Critical care management of obstetric emergencies

Obstetric cases are normally well managed on the labour suite. However, emergencies are not uncommon, and when they occur, they can be major. It is vital that rapid assessment and management are put in place. Critical care doctors are often called upon to do so.

The Intensive Care National Audit and Research Centre (2009) report demonstrated that obstetric admissions to the adult intensive care unit are becoming increasingly more common. This article considers the management of some of the more complex cases.

Ovarian hyperstimulation syndrome

Epidemiology

This potentially fatal complication of ovarian stimulation rarely occurs spontaneously and is commonly caused by exogenous administration of gonadotrophins during assisted reproduction. Around 33% of in-vitro fertilization patients will suffer a mild form, while 3–8% will suffer a severe form (Delvigne and Rozenberg, 2002). The World Health Organization estimated a worldwide incidence of severe ovarian hyperstimulation syndrome at 0.2–1% of all assisted conception cycles and a UK mortality of approximately 1:30 000 in-vitro fertilization cycles (Balen, 2008).

Factors increasing the risk of ovarian hyperstimulation syndrome include presence of polycystic ovaries, age <35 years, use of gonadotrophin-releasing hormone agonists, development of multiple follicles during treatment, previous history of ovarian hyperstimulation syndrome, exposure to luteinizing hormone or human chorionic gonadotrophin (Balen, 2008).

Pathophysiology

The critical care team will most likely become involved with patients suffering from severe or critical ovarian hyperstimulation syndrome. Ovarian hyperstimulation syndrome results in abnormal fluid shifts. Evidence suggests that increased plasma levels of various vasoactive cytokines (vascular endothelial growth factor, interleukins or tumour necrosis factor-α) lead to increased vascular permeability and capillary leakage of protein-rich exudates (Alper et al, 2009). Ultimately, fluid shifts from the intravascular compartment to the third space, leading to a hypovolaemic, haemoconcentrated, thrombophilic circulation.

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Clinical features

Severity of ovarian hyperstimulation syndrome and corresponding symptoms are shown in *Table 1*.

Laboratory findings

Full blood count will show rising haematocrit, leucocytosis and thrombocytosis because a hypercoagulable state results. Clotting will show general derangement. Excessive tubular leakage of Na+, resultant retention of K+ along with fluid shifts and microthrombi causing acute renal failure will lead to hyponatraemia, hyperkalaemia and deranged liver function on urea and electrolytes and liver function tests. Arterial blood gases show metabolic acidosis as a result of K+ and H+ being retained secondary to Na+ loss from renal tubules.

Table 1. Classification of ovarian hyperstimulation syndrome

Classification	Clinical features
Mild	Abdominal bloating
	Mild abdominal pain
	Ovarian size <8 cm
Moderate	Moderate abdominal pain
	Nausea and vomiting
	Ultrasound evidence of ascites
	Ovarian size 8–12 cm
Severe	Clinical ascites, hydrothorax or pericardial effusion
	Cerebral oedema secondary to hyponatraemia
	Oliguria
	Diarrhoea
	Haemoconcentration (haematocrit >45%)
	Hypoproteinaemia
	Ovarian size >12 cm
Critical (severe + any of the following)	Haematocrit >60%
	Acute renal failure
	Thromboembolism
	Acute respiratory distress syndrome

Adapted from Jenkins et al (2006)

Differential diagnoses
These can include ovarian cyst (torsion, haemorrhage), pelvic infection, intra-abdominal haemorrhage, ectopic pregnancy or appendicitis.

Critical care management
Care on either the high dependency unit or the intensive care unit is needed. Severe or critical ovarian hyperstimulation syndrome requires one-to-one intensive nursing care and supportive therapy until the condition resolves spontaneously with a fall in serum human chorionic gonadotrophin levels (Budev et al, 2005).

Symptom relief
Airway
Intubation and ventilation may be required in the worst cases to permit treatment of severe or critical symptoms.

Breathing
Paracenteses and/or insertion of chest drains are often required to relieve the symptoms and clinical consequences of large volume ascites and pleural effusions.

Circulation and fluid management
Central venous pressure and an arterial line will be needed in order to guide fluid management. Care should be taken to avoid large volume fluid resuscitation in the face of increased vascular permeability and reduced oncotic pressure. Haemofiltration may be required in patients with persistent oliguria or evidence of acute renal failure.

Thromboses occur in 0.7–10% of cases (Jenkins et al, 2006), with preponderance for upper body sites and frequent involvement of the arterial system. Low molecular weight heparin and compression stockings are strongly recommended for thromboprophylaxis.

Amniotic fluid embolism
Epidemiology
Amniotic fluid embolism is a rare but potentially life-threatening obstetric emergency, resulting from embolism of amniotic fluid into the maternal circulation and causing sudden cardiovascular collapse. It has an estimated UK incidence of 1.8 cases per 100 000 maternities with a case fatality rate of 24% (Knight et al, 2008). In the most recent Confidential Enquiry into Maternal and Child Health report it was named as the second leading cause of direct maternal death (Lewis, 2007). Women who survive the initial insult are likely to suffer from long-term sequelae, in particular neurological complications (Bourjeily and Miller, 2009).

Several factors increase the risk of amniotic fluid embolism: maternal age of 35 years or older, caesarean delivery, forceps-assisted and vacuum-assisted vaginal deliveries, placenta praevia or placental abruption, eclampsia, polyhydramnios, cervical laceration or uterine rupture (Conde-Agudelo and Romero, 2009).

Pathophysiology
It is theorized that amniotic fluid enters the maternal systemic circulation down a pressure gradient via the endocervical veins, placental attachment sites or sites of uterine trauma (Moore and Baldisseri, 2005). The rapid cardiovascular collapse was assumed to be the result of pulmonary capillary obstruction by amniotic fluid emboli. However, the latest evidence points more towards a massive immuno-humoral or anaphylaxis-type response to the presence of fetal debris within the maternal circulation, mediated by histamine, bradykinin and other arachidonic acid metabolites (Conde-Agudelo and Romero, 2009). The overall result is intense, widespread vasospasm and vessel occlusion.

Clinical features
Amniotic fluid embolism typically presents during labour or immediately post-partum in a biphasic manner (Moore and Baldisseri, 2005). The early phase lasts 15–30 minutes and is often undetectable. This results in right heart failure with consequent acute cor pulmonale, pulmonary hypertension and/or systemic hypotension. The late phase results in acute left heart failure, pulmonary oedema, severe haemodynamic instability or cardiogenic shock. The principal features of amniotic fluid embolism are:

Hypoxia
This is often profound and associated with cyanosis and respiratory arrest. Initially thought to be the result of ventilation–perfusion mismatch, ongoing hypoxia is secondary to both cardiogenic and non-cardiogenic pulmonary oedema.

Hypotension and shock
This may be cardiogenic, obstructive and distributive with a 'septic shock-like' process later in the disease.

Neurological impairment
Encephalopathy secondary to hypoxia may be the only presenting feature and is associated with seizure activity in ~50% of cases. Hypoxic brain injury can lead to permanent neurological sequelae in patients who survive the initial insult.

Disseminated intravascular coagulation
This typically presents as massive haemorrhage which can lead to haemorrhagic shock with further cardiovascular collapse.

Laboratory findings
The diagnosis remains clinical and one of exclusion. There are currently no specific laboratory investigations that confirm or deny the diagnosis, although experiments have been done using immunohistochemical staining with the monoclonal antibody TKH-2 which reacts with meconium and mucin from amniotic fluid to stain maternal lung tissue (Benson et al, 2001). Other possible

markers include measurement of zinc coproporphyrin and serum tryptase (Conde-Agudelo and Romero, 2009).

Differential diagnoses

The differential diagnoses of amniotic fluid embolism include pulmonary embolism, air embolism, sepsis, massive obstetric haemorrhage, peripartum cardiomyopathy or eclampsia.

Critical care management

Intensive care unit care is needed as the management of amniotic fluid embolism predominantly involves acute life support.

Airway and breathing

Intubation and ventilation may be required as 100% oxygen is needed in order to prevent further cellular hypoxia.

Circulation and fluid management

Fluid resuscitation is often aggressive, to counteract the profound cardiovascular collapse. Vasopressor and inotropic therapy may be needed because of the continued phase 2 response. Invasive monitoring and echocardiography will guide therapy. Haemorrhage and disseminated intravascular coagulation should be managed with red cell transfusions and clotting products as indicated in close consultation with haematologists. Consideration should be given to use of recombinant factor VII as required. Uterine atony is common and may lead to profound haemorrhage; this should be treated with medical and/or surgical intervention as necessary.

Delivery of the fetus

Immediate delivery of the fetus is essential to prevent further hypoxic damage and facilitate maternal resuscitation. In the event of maternal cardiac arrest, cardiopulmonary resuscitation should follow the standard advanced life support algorithm with the addition of left uterine displacement in order to avoid aortocaval compression. Perimortem caesarean section should be performed within 5 minutes of the onset of cardiopulmonary resuscitation (Howell et al, 2007).

Potential therapies

A number of potential treatments for amniotic fluid embolism have been described. These include plasma exchange transfusion, inhaled nitric oxide or prostacyclin, extracorporeal membrane oxygenation and an intra-aortic balloon pump (Conde-Agudelo and Romero, 2009).

Pre-eclampsia and eclampsia

Epidemiology

Pre-eclamptic toxaemia is a multisystem disorder that complicates 3–5% of all pregnancies (Gogarten, 2009) and is defined as pregnancy-induced hypertension associated with proteinuria, occurring after 20 weeks' gestation. Despite improvements in detection and management it remains a leading cause of maternal and particularly fetal morbidity and mortality (Cudihy and Lee, 2009).

Eclampsia is defined as convulsions in a woman with pre-eclampsia and occurs in 5/10 000 maternities. It has a fatality rate of 1.8% with a further 35% of women experiencing a major complication (Tuffnell et al, 2006).

Several factors increase the risk of pre-eclamptic toxaemia: low maternal age, high body mass index, chronic hypertension, previous history of pre-eclamptic toxaemia, and insulin resistance (Cudihy and Lee, 2009).

Pathophysiology

The condition occurs in two stages:

1. Pre-clinical stage (up to 20 weeks' gestation). Inadequate trophoblast invasion soon after implantation leads to incomplete destruction of the muscularis layer of maternal spiral arteries. There is absent or inadequate remodelling of those arteries into the low resistance dilated vessels necessary for placental perfusion
2. Clinical stage (after 20 weeks' gestation). An inflammatory response occurs as a consequence of placental hypoperfusion and hypoxia. The release of inflammatory mediators causes generalized endothelial dysfunction, manifesting as the symptoms and signs of pre-eclampsia (Cudihy and Lee, 2009).

Pre-eclampsia also affects fetal development and wellbeing with decreased placental perfusion leading to intrauterine growth retardation and an increased risk of preterm labour and placental abruption.

Eclampsia is thought to be related to cerebral vasospasm with localized ischaemia; hypertensive encephalopathy and vasogenic oedema may also play a role (Bourjeily and Miller, 2009).

Clinical features

Pre-eclampsia

Pregnancy-induced hypertension (systolic blood pressure >140 mmHg), associated proteinuria (>0.3 g in 24 hours) and/or oedema may affect virtually any organ system to varying degrees (*Table 2*).

Severe pre-eclampsia

Severe hypertension (systolic blood pressure >170 mmHg or diastolic blood pressure >110 mmHg) on two occasions with significant proteinuria (≥1 g/litre).

Critical care management

High dependency unit or intensive care unit care is needed. The definitive maternal treatment for pre-eclampsia is delivery of the fetoplacental unit. It should be noted, however, that up to 44% of cases of eclampsia occurs post-partum with no significant prodrome (Tuffnell et al, 2006). The management of severe pre-eclampsia is based on careful clinical assessment, stabilization, monitoring and timely delivery. The critical care team should be involved early in order to deal with airway and circulatory issues in particular.

Airway and breathing

Persistent seizures will require intubation and ventilation in order to prevent secondary hypoxic brain injury, and transfer to the intensive care unit for further management.

Magnesium sulphate is the treatment of choice for the prevention and treatment of eclamptic seizures. A loading dose of 4 g is given over 5–10 minutes followed by a further infusion of 1 g/hour maintained for 24 hours following delivery or after the last seizure, whichever is the later. Most eclamptic seizures are self-limiting, lasting 3–4 minutes at most; recurrent seizures should be treated with a further 2 g bolus of magnesium or by increasing the infusion rate to 1.5–2.0 g/hour.

Circulation and fluid management

Antihypertensive medication should be commenced in women with systolic blood pressure >160 mmHg or a diastolic blood pressure >110 mmHg, although it may be started earlier in those with additional features of severe pre-eclampsia. Labetalol, nifedipine or intravenous hydralazine can be used as indicated either as single or combination therapy.

Fluid therapy should be restricted to crystalloid at 80 ml/hour or 1 ml/kg/hour to avoid the risk of volume overload and pulmonary oedema. Pulmonary oedema complicates 2.9% of all cases of pre-eclamptic toxaemia and can be cardiogenic or non-cardiogenic in nature (Bourjeily and Miller, 2009). There is a reduction in plasma oncotic pressure secondary to renal albumin loss and reduced hepatic synthesis of plasma proteins; this is further exacerbated by postpartum fluid shift, excessive crystalloid infusions and autotransfusion during labour. Left ventricular dysfunction is also present and can be either systolic or diastolic. Management involves use of low-dose diuretics (furosemide 10 mg), reduction of afterload and blood pressure control.

Oliguria is well tolerated and there is no evidence that maintenance of a specific urine output is important in the prevention of renal failure.

Invasive monitoring is often required, particularly in the face of maternal haemorrhage when fluid restriction becomes inappropriate.

HELLP syndrome

Epidemiology

The syndrome of haemolysis, elevated liver enzymes, and low platelets (HELLP) complicates 0.5–0.9% of all pregnancies and occurs in 10–20% of women with severe pre-eclampsia; 70% of cases are diagnosed antenatally (Haram et al, 2009). Maternal mortality is reported as ~1%, with cerebral haemorrhage or stroke being the leading cause of death. Perinatal mortality is much higher being between 7.4 and 34% depending on gestational age (Sibai, 2004). It is unclear whether HELLP should be classed as a feature of severe pre-eclampsia or whether it is a clinical entity in its own right (Bourjeily and Miller, 2009).

Pathophysiology

The pathophysiology of HELLP syndrome is closely related to that of pre-eclampsia (Haram et al, 2009). Haemolysis is caused by a microangiopathic haemolytic anaemia secondary to red cell fragmentation as they pass through damaged vascular endothelium. This results in an increased plasma lactate dehydrogenase level and reticulocyte count with a reduction in serum haemoglobin concentration.

Elevation of liver enzyme levels reflects hepatic vascular injury as well as haemolysis. Very high levels of aspartate aminotransferase or alanine aminotransferase should prompt further investigation for hepatic infarction or severe congestion. Thrombocytopenia occurs as a consequence of widespread platelet activation in response to endothelial damage. There is increased platelet turnover along with a reduction in platelet lifespan.

Table 2. Organ systems and symptoms associated with pre-eclamptic toxaemia

System	Clinical features
Cardiovascular	Hypertension
	Increased vascular permeability and oedema
	Left ventricular failure
	Decreased circulating blood volume
Respiratory	Pulmonary oedema
	Facial or airway oedema
	Acute respiratory distress syndrome
CNS	Headaches
	Visual disturbance
	Hyper-reflexia
	Cerebral haemorrhage
	Eclampsia
Renal	Reduced glomerular filtration rate
	Proteinuria
	Reduced urea clearance and high uric acid levels
	Oliguria
	Acute renal failure
Liver	Abnormal liver function tests
	Subcapsular haemorrhage
	Epigastric pain or hepatic rupture
Coagulation	Thrombocytopenia
	Impaired platelet function
	Disseminated intravascular coagulopathy
	Haemolytic anaemia, elevated liver enzymes and low platelet count (HELLP syndrome)

Adapted from Tuffnell et al (2006)

Clinical features
The onset of HELLP is usually rapid and preceded by generalized oedema in >50% of cases (Sibai, 1990) (*Table 3*).

Laboratory findings
Signs include the classic triad of: haemolysis (lactate dehydrogenase ≥600 IU/litre), elevated liver enzymes (aspartate aminotransferase or alanine aminotransferase ≥70 IU/litre), low platelets (≤100×10^9/litre).

Critical care management
High dependency unit care may be needed. Management of HELLP syndrome is guided by maternal clinical condition in association with fetal gestational age and wellbeing; there are three main management strategies (Haram et al, 2009):

1. Immediate delivery – preferred option at ≥34 weeks gestation or in life-threatening disease
2. Delivery within 48 hours after maternal stabilization – appropriate at 27–34 weeks' gestation when clinical condition allows
3. Conservative management for more than 48–72 hours – can be considered in women ≤27 weeks' gestation.

Treatment measures are as for pre-eclampsia. The use of corticosteroids for the management of HELLP syndrome (as opposed to the encouragement of fetal lung maturity) does not improve maternal complications, need for transfusion or length of hospital stay (Fonseca et al, 2005).

Table 3. Symptoms seen in haemolytic anaemia elevated liver enzymes and low platelet count (HELLP) syndrome

Systems	Clinical features
Classical	Epigastric or right upper quadrant pain
	Nausea and vomiting
	Headache
	Visual disturbance
	Non-specific viral symptoms
Additional	Hepatic rupture or infarction
	Disseminated intravascular coagulopathy
	Acute renal failure
	Pulmonary oedema
	Cerebral oedema, infarction or haemorrhage
	Placental abruption

Table 4. Causes of massive obstetric haemorrhage

Haemorrhage type	Condition associated
Antepartum haemorrhage	Placenta praevia
	Placental abruption
	Uterine rupture
	Trauma
Postpartum haemorrhage (primary)	Uterine atony
	Retained placenta or products of conception
	Clotting abnormalities
	Placenta accreta, increta or percreta
	Uterine inversion
	Genital tract trauma
Postpartum haemorrhage (secondary)	Puerperal sepsis
	Retained products of conception

Massive obstetric haemorrhage
Epidemiology
In the Confidential Enquiry into Maternal and Child Health report, haemorrhage was the third highest direct cause of maternal death accounting for 6.6 deaths/million maternities. Haemorrhage is also a leading cause of admission to the intensive care unit, being responsible for approximately 20% of direct obstetric admissions to intensive care unit (Lewis, 2007).

Table 4 lists the major causes of massive obstetric haemorrhage. There are several others: previous postpartum haemorrhage, multiple pregnancy, pre-eclampsia or pregnancy-induced hypertension, obesity, prolonged labour, large baby, pyrexia in labour or age >40 years (Arulkumaran et al, 2009).

Aetiology and presentation
Massive obstetric haemorrhage is defined as: blood loss >1500 ml, a decrease in haemoglobin >4 g/dl or acute transfusion requirement of >4 units (Shevell and Malone, 2003).

The normal maternal physiological changes to pregnancy may complicate detection of severe haemorrhage. By term, cardiac output, stroke volume and blood volume have all increased and tachycardia may be the only presenting sign of haemorrhage until 30–40% of the circulating blood volume has been lost (Carlin and Alfirevic, 2008). The haemodynamic effects of massive blood loss will be compounded by aortocaval compression.

Critical care management
It is likely that high dependency unit will be the minimum level of care needed. The initial management of massive obstetric haemorrhage involves maternal resuscitation to restore oxygen-carrying capacity and adequate tissue perfusion as well as simultaneous diagnosis and treatment of the source of blood loss.

Airway and breathing
The patient will need high-flow 100% oxygen. Intubation may be required if moribund.

Circulation and fluid management
Large bore intravenous access will be needed. Blood samples should be sent for full blood count, clotting screen, urea and electrolytes and crossmatch (at least 4 units).

Immediate fluid resuscitation is needed with warmed crystalloid (2 litres) and/or colloid (1–2 litres) as rapidly as required.

Crossmatched blood should be used preferentially to restore circulating volume. Where crossmatched blood is delayed or unavailable, group specific or O RhD negative blood may be used. Aim for a haemoglobin level >8 g/dl.

Fresh frozen plasma should be given in combination with packed cells to prevent dilutional coagulopathy in a ratio of 4 units fresh frozen plasma for every 6 units of red cells. Aim for a prothrombin time <1.5 × mean control. Platelets should be given if the platelet count is $<50\times10^9$/litre, aiming for a count $>75\times10^9$/litre. Cryoprecipitate will be needed if fibrinogen <1 g/litre, aiming for fibrinogen >1.0 g/litre.

Recombinant factor VII may be used in the face of life-threatening haemorrhage in consultation with a haematologist as an adjuvant to standard surgical and medical therapies. Methods of controlling massive obstetric haemorrhage are listed in *Table 5*.

Conclusions

Obstetric emergencies are daunting, particularly as they often involve the mother and baby together. With vigilance and swift management, most obstetric emergencies will avoid the intensive care unit. The outcome is normally favourable for those that do require critical care. **BJHM**

Conflict of interest: none.

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Table 5. Controlling massive haemorrhage

Method	Management
Pharmacological	Oxytocin – 5 units by slow intravenous injection (dose may be repeated) or as an infusion (40 units over 4 hours)
	Ergometrine – 0.5 mg by slow intravenous or intramuscular injection
	Carboprost – 0.25 mg by intramuscular injection repeated at 15-minute intervals to a maximum of eight doses
	Misoprostol – 1000 mg rectally
Surgical	Intrauterine balloon tamponade
	Haemostatic brace suturing
	Bilateral ligation of uterine arteries
	Bilateral ligation or embolization of internal iliac arteries
	Hysterectomy

Adapted from Arulkumaran et al (2009)

KEY POINTS

- Obstetric emergencies requiring critical care are becoming increasingly more frequent.
- Management always involves a standard 'airway, breathing, circulation' approach.
- Control of catastrophic haemorrhage is often required with the involvement of haematologists.
- Communication between obstetricians, anaesthetists, physicians, midwives and the critical care team is the key to rapid management.