2 L) of the patient's blood volume, resulting in changes in the haemodynamic parameters which lead to moderate or severe shock. Consequences of such massive blood loss include sudden and rapid cardiovascular decompensation and coagulopathy, as well as iatrogenic complications of fluid replacement and multiple blood transfusions (pulmonary oedema, transfusion reactions and adult respiratory distress syndrome).

**Keywords** coagulopathy; disseminated intravascular coagulation; hysterectomy; shock index; uterine tamponade

The case discussed below illustrates the occurrence of coagulopathy due to massive postpartum haemorrhage (PPH) and the issues relating to its management.

**Case**

A 28-year-old woman in her fourth pregnancy (body weight 60 kg, body mass index 22) with a previous preterm birth and two subsequent miscarriages (gravida 4, para 1) was admitted at 23 weeks and 1 day with a history of lower abdominal pain. On general examination, she was afebrile with a blood pressure (BP) of 125/62 mmHg and a pulse rate of 82 beats/min. On abdominal examination palpable uterine contractions were noted and on speculum examination, the cervix was dilated 3 cm with intact membranes. Both high and low vaginal swabs were taken. The patient was counselled regarding the very poor prognosis, especially in view of 'pre-viability', and was seen by the neonatal team. A joint plan was made to manage the patient conservatively. She was managed as an inpatient with 4 hourly observations of temperature, pulse and blood pressure as per departmental protocol.

After 5 days of admission, she developed lower abdominal pain and a yellowish vaginal discharge. On examination, her temperature was 37.3°C, pulse rate was 103 beats/min and BP was 121/63 mmHg. Palpable uterine contractions were noted. The cervix was dilated 4 cm on vaginal examination. A diagnosis of preterm labour secondary to probable chorioamnionitis was made and the patient was transferred to the labour ward. She had a normal vaginal birth and the live baby boy was handed over to the neonatal team. The estimated blood loss (EBL) after the delivery was 200 ml. Within an hour of delivery, there was a 'big gush' of vaginal bleeding. This was followed by profuse bleeding due to an atomic uterus that did not respond to uterine massage. Intravenous syntocinon infusion and colloids (Gelofusin) were commenced and an urgent request was sent for cross matching for 4 U of blood and for full blood count (FBC) and coagulation profile.

Within 15 min a very rapid deterioration of the patient's clinical condition was noted. Her BP was 91/57 mmHg and pulse rate was 156 beats/min. A 'Code Blue' (local protocol for massive obstetric haemorrhage) was activated to involve the multidisciplinary team (obstetric consultant, second anaesthetist, haematologist, porters, blood bank and to alert the Intensive Treatment Unit (ITU). The patient continued to bleed profusely despite uterine massage, additional oxytocics and three doses of haemabate (PGF2α). She was transferred to the theatre and an examination under anaesthesia (EUA) was performed. An attempt was made to insert a uterine balloon to arrest haemorrhage by tamponade after excluding traumatic causes and retained tissues. However, bleeding continued and the EBL at this stage was 7 L; therefore, a decision was made to proceed with an emergency laparotomy to control bleeding.

Due to the deteriorating clinical condition of the patient and continuous profuse bleeding, it was decided to proceed to a subtotal hysterectomy as a life saving measure. Haematologist and blood transfusion services helped to determine the degree of depletion of blood and clotting factors and provided appropriate quantities of these for replacement. The patient was transferred to ITU care after surgery. She had 14 U of blood and 6 U of fresh frozen plasma (FFP) and platelets. If there was no response to the use of clotting factors, use of recombinant activated factor VIIa was contemplated. She made a good recovery within 72 h and was transferred to the postnatal ward. She was discharged home in good health 7 days after delivery.

This clinical problem illustrates the possibility of a massive PPH, even in earlier gestations. The main driving force for preterm labour is often infection and this patient showed clinical signs and symptoms of chorioamnionitis 5 days after admission. Her labour progressed well and she had a spontaneous vaginal delivery followed by a massive PPH. This could be due to the direct effect of chorioamnionitis on the uterine myometrium; decreasing the contractility and thereby the ability of the myometrium to act as 'living ligatures' after delivery. Infection also can trigger disseminated intravascular coagulation (DIC), and these two effects could be additive. This may explain the rapid deterioration of her clinical condition and the unresponsiveness of...
the uterus to the uterotonie agents. Bimanual compression, syntocinon infusion and prostaglandins (Haemabate) failed to reduce or arrest bleeding.

Young, healthy and fit adults can withstand and compensate for considerable blood loss without demonstrating any cardiovascular changes for lengthy periods. This patient maintained her pulse rate and blood pressure despite the loss of 2–3 L of blood. By the time changes in cardiovascular parameters were seen (BP - 94/25 mmHg, pulse - 126 beats/min), she is likely to have lost at least 30% of her blood volume (estimated blood volume based on her body weight is 60 x 80 = 4800 ml). For practical management we propose a ‘rule of 30’ or a change in the ‘shock index’ to be valuable in monitoring women with haemorrhage. If the systolic blood pressure falls by 30 mmHg, pulse rate rises by 30 beats/min, respiratory rate increases to >30 breaths/min and haemoglobin (Hb) or haematocrit drop by 30%, then the patient is most likely to have lost at least 30% of her blood volume and is in moderate shock leading to severe shock. Shock index (SI) refers to pulse rate divided by the systolic blood pressure. The normal value is 0.5–0.7, but with significant haemorrhage the SI increases to 0.9–11.1. Rady et al. reported that an SI >0.9 was associated with the need for intensive therapy on admission. In this patient, the SI was 0.656 prior to haemorrhage and was 1.71 when she was taken to theatre. It returned to 0.678 when adequate perfusion and arrest of haemorrhage was achieved. The value of SI in a population may be influenced by various factors but the change of SI in the same individual may provide useful information.

The morbidity and mortality in this group of patients with massive PPH are also very high. Massive obstetric haemorrhage should be managed swiftly and appropriately with a multi-disciplinary input. It is prudent to have local protocols (like ‘Code Blue’), which can be activated to alert all the relevant senior staff/departments whose involvement is vital. Group specific Rhesus negative or ‘O negative’ blood should be transfused until cross-matched group specific blood is available.

This patient was transferred to theatre after conservative measures failed. As a general rule, if there are haemodynamic changes and/or bleeding persists after three doses of Haemabate and all conservative measures (uterine massage, bimanual compression, syntometrine, syntocinon infusion) have been instituted, transfer to theatre should be considered before the situation deteriorates too far.

**Surgical management of postpartum haemorrhage**

Examination under anaesthesia (EUA) should be carried out to exclude traumatic cause of bleeding and retained products. Bimanual compression could be tried again at this juncture. ‘Uterine tamponade’ using a balloon could be attempted at this stage. The available balloons include the gastric balloon of the Sengstaken-Blackmore tube or a Rüschi balloon. The balloon could be inflated with 200–600 ml of warm sterile water or saline, depending on the size of the uterine cavity, to arrest bleeding. The ‘tamponade test’ rapidly identifies women with massive PPH who require a laparotomy. If the ‘tamponade test’ is positive (i.e. bleeding is arrested immediately with uterine tamponade), as is the case in nearly 80–85% of case series, patients do not require a laparotomy. A smaller uterine cavity in earlier gestations may not accommodate these standard balloons and a Foley’s catheter balloon can be tried instead. Uterine tamponade works by exerting a counter pressure on the placental bed, arresting bleeding. This often helps to correct coagulation abnormalities by providing a window of opportunity to replace platelets and clotting factors. Unfortunately, attempted uterine tamponade failed in this patient, which necessitated a laparotomy.

The logical next step is laparotomy to insert uterine compression sutures (B-Lynch or one of the various modifications). Systematic pelvic devascularisation (uterine artery ligation, ‘quadruple ligation’, including both tubal branches of the ovarian artery and internal iliac artery ligation), uterine artery embolisation and total or subtotal hysterectomy are reserved as subsequent step-wise procedures. At the time of laparotomy, this patient had already lost 7 L of blood and was haemodynamically very unstable. Attempting conservative surgical procedures at this stage would have further delayed action that is rapidly required to arrest bleeding. If the patient was haemodynamically stable, uterine compression sutures could have been attempted. These may have helped to reduce the bleeding from the placental site, allowing time to correct the coagulation abnormalities. Uterine artery ligation does not require any special surgical skills; however, internal iliac artery ligation may require the assistance of a vascular surgeon or a gynaecology-oncologist, who is familiar with the anatomy of the lateral pelvic wall so as to avoid unintentional damage to ureters or iliac vessels. Internal artery ligation may reduce the pulse pressure by up to 80%, reducing the bleeding from the raw placental surface and providing additional time to replace blood and clotting factors to correct the DIC. As mentioned earlier, the rapidity and severity of deterioration in clinical and haemodynamic parameters in this patient made the institution of the above relatively conservative surgical measures unsuitable. Uterine artery embolisation requires the availability of an interventional radiologist and equipment for the placement of an embolisation catheter, and hence it was not an ideal option in this case or in any such emergency situation.

The decision to perform a hysterectomy should be made at the consultant level, in good faith to save life when all other conservative measures have failed or are inappropriate for the clinical situation. This decision needs to be made earlier than later to avoid maternal morbidity and mortality. The patient, or if she is under general anaesthesia, her relatives should be informed prior to the procedure, if possible. Although, this is unlikely to have any significance from a medico-legal point of view, it is good medical practice to keep the patient’s partner or close family members informed. If the bleeding is from a low lying placenta, then a total abdominal hysterectomy may be warranted to arrest bleeding from the placental site. If the placenta is fundal, a subtotal hysterectomy is a simpler and quicker operation.

This patient had a subtotal hysterectomy as the bleeding was due to an atomic uterus with superimposed coagulopathy, the placental site being fundal. Performing a hysterectomy in the presence of coagulopathy may be hazardous as it may cause bleeding and oozing from small vessels in incised tissues. This bleeding is minimal as compared to that from the uterus (70 ml/min). It is essential to rapidly control bleeding so as to prevent further decompenensation and therefore a decision was
made to proceed with a hysterectomy. The difficulty with haemostasis due to coagulopathy is controlled by infusion of clotting factors and if necessary by packing the pelvic cavity at the time of the operation. The pack can be removed after 24–48 h when the patient is stable and coagulopathy has been controlled. This patient had peritoneal and sub-rectus drains to assess the degree of postoperative intra-abdominal bleeding.

The history of this patient illustrates how young, fit and healthy adults can withstand massive blood loss until they reach a ‘critical point’, when they rapidly deteriorate.

**Management of coagulopathy**

Coagulopathy could be due to several factors: DIC, utilisation of clotting factors with the blood clots that are lost to the exterior, dilution of existing clotting factors with replacement of crystalloids, lack of clotting factors in stored blood, hypothermia, and acidosis caused by hypoxia, which hinders blood clotting.

In this case, DIC may have been caused by sepsis. The latter could have triggered a damage to endothelial cells and also release of inflammatory mediators that may have activated the extrinsic arm of the coagulation cascade. Massive blood loss and resultant multiple blood transfusions may also trigger DIC. The breakdown products of fibrin may inhibit clotting, but predominantly the lack of clotting factors due to excess bleeding with clots (‘washout phenomenon’) is the predominant component. Management of this condition involves rapid replacement of fresh blood, FFP and platelets, and correction of coagulopathy and any underlying cause.

The patient was given broad spectrum intravenous antibiotics to treat sepsis after blood was sent for culture and antibiotic sensitivity test. The atonic uterus, causing continuous profuse haemorrhage (usually about 70 ml/min), made volume and clotting factor replacement very difficult, and hence the patient had to undergo an emergency subtotal hysterectomy. The difficulty with clotting factor replacement very difficult, and hence the patient had to undergo an emergency subtotal hysterectomy to control bleeding. This procedure helped to stop further bleeding and enabled rapid resuscitation by replacement of blood volume. Fibrin degradation products (FDP), which are formed in DIC, have been shown to be toxic to the smooth muscles of the myometrium and myocardium, suppressing their contractility and activity, and this may have contributed to the atonic PPH.

**Postoperative care**

Management of PPH does not end with the control of haemorrhage and replacement of blood volume and clotting factors. It is essential that these patients are cared for in a high dependency or intensive care setting to recognise early and treat any further complications. These include surgical (intraperitoneal bleeding, haematoma formation, wound complications), iatrogenic (pulmonary oedema due to fluid overload, transfusion reactions and adult respiratory distress syndrome [ARDS] due to multiple blood transfusions) and multi-organ failure secondary to hypovolaemic ischaemic injury. It is also very important to ‘de-brief’ the patient and her partner postoperatively and to explain the events leading to her hysterectomy. Some patients develop postnatal depression due to losing their uterus and the loss of future fertility may require counselling and support.

**Algorithm for the management of postpartum haemorrhage (‘HAEMOSTASIS’)**

- H – call for Help
- A – Assess (haemodynamic status and blood loss) and resuscitate
- E – Establish Etiology, Ecblolics (syntocinon/syntometrine/ergometrine bolus), Ensure availability of blood
- M – Massage the uterus
- O – Oxycotics (syntocinon infusion/prostaglandins i.m., per rectum, intra-myometrial)
- S – Shift to theatre, EUA, exclude trauma/retained products, bimanual compression
- T – Tamponade (balloon or uterine packing)
- A – Apply compression sutures (B-Lynch/modified)
- S – Systemic pelvic devascularisation
- I – Interventional radiology (uterine artery embolisation)
- S – Subtotal or total abdominal hysterectomy

**Table 1**

| A | Assess (haemodynamic status and blood loss) and resuscitate |
| E | Establish Etiology, Ecblolics (syntocinon/syntometrine/ergometrine bolus), Ensure availability of blood |
| M | Massage the uterus |
| O | Oxycotics (syntocinon infusion/prostaglandins i.m., per rectum, intra-myometrial) |
| S | Shift to theatre, EUA, exclude trauma/retained products, bimanual compression |
| T | Tamponade (balloon or uterine packing) |
| A | Apply compression sutures (B-Lynch/modified) |
| S | Systemic pelvic devascularisation |
| I | Interventional radiology (uterine artery embolisation) |
| S | Subtotal or total abdominal hysterectomy |

**Conclusion**

In the case discussed atonic uterus led to massive PPH. The loss of clotting factors with extensive blood loss, some element of DIC, dilutional effect, hypoxia, metabolic acidosis and hypothermia may have contributed to coagulopathy and continued blood loss. The rapid clinical deterioration of the patient necessitated a subtotal hysterectomy. Awareness of the occurrence of massive bleeding is needed, even in early gestation, as well as the detrimental effect of sepsis both on the coagulation system and the myometrium affecting uterine tone. Young healthy women maintain their cardiovascular parameters by effectively compensating for their blood loss until very late in the process, when they can suddenly and rapidly deteriorate. Activation of a ‘Massive Obstetric Haemorrhage Protocol’, effective fluid and blood volume replacement, regular emergency ‘PPH drills’ in the labour ward, involvement of senior input very early in the process and a multi-disciplinary approach may reduce mortality and morbidity due to massive PPH complicated by coagulopathy. Such measures reduce the maternal deaths due to the phenomenon of ‘too little too late’ quoted in several confidential enquiries into maternal deaths. A management algorithm for PPH is given in Table 1.

**FURTHER READING**


Practice points

- Massive postpartum haemorrhage is associated with increased maternal morbidity and mortality even in earlier gestations.
- Young, fit and healthy adults may maintain their cardiovascular parameters despite of significant blood loss for sometime and may suddenly decompensate.
- ‘Rule of 30’ and ‘Shock Index’ may help in determining the degree of blood loss.
- Multi-disciplinary approach and replacement of blood and blood products and timely and appropriate institution of surgical measures are likely to save lives.