Massive haemorrhage in pregnancy

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Massive haemorrhage is a major cause of maternal mortality. Life-threatening haemorrhage may occur as frequently as 6.7 per 1000 deliveries.[1] This equates to 1400 cases yr^{-1} in the UK or 33.5 yr^{-1} in an obstetric unit with 5000 deliveries annually. Pregnancy-related conditions and complications account for 0.8% of intensive care admissions; 35% of these arise from massive haemorrhage.[1][2]

Management of massive obstetric haemorrhage is often substandard. Many factors contribute to this. Blood loss can be underestimated because bleeding may be concealed and the presence of amniotic fluid makes accurate measurement difficult. The physiological changes of pregnancy mask the magnitude of the blood loss. Poor communication and failure to call for senior assistance are also factors.[1]

Definitions

Massive obstetric haemorrhage is variably defined as: blood loss >1500 ml; a decrease in haemoglobin >4 g dl^{-1}; or acute transfusion requirement >4 units.[3]

Obstetric haemorrhage is classified as antepartum (APH); bleeding occurring after 24 weeks gestation and before delivery, or postpartum (PPH). Postpartum haemorrhage can be primary (within 24 h of delivery) or secondary (24 h to six weeks after delivery).

Physiology

In some respects, pregnancy induced cardiovascular changes protect against the effects of haemorrhage. By term, cardiac output has increased by 50%, stroke volume by 25% and blood volume from 70 ml kg^{-1} to almost 100 ml kg^{-1}.[4] Tachycardia may be the only sign of haemorrhage until 30–40% of the circulating volume has been lost, when hypotension and peripheral vasoconstriction develop. Aortocaval compression will compound the haemodynamic instability caused by haemorrhage.

The uterine and ovarian arteries supply the uteroplacental unit. Uterine blood flow increases from <5% to 12% of cardiac output during pregnancy (700–900 ml min^{-1}).[4] Hence, blood loss from the uteroplacental bed can be brisk and difficult to control. In APH, cardiotocographic signs of fetal distress, attributable to uterine hypoperfusion, can precede maternal evidence of haemodynamic compromise, particularly in the case of abruption.

Causes of haemorrhage

The causes of obstetric haemorrhage are outlined in Table 1.

Antepartum haemorrhage

Placenta praevia and abruption are major causes of significant haemorrhage in the third trimester. In placenta praevia, the placenta implants in the lower segment of the uterus over, or very near, the internal os. It is usually diagnosed during routine antenatal ultrasound screening and is classically associated with painless vaginal bleeding.[3] Placenta accreta, increta and percreta are conditions of abnormal placentation, in which there is an increasing degree of abnormal invasion of the placenta into the myometrium. They are usually associated with placenta praevia and with scarring

Table 1 Causes of haemorrhage related to pregnancy

Early pregnancy
Incomplete abortion
Septic abortion
Ruptured ectopic

Antepartum haemorrhage
Placenta praevia
Placental abruption
Uterine rupture
Trauma

Primary postpartum haemorrhage
Uterine atony
Retained products of conception
Genital tract trauma
Abnormally adherent placenta
Clotting defects
Acute uterine inversion

Secondary postpartum haemorrhage
Puerperal sepsis
Retained products of conception

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of the lower segment of the uterus. Because of the increasing Caesarean section rate, the incidence of abnormal placentation is on the increase.

Placental abruption refers to the premature separation of a normally implanted placenta with bleeding that can be either concealed or per vagina. Risk factors include maternal hypertension, uterine over-distension, previous abruption, abdominal trauma, smoking and cocaine use.[3] There is often associated increased uterine tone, abdominal pain and premature labour. Fetal distress is common and can be the presenting feature.

Uterine rupture is uncommon but potentially fatal to both mother and baby. The risk is increased by previous uterine incision but it can also occur in an unscarred uterus. Rupture may be painless, with the early signs being limited to slow progress in labour and a deterioration in the CTG. A high degree of suspicion is required in labour after previous Caesarean section. Haemorrhage can be torrential.

Less common causes of massive obstetric haemorrhage include splenic and renal artery aneurysm rupture, which are more common during pregnancy, particularly in the third trimester. They have a high maternal and fetal mortality rate.

Postpartum haemorrhage

Uterine atony accounts for 80% of primary PPH and follows 1 in 20 deliveries. Risk factors include; prolonged, augmented or precipitant labour; uterine overdistension and abnormalities; placenta praevia; increasing parity; and advanced maternal age.[3]

Trauma to the perineum, cervix and vagina are common after vaginal delivery, the risk being increased by forceps delivery or vacuum extraction. Genital tract trauma must be considered where there is ongoing bleeding with a well contracted uterus. Retained placental fragments (often associated with uterine atony) are another common cause of both primary and secondary PPH.

Uterine inversion is rare but serious. Cardiovascular instability, attributable to autonomic stimulation, may be out of proportion to actual blood loss, although this may be great. Early reduction of the uterus is vital and this may require uterine relaxation; nitrates, β_2-agonists, volatile agents and magnesium have all been used.

Coagulopathies

Coagulopathies, both congenital and acquired, contribute to massive obstetric haemorrhage. There are many causes of disseminated intravascular coagulopathy (DIC) associated with pregnancy (Table 2).

Management of haemorrhage

General

Initial management of massive obstetric haemorrhage is the same regardless of the cause. Immediate management is aimed

Table 2 Causes of DIC related to obstetrics

Intrauterine death (>2 weeks previously)
Amniotic fluid embolus
Sepsis
Pre-eclampsia
Placental abruption
Retained products of conception
Induced abortion
Excessive bleeding
Acute fatty liver

Table 3 Management plan for massive obstetric haemorrhage

Two large-bore i.v. cannulae
Commence rapid saline infusion
Full left lateral if APH – head down, feet up
Oxygen 100% via facemask
Send blood for:
    Crossmatch 6 units
    Full blood count
    Coagulation screen
Call for senior help
Alert haematologist, mobilize portering service
Transfuse red cells as soon as possible
Until then:
    Crystalloid, maximum 2 litre
    Colloid, maximum 1.5 litre
    Use group specific or O RH negative blood
    Warm resuscitation fluids
    Restore normovolaemia, monitor haematocrit and haemoglobin
    If massive bleeding continues, give FFP 1 litre, cryoprecipitate
        10 units while awaiting lab results
    Use coagulation screens to guide and monitor use of blood products
    Monitor pulse rate, blood pressure, blood gases and urine output
    Consider invasive monitoring to guide therapy

at maternal resuscitation (Table 3). However, diagnosis and definitive treatment should also be initiated during the primary resuscitation.

Specific treatments

Physical
If the uterus is atonic, stimulation of uterine activity by 'rubbing up' a contraction, using vigorous bimanual massage, may be effective.

Pharmacological
Oxytocin, the first line agent for treating uterine atony, causes short-lived uterine contraction. It is administered as a slow bolus injection of 5 units followed by a continuous infusion of 5–40 units in saline 0.9%, 500 ml at a rate that abolishes uterine atony. Bolus injections of oxytocin can precipitate hypotension and tachycardia and should be used with caution, particularly if the patient is unstable.

Ergometrine, an ergot alkaloid, causes uterine and vascular smooth muscle contraction. It is administered as an intravenous or intramuscular injection of 250–500 μg. It can cause hypertension and should be avoided in pre-eclamptic patients. Nausea and vomiting are very common.

15-Methyl prostaglandin $F_{2-\alpha}$ (Carboprost) is used for bleeding that is unresponsive to oxytocin and ergometrine. It is administered as a deep intramuscular or intramyometrial injection of 250 µg repeated at intervals of 15–30 min, the total dose not exceeding 2 mg. Bronchospasm, flushing, nausea and vomiting can occur; asthma is a contraindication. Increased pulmonary shunting may cause maternal hypoxia.

Surgical

Surgical intervention may be required to control obstetric haemorrhage. The cause of the bleeding, experience and expertise of the operator and the facilities available will dictate the intervention chosen. Surgical options include: manual removal of placenta; uterine packing; uterine and hypogastric artery ligation; B-Lynch suture to the uterus; and hysterectomy.[3] B-Lynch suture is a new technique used in patients who have responded well to bimanual uterine compression. It utilises a continuous suture through the uterus to provide compression.

Optimal timing of hysterectomy is difficult. With good supportive management, it is possible to maintain haemodynamic stability and coagulation function in the face of ongoing blood loss. A hysterectomy should be performed, despite concerns for future fertility, if haemodynamic stability cannot be achieved or when other methods have failed to arrest the haemorrhage. It should not be delayed until supportive measures are failing.[3]

Radiological

Selective embolization of the pelvic vessels, using interventional radiological techniques, has been used to control haemorrhage and avoid the need for hysterectomy.[5] Not all centres have access to staff and facilities and there may be logistical problems transferring an unstable patient to an angiography suite.

Blood and blood products

A standing order, or agreement, should exist with blood bank to ensure rapid delivery of blood products once the massive obstetric haemorrhage protocol is initiated. Transfusion of packed red cells should occur early and group-specific blood used until crossmatched blood is available. All obstetric units should have immediate access to at least two units of O-Rhesus negative blood, and more should be available in units without on-site transfusion services. Coagulation screens help assess the adequacy of treatment and guide blood product use but should not delay the initial issue of fresh frozen plasma (FFP) 1 litre and cryoprecipitate 10 units.

Recombinant, activated Factor VII (rFVIIa, NovoSeven, Novo Nordisk, Denmark), appears to be highly effective in cases of refractory haemorrhage at doses of up to 100 µg kg^{-1} i.v. (total dose 7.2–19 mg). At present, it is not widely available and its expense limits its use to: patients unresponsive to conventional treatment; where embolization is unavailable; or hysterectomy is the only alternative. It is not licensed for use in obstetrics and there are concerns about the risks of thrombotic complications.

Anaesthesia for obstetric haemorrhage

The anaesthetic management plan will be determined by both maternal and fetal considerations. When haemodynamic stability is lost, general anaesthesia is usually indicated. Etomidate or ketamine may be preferable to thiopental or propofol in the presence of severe hypovolaemia. Early use of invasive monitoring is advisable but should not delay initial fluid resuscitation. Because of the risk of coagulopathy, peripheral long lines or internal jugular central access should be used in preference to the subclavian approach.

Abruption raises particular issues. In order to avoid surgery in a woman who may be hypovolaemic and coagulopathic, vaginal delivery is recommended when there is fetal demise. However, acute fetal distress will require a Caesarean section. Owing to the risk of DIC, especially if fetal compromise is associated with maternal haemodynamic changes, general anaesthesia is necessary. In the absence of fetal compromise and maternal instability, an expectant strategy can be adopted. This may include tocolysis and dexamethasone administration to promote fetal lung maturation.

Not all massive haemorrhage is unexpected. Some women will present for elective Caesarean section where there is 'anticipated' massive blood loss; for example, where abnormal placentation has been diagnosed on ultrasound. High risk cases should be identified early and senior obstetricians and anaesthetists should be involved.[1]

Elective management allows a broader range of anaesthetic options. The Royal College of Obstetrics and Gynaecology (RCOG) state that the choice of anaesthetic should be the decision of the anaesthetist. In all cases, adequate intravenous access must be secured before surgery (i.e. two large-bore cannulae) and invasive monitoring used if likely to be required. Traditionally, patients with placenta praevia have received a general anaesthetic because of concerns about sympathetic blockade caused by regional anaesthesia with hypovolaemia and the potential for prolonged operating time. However, regional techniques are increasingly being used but the patient must be warned that haemorrhage may necessitate conversion to general anaesthesia. Severe degrees of abnormal placental implantation (percreta, acreta) usually warrant general anaesthesia. If a regional technique is used, a combined spinal–epidural has advantages over a single shot spinal if a prolonged operative time is anticipated.

Autologous transfusion

Intraoperative cell salvage has been adopted in the general theatre setting owing to concerns about the potential risks of homologous transfusion and the increasing price and decreasing availability of donor blood. The potential risks of 'amniotic fluid embolism', contamination with fetal debris and iso-immunization of a rhesus negative mother by fetal red cells have not been realized and there is increasing evidence that cell salvage in obstetrics is safe.[6] Separate suction should be used for amniotic fluid to reduce

contamination risk and an appropriate leucocyte depletion filter used to administer salvaged blood.

Antepartum donation and acute normovolaemic haemodilution are not commonly utilised because of concerns about fetal wellbeing and the difficulty in identifying those who will have significant blood loss.

Protocols and fire drills

Successful management of massive obstetric haemorrhage, in both the emergency and elective situation, requires team work. Obstetric units should have a regularly reviewed major obstetric haemorrhage protocol in place. Early involvement of senior obstetricians, anaesthetists, haematologists and additional staff, including a dedicated porter, is vital. Individual roles should be clearly defined. Regular departmental 'fire drills' are recommended.[1] They help ensure familiarity with protocols and identify deficiencies within systems while engendering a team approach to obstetric emergencies. Courses such as 'Managing Obstetric Emergencies and Trauma' (MOET) and 'Advanced Life Support in Obstetrics' (ALSO) are useful.

Problems in early pregnancy

Significant haemorrhage may occur early in pregnancy. Ectopic pregnancy is associated with a mortality of 0.5 per 1000 cases. Massive maternal haemorrhage is the main cause of death. Diagnosis may be delayed and blood loss concealed. Clinical signs do not correlate well with the severity of blood loss.

RCOG guidelines recommend laparoscopic surgery for ectopic pregnancy only in the absence of significant haemodynamic compromise and provided that the surgeon is of sufficient experience. Cell salvage has been used successfully in the management of ectopic pregnancy in the presence of significant haemoperitoneum. Cervical pregnancy, a rare and life-threatening form of ectopic pregnancy, is associated with torrential haemorrhage. Hysterectomy may be required to control the bleeding.

Spontaneous or induced abortion account for 5% of all pregnancy related deaths, 20% of which are caused by haemorrhage. The clinical presentation is variable but there may be marked haemodynamic instability caused by haemorrhage, sepsis or both. Surgical management involves evacuation of retained products of conception. The choice of anaesthetic will be dependent on the patient's condition; etomidate or ketamine is often used in unstable patients.

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Web resources

www.transfusionguidelines.org.uk
www.transfusionguidelines.org.uk/index.asp?Publication=HTM&Section=9&pageid=118

See multiple choice questions 143–146