

Maternal critical care: part II

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Learning objectives

By reading this article you should be able to;

- Apply the principles of care of the pregnant or recently pregnant woman in critical care.
- Describe the pregnancy-specific conditions in patients presenting to critical care.
- Consider the pitfalls in managing the obstetric patient with pregnancy-related conditions.

Pregnancy is becoming more complex to manage because of the increasing prevalence of multiple morbidities and advanced maternal age. In Part I of this article we covered the general principles of management.¹ In Part II we discuss pregnancy-specific conditions and pitfalls in the setting of critical care. The commonest reasons for admission to critical care in pregnant women are massive or major obstetric haemorrhage (MOH), sepsis, and pre-eclampsia (PET).² Women who have undergone fertility treatment are generally more likely to be admitted to ICU.

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Key points

- Multiple comorbidities in pregnant women can be more challenging to manage in critical illness.
- Close working between the critical care, other specialist, and maternity teams is essential.
- Maternal admissions to critical care result most frequently from major obstetric haemorrhage, sepsis, and pre-eclampsia.
- Non-obstetric causes of deterioration should always be considered.

MOH, sepsis, and PET have been covered separately in previous articles in this journal. Here we emphasise their management in the context of critical illness. Challenges arise because of alterations in maternal physiology. Critical care specialists should be involved early in the management of pregnant and peripartum women, whose condition may deteriorate rapidly. For these patients, especially in the context of changing physiology, the emphasis should be on making, not simply excluding a diagnosis. Many medical disorders can mimic obstetric disorders. Expediting delivery alters the progression of obstetric but not medical disorders.

MOH

Uterine blood flow at term is 600–900 ml min⁻¹. Uncontrolled bleeding from the uteroplacental bed is the commonest cause of maternal death worldwide and it is the most frequent obstetric reason for admission to critical care. It is postpartum haemorrhage (PPH) and not antepartum haemorrhage that is associated with the vast majority of maternal morbidity.

Most PPH cases referred to critical care are primary (occurring within the first 24 h after delivery). Emergency Caesarean section, prolonged labour, multiple pregnancy, polyhydramnios, macrosomia, obesity, intrapartum sepsis,

and uterine inversion are all associated with an increased risk. Secondary PPH is defined as excessive vaginal bleeding in the period from 24 h after delivery to 12 weeks postpartum. The commonest cause of secondary PPH is endometritis or retained products of conception.

Blood loss of ≤ 500 ml after a vaginal delivery or ≤ 1000 ml after Caesarean section is considered normal. There is no universally agreed definition of MOH and while a loss of ≥ 1500 ml is commonly suggested for convenience, women of low weight and stature will have reduced reserve. Activation of the MOH protocol ensures the urgent availability of the requisite clinical and support staff, appropriate initial blood products, and established lines of communication with laboratory staff. It is essential to be familiar with local systems and guidelines.

While PPH can be extremely brisk, it can also be insidious manifesting only as a trickle of ongoing vaginal loss. Quantifying losses is problematic and a lag between actual and calculated losses can cause delay in activating the MOH protocol and transferring to the operating theatre for evaluation and intervention. An obstetric shock index (HR/systolic BP) >1 has been shown to be associated with PPH and the need for intensive resuscitation and blood transfusion.^{3,4}

The usual cause of MOH is uterine atony, but other causes must also be ruled out systematically (Table 1). Usually women with PPH will be referred to critical care after resuscitative efforts and a plan for haemostasis has been initiated in the maternity unit. Once an MOH is declared, urgent obstetric and anaesthetic assessment, i.v. access, volume replacement, and emptying the bladder are undertaken. Bimanual uterine compression may temporise losses. Uterine atony must be immediately corrected with uterotonic drugs given in sequence (Table 2). Tranexamic acid should also be given. Transfusion should be via an appropriate warmer in appropriate ratios of packed red cells to clotting components: point of care thromboelastometry is invaluable. Transfusion targets are the same as in non-pregnant individuals, as is the need to ensure normothermia, and correct hypocalcaemia and acidaemia.

Uterine-preserving interventions include arterial ligation, B-Lynch compression sutures, uterine packing and uterine balloon tamponade. Timely hysterectomy should be performed if necessary to save life. Where MOH is anticipated (morbidly adherent placenta) prophylactic bilateral placement of arterial balloon catheters before Caesarean section may be uterus-preserving. In cases of unanticipated MOH, the role of interventional radiology services is less well-defined: they should be accessed in accordance with the locally agreed policy. In some cases, it may be difficult for resuscitative efforts to keep pace with losses, requiring temporising

measures such as manual pressure on the aorta or aortic cross-clamp. Resuscitative endovascular balloon occlusion of the aorta (REBOA) does not require radiological expertise, has an established role in major trauma, and may have an emerging role in MOH.⁵

There are no universally agreed criteria for when critical care admission is necessary, but it includes massive transfusion with metabolic insult and organ impairment. Before admission to critical care, it is important to ascertain which procedures have been performed and if the uterus is still *in situ*. A plan of action in the context of recurrent bleeding must be agreed (e.g. return to theatre, transfer to interventional radiology suite). Uterotonic drugs must be immediately available (Table 2).⁶ It is important to discuss the ongoing need for uterotonics with the obstetricians on an individualised case-by-case basis.

Contact the obstetric team if there is evidence of uterine tenderness, offensive vaginal discharge, passage of large clots, or continuous bleeding. Early clinical assessment and an ultrasound scan are warranted to exclude retained products of conception. Occasionally, there may be an intrauterine balloon tamponade system in place (e.g. Bakri).

It is important to ensure that the uterus is well contracted around the balloon—an increasing symphysis-fundal height and new blood loss into the drains indicates fresh bleeding and should prompt immediate attendance by the obstetric team.

The critical care management of MOH is otherwise as for any major haemorrhage/transfusion: correction of coagulopathy, monitoring for rebleeding and transfusion reactions, establishing normothermia, and ensuring the resolution of metabolic endpoints such as serum lactate. An analgesia plan will also be required before ‘waking and weaning’ from mechanical ventilation.

Complications of PPH include Sheehan’s syndrome (pituitary necrosis).⁷ Hypoperfusion of other organ systems may result in brain injury, type II myocardial infarction, ischaemic hepatitis, and acute kidney injury (AKI).

Sepsis

The physiology of normal pregnancy and response to labour may mask the presence of sepsis. Intrapartum, women may be tachypnoeic with increases in temperature, white cell count, and lactate even in the absence of infection. Assessment by the multidisciplinary team (MDT) with close attention to physiological trends and a high index of suspicion. Delirium is an ominous sign in maternal sepsis; it is seen infrequently in the labour suite and may be misdiagnosed as puerperal psychosis.

The Sepsis-3 definition of sepsis makes reference to the Sequential Organ Failure Assessment score (SOFA), which is not formally validated in pregnancy. The WHO in 2017 adopted the following definition of maternal sepsis:

‘Maternal sepsis is a life-threatening condition defined as organ dysfunction resulting from infection during pregnancy, childbirth, post-abortion, or postpartum period.’⁸

The maternal sepsis bundle should be initiated without delay (refer to Inpatient Maternal Sepsis Tool, UK Sepsis Trust).⁹ Fluid resuscitation of 30 ml kg⁻¹ may be deemed excessive, especially peripartum, as the maternal circulation

Table 1 Causes for obstetric haemorrhage—‘the 4 Ts’

Tone	Uterine atony
Trauma	Haematoma Laceration Inversion, uterine rupture, or both
Tissue	Retained tissue Invasive placenta
Thrombin	Coagulopathy

Table 2 Uterotonic drugs used in PPH. ^aSyntometrine is ergometrine 500 µg with oxytocin 5 U

Drug class	Example	Mode of action	Important considerations
Oxytocin	Syntocinon ^a 5 U i.v. or i.m.	Myometrial contraction	Onset within 1 min i.v. (4 min i.m.). May be repeated to total 10 U followed by infusion (e.g. 40 U over 4 h) Causes transient hypotension, arrhythmias, and nausea. Structurally similar to antidiuretic hormone: causes water retention and hyponatraemia
Ergot alkaloids	Ergometrine ^a 250–500 µg i.m. I.V. injection slowly with caution	Vasoconstriction and myometrial contraction	Highly emetogenic—give antiemetic. Causes hypertension (can be severe), coronary vasospasm, and pulmonary hypertension. Avoid in PET, hypertension, coronary disease, aortopathies, aneurysms
Prostaglandin analogues	1. Carboprost (e.g. Hemabate) 250 µg i.m.	Prostaglandin F _{2α} agonist: myometrial contraction	May only be given i.m. Dose of 250 µg may be repeated eight times at 15 min intervals. Prostaglandin analogues can cause bronchospasm but may be given in asthma with atonic bleeding because of threat to life. Cause severe diarrhoea, hyperthermia, rash, dizziness See note above regarding unwanted effects in prostaglandin analogues
	2. Misoprostol 600 µg per rectum	Prostaglandin E ₁ agonist: myometrial contraction and cervical relaxation	

is vulnerable to fluid overload. Reassessment after each 500 ml is prudent. Special care should be applied in the context of comorbid PET. Vasopressor therapy may be needed concurrently with resuscitation with i.v. fluids. Noradrenaline (norepinephrine) is the first-line agent. A target MAP of ≥ 65 mmHg applies to non-pregnant patients, but is nevertheless a reasonable starting point.

Empiric antimicrobial therapy (to be given within 1 h on suspicion of sepsis) should be guided by local hospital policy and microbiological advice. Sepsis may be attributable to obstetric or non-obstetric causes.¹⁰ Obstetric infections are usually polymicrobial and initial antimicrobial therapy must cover both Group A *Streptococcus* and *E. coli* (the organisms most commonly associated with maternal mortality). In a recent meta-analysis, i.v. immunoglobulin, used as an adjunct to clindamycin for the treatment of streptococcal toxic shock, was found to be beneficial.¹¹

Source control is paramount. Obstetric sites of infection include retained products of conception, chorioamnionitis, endometritis, pelvic abscess, and wound infection. Chorioamnionitis should be suspected if there is foul-smelling liquor, pyrexia, maternal and fetal tachycardia. The risk is increased in the presence of prolonged rupture of membranes. Chorioamnionitis is the only definite indication for delivery in maternal sepsis; otherwise, delivery should be guided by obstetric indications.

Non-obstetric infection may arise from any site, with pneumonia and pyelonephritis being common. A viral or fungal aetiology should be considered especially if the patient is immuno-naïve (e.g. herpes simplex virus [HSV]) or immunocompromised. COVID-19 and the other newly-identified associated diseases of the 21st century may pose serious health risks to obstetric patients as seen in the current pandemic.¹² Diseases that may masquerade as infection are important to consider in the context of deterioration without an identifiable cause. These include

haemophagocytic lymphohistiocytosis, thrombotic microangiopathy, malignancy, and autoimmune diseases.

PET

PET affects 5–7% of pregnancies and is one of a heterogeneous group of hypertensive disorders in pregnancy that include chronic essential hypertension, gestational hypertension, and superimposed PET on chronic hypertension. It is defined as new-onset hypertension after 20 weeks' gestation associated with proteinuria, evidence of end-organ involvement, or utero-placental dysfunction.¹³ The diagnosis of hypertension is made by recording a BP of $\geq 140/90$ mmHg on two separate occasions, at least 4 h apart. A protein-creatinine ratio of ≥ 30 mg mmol⁻¹ is used as a surrogate for 24-h urine collection. While there are a range of differential diagnoses, it is safest to assume that new-onset hypertension in pregnancy with proteinuria on urinalysis could be attributable to PET in the first instance. The early diagnosis and management of PET includes MDT input; midwives, obstetricians, maternal fetal specialists, obstetric physicians, neonatologists, anaesthetists, and critical care. The National Institute of Health and Care Excellence (NICE) Guideline on Hypertension in Pregnancy provides comprehensive guidance on the clinical criteria for the choice of the critical care level bed required for mild, moderate, and severe PET.

Once diagnosed, it is recognised that PET can rapidly progress from a 'mild' condition to one with severe end-organ involvement compromising the physiology of both mother and fetus. Early-onset PET often causes severe morbidity to mother and fetus.¹⁴ A diagnosis of 'PET with severe features' is made if the patient has a systolic BP (SBP) ≥ 160 mmHg or any of the following features: cerebral involvement (e.g. headache, visual disturbance, or altered mental state), an otherwise unexplained severe right upper quadrant pain, serum transaminases more than twice the normal limit, platelets less

than $100 \times 10^9 \text{ L}^{-1}$, progressive renal insufficiency, or pulmonary oedema.

Treatment

The treatment of PET is delivery of the placenta. It should be noted that PET is frequently diagnosed *after* birth and can persist and worsen initially before resolving. While risk to the mother takes precedence, those with mild disease are frequently monitored, allowing the pregnancy to continue with the intention of improving fetal outcome. Women with PET frequently labour quickly and Caesarean section is not always necessary, but in the presence of severe disease, birth must be expedited once supportive measures have been instituted. Supportive measures include control of hypertension, reducing the risk of eclampsia, and correction of coagulopathy. At all times, attention must also be given to preventing fluid overload.

Supportive measures

In the context of PET, a BP of 160/110 mmHg is an obstetric emergency. The goal of treatment is to aim for a SBP of <140 mmHg; it is systolic pressure that is associated with adverse endpoints such as intracerebral haemorrhage. $\text{SBP} \geq 180$ mmHg is an indication for urgent treatment with boluses of an i.v. drug followed by an infusion, or oral nifedipine modified release (MR) (see Table 3).^{15,16} Improvement of symptoms such as headache may be seen as BP improves. Infusion of an antihypertensive agent is usually considered an indication for invasive monitoring of arterial BP. A central venous catheter is not required initially and should not delay therapy. Once BP control has been achieved the woman can be weaned from i.v. to enteric therapies in a controlled manner: significant hypertension may persist for some time. All women with PET should be under close monitoring postpartum because of the risk of worsening BP control: a physiological increase in BP occurs 3–5 days postpartum because of volume expansion and fluid shift.

Women are highly vulnerable to fluid overload in the peripartum period even in the absence of PET. In PET, pulmonary oedema occurs in 9.5% of women with severe disease.¹⁷ In contrast with sepsis, the capillary leak is subject to an increased driving pressure (i.e. hypertension is present). The increased afterload and reduced vascular compliance result in ventricular remodelling and diastolic dysfunction. As a default, it is important to restrict fluids in severe disease, even at the expense of kidney function (which may also be adversely affected by the PET itself). Reducing the fluid

volume in which drugs are diluted through the use of concentrated formulations can be helpful, as can the use of concentrated feeds. Assessing fluid requirements in PET is notoriously difficult, especially as the intensivist will often become involved when other problems are present, such as PET combined with sepsis and AKI. A thorough assessment must be made at the bedside, taking into account symptoms, trends, and changing physiology. Adjuncts such as focussed echocardiography have a role. Table 4 describes the therapeutic options available for life-threatening emergencies of PET.

Once pulmonary oedema is established, efforts to reduce both preload and afterload while paying attention to factors that exacerbate cardiac ischaemia are equally important. In this respect nitrate infusions, opioids (e.g. diamorphine), furosemide, and CPAP all have a role. If the patient deteriorates to require tracheal intubation and artificial ventilation, it is vital that BP is controlled and that the pressor response to direct laryngoscopy is attenuated. While invasive ventilation may ameliorate pulmonary oedema, the intensivist should be mindful that the brain is also vulnerable (see posterior reversible encephalopathy syndrome [PRES] below).

Magnesium sulphate infusions reduce the risk of eclampsia and should be instituted in the presence of PET with severe features. While magnesium is a neuromuscular blocking agent, it does not reduce blood pressure significantly in PET: conventional antihypertensive drugs will be required. Thrombocytopenia may occur even in the absence of the haemolysis, elevated liver enzymes, and low platelets (HELLP) syndrome. Correction of platelets to above $50 \times 10^9 \text{ L}^{-1}$ is important before delivery ($>80 \times 10^9 \text{ L}^{-1}$ if a neuraxial technique is being considered). NSAIDs should be avoided in women with PET and thrombocytopenia or AKI.

Subcapsular haematoma and liver rupture

The overall mortality from subcapsular haematoma, liver infarction, and liver rupture is up to 50% in those with PET or HELLP. The incidence of liver rupture in pregnancy is estimated as one per 67,000 deliveries and one per 2000 patients with PET or HELLP. The current theories suggest that subsequent vascular injury from endothelial damage leads to formation of microvascular thrombi and necrosis that result in rupture.¹⁸ The identification of a triad, epigastric/right upper quadrant pain, hypotension without evidence of a cause for bleeding, and clinical confirmation of a hypertensive disorder, should steer to a high index of suspicion to consider these life-threatening conditions.¹⁹ Management with an MDT approach is paramount. Options range from a conservative

Table 3 Commonly used antihypertensive agents in PET

Drug	Route			Adverse effect/interactions
	Enteric	I.V. bolus	I.V. infusion	
Hydralazine		Y	Y	Maternal hypotension and tachycardia. Adequate hydration before administration is important
Labetalol	Y	Y	Y	Nausea (4.1%) Dizziness (4.1%)
Nifedipine MR	Y			Headache (5.4%), maternal tachycardia (4.1%), dizziness (2.7%)

approach to more aggressive interventions and should be individualised. The surgical options depend on the severity of the rupture intervention and include partial resection, embolisation and hepatic artery ligation, packing of the bleeding and liver transplantation.

Table 4 PET emergencies—therapeutic options and considerations

	Initial management	Clinical tips
Pulmonary oedema	Conventional supportive management including oxygen, diuretics and where necessary CPAP Once stabilised expedite delivery Neuraxial anaesthesia preferable where possible	Most cases of PET pulmonary oedema are postpartum Differential diagnoses: 1. Acute coronary syndrome (e.g. related to spontaneous coronary artery dissection) 2. Peripartum cardiomyopathy 3. Overzealous fluid administration (common) 4. Tocolytic use 5. Lung injury (e.g. transfusion/infection/ARDS)
Hypertensive encephalopathy: • Cerebral oedema • Intracranial haemorrhage	Supportive management and urgent optimisation of BP control Magnesium infusion as eclampsia prophylaxis in all symptomatic PET	Cortical blindness is managed by controlling and optimising the BP Differential diagnoses: metabolic abnormalities, drugs, infection, and cerebral venous sinus thrombosis
Eclampsia	A 4 g loading dose of magnesium sulphate (MgSO ₄) followed by an infusion 1–2 g i.v. ^a ^a AKI: halve the infusion dose. Give additional bolus of MgSO ₄ should the woman have a fit while on MgSO ₄ . If refractory to MgSO ₄ give antiepileptic drugs (e.g. benzodiazepines, levetiracetam)	1% Risk of recurrence of eclampsia in a future pregnancy
Subcapsular liver rupture	Stabilise and contact nearest liver unit	Subsequent pregnancies are infrequently complicated by HELLP (2–19%)

Hypertensive encephalopathy

The risk of stroke in women with PET/eclampsia appears highest in the postpartum period. The most common symptoms of stroke include severe headache, impairment of consciousness, and increased systolic BP. Women with severe PET and a high isolated systolic BP should be immediately treated with antihypertensive therapy because of a very high risk for haemorrhagic stroke. PET is also associated with PRES and reversible cerebral vasoconstriction syndrome (RCVS). RCVS can be complicated by seizures, ischaemic and haemorrhagic stroke, including subarachnoid haemorrhage.²⁰ PRES in severe cases can cause seizure, status epilepticus, coma, or all three. There should be a low threshold for CT scanning to exclude a haemorrhagic stroke or an MRI scan to confirm the diagnosis of PRES or RCVS.

Other conditions

Respiratory, cardiovascular, haematological, and renal system disorders are common causes for admission to critical care; neurological dysfunction is less frequent. It is important to involve specialist medical teams when organ dysfunction is present.

Respiratory disorders

Chronic maternal hypoxia (Spo₂<85%) affects fetal growth and well-being. Asthma can worsen during pregnancy; the management is the same as in non-obstetric patients. Decomensation ('tiring') should be considered at Paco₂≥4.0 given the physiological respiratory alkalosis of pregnancy.

Acute respiratory distress syndrome (ARDS) may arise as a result of obstetric causes including MOH, sepsis and amniotic fluid embolism (AFE). There is a variety of non-obstetric causes including viral pneumonitis (e.g. COVID-19, H1N1). Management of mechanical ventilation is outlined in Part I.¹

Cardiovascular disorders

The aetiology of cardiac disease in pregnancy includes PET; dilated cardiomyopathy; obesity cardiomyopathy; peripartum cardiomyopathy (PPCM); myocarditis; hypertrophic obstructive cardiomyopathy; arrhythmogenic right ventricular cardiomyopathy and ventricular dysfunction.²¹ Management of pulmonary oedema is described above. Ischaemic heart disease in pregnancy is likely to become more prevalent because of the increased prevalence of cardiovascular risk factors in women of childbearing age such as obesity, hypertensive disorders, smoking and advanced maternal age.²² PPCM is characterised by reduced ejection fraction (<45%), without known pre-existing structural heart disease. PPCM presents in the 3rd trimester or within 5 months of delivery. The incidence is one in 300–3000. Management includes standard treatments for heart failure and anticoagulants to prevent intracardiac mural thrombus. A toxic metabolite of prolactin may drive PPCM and bromocriptine has been used as a treatment in very small studies—its use remains controversial but can be considered on a case-by-case basis.²³

Morbidity and mortality are high in obstetric patients with pulmonary arterial hypertension.²⁴ In the 3rd trimester and in the initial postpartum period there is a greater risk of venous thromboembolism, refractory right heart failure, and

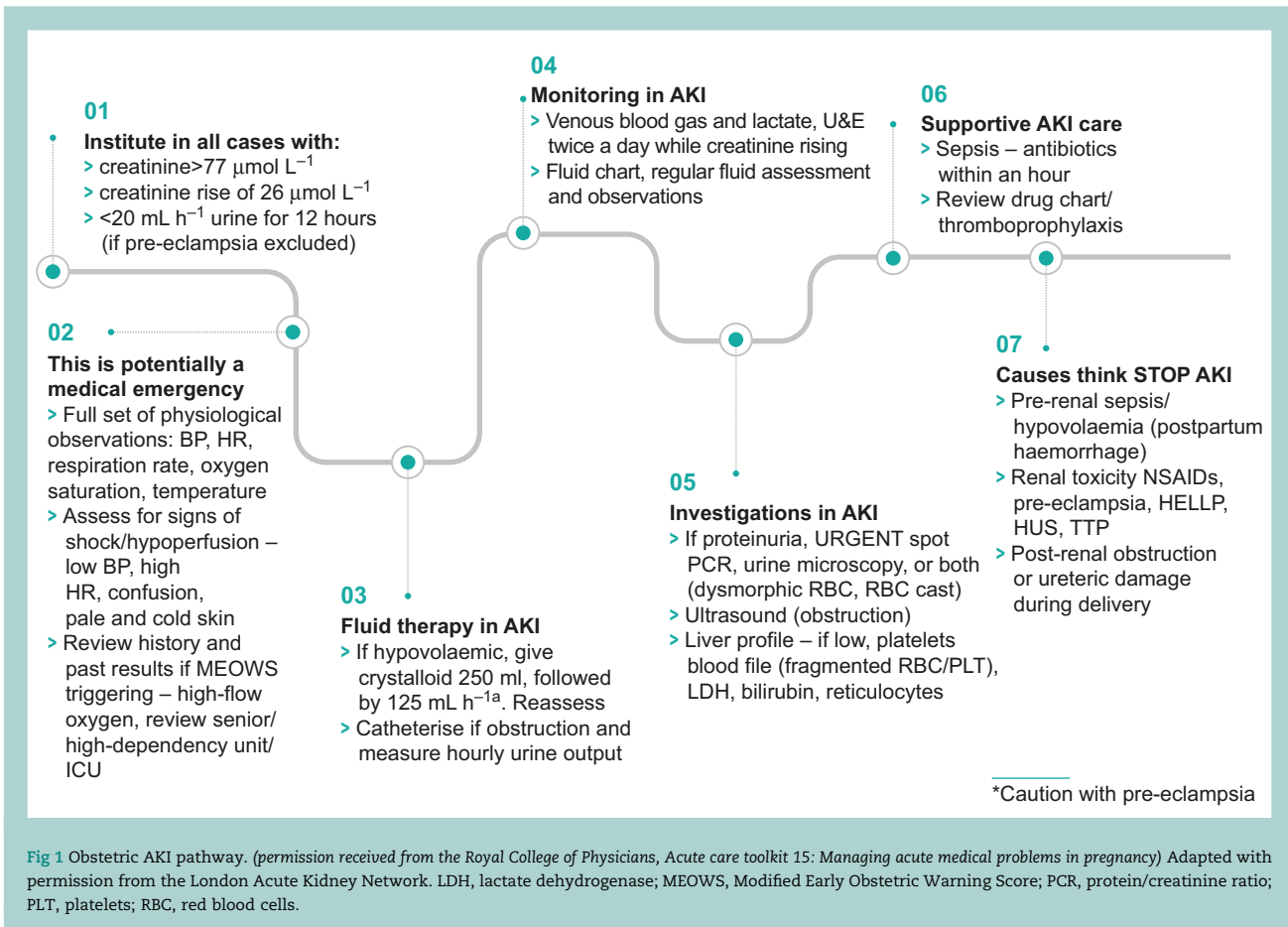


Fig 1 Obstetric AKI pathway. (permission received from the Royal College of Physicians, Acute care toolkit 15: Managing acute medical problems in pregnancy) Adapted with permission from the London Acute Kidney Network. LDH, lactate dehydrogenase; MEOWS, Modified Early Obstetric Warning Score; PCR, protein/creatinine ratio; PLT, platelets; RBC, red blood cells.

pulmonary hypertensive crisis. Therapeutic options include inhaled iloprost or i.v. prostaglandin. Maternal mortality has improved to 17–33%, and neonatal survival rate to 87–89% with the introduction of newer agents for the management of pulmonary arterial hypertension.

Renal disorders

The indications for renal replacement therapy are the same as for non-obstetric causes of AKI. However, in the antenatal period a persistent maternal urea concentration of $17\text{--}20 \text{ mmol L}^{-1}$ should also be considered as a trigger to offer renal replacement therapy for fetal well-being.²⁵ The incidence of AKI is higher in the postpartum and third trimester. All AKIs in pregnancy regardless of aetiology are associated with increased morbidity and mortality. Some causes are pregnancy-specific or are more common during pregnancy. The aetiology can vary and usually coexists (e.g. PPH and/or PET and/or sepsis).²⁶ The development of an obstetric AKI bundle of care (Fig. 1) provides a structured approach.

Lupus nephritis can present during pregnancy (20–49% of women with systemic lupus erythematosus have renal involvement). In the first trimester this may present as proteinuria, hypertension, and AKI. A scleroderma renal crisis is a life-threatening condition. This is the only time ACE inhibitors

should be considered (they are otherwise contraindicated in pregnancy).²⁷

Haematological disorders

Thromboembolism remains the leading causes of direct maternal death. If a woman presents with a sub-massive pulmonary embolism, aim not to deliver the baby at this time but treat the pulmonary embolism. An individualised approach with timely intervention and definitive treatment is necessary. Options to treat the thrombus include i.v. unfractionated heparin, thrombolysis, percutaneous catheter-based embolectomy, or surgical embolectomy depending upon local resources.²⁸

Disseminated intravascular coagulopathy may arise as a result of AFE, MOH, and sepsis. Close working with a haematologist and access to blood products is important. AFE is a life-threatening inflammatory anaphylactoid response to fetal antigens entering the maternal vasculature through a disruption in the maternal fetal barrier. This leads to increased pulmonary and systemic vasculature resistance, decreased left ventricular function, and coagulopathy resulting in respiratory failure and cardiogenic shock. Management is supportive. The predisposing conditions include rapid labour, meconium, older maternal age, post-term pregnancy,

Table 5 TTP, HUS, and HELLP: common clinical features

Disease	TTP	Atypical HUS	HELLP
Incidence	1 in 25,000	1 in 2500	10–20% of PET
Trimester-specific presentation	2nd trimester	Majority postpartum	3rd trimester and postpartum
Evidence of AKI	30–80%	70% Dialysis-dependent	3–15%
Neurological sequelae	Common	Uncommon	Seizure secondary to eclampsia
Increased hepatic enzymes	No	No	Yes
ADAMTS-13 present	Deficient	Present	Reduced
Coagulopathy present	No	No	Sometimes
Management	Plasma exchange	Disease-specific	Supportive
Timing of delivery	Not affected	Not affected	Affected

eclampsia, Caesarean section, placental abruption, and polyhydramnios.

The thrombotic microangiopathy are rare (estimated 1:25,000 pregnancies) and can be a challenge to diagnose. Thrombotic thrombocytopenic purpura (TTP) and haemolytic uremic syndrome (HUS) are the most common examples that may both masquerade as and coexist with PET.²⁹ Table 5 depicts the differences between these conditions and HELLP.

Metabolic and electrolyte disorders

Acute fatty liver of pregnancy (AFLP) <1:7000 births may be confused with PET and the HELLP syndrome, which may also be present, and are cited as potential risk factors. The cause is unknown but it has been linked with deficiency of fetal long-chain 3-hydroxyacyl CoA dehydrogenase. Multiple gestation, male fetal sex, and a low BMI are also risk factors. It usually presents in the third trimester or in the first few days postpartum. They have raised aminotransferases (5–10 × normal range) and develop signs and symptoms of acute liver failure including jaundice, ascites, encephalopathy, disseminated intravascular coagulopathy, hypoglycaemia, AKI, and multiorgan failure. The Swansea

criteria: 14 clinical, pathological, and radiological features (Table 6) may aid diagnosis.³⁰ The presence of more than six features in the absence of another reason is consistent with a diagnosis of AFLP. Management involves delivery of the fetus, ongoing supportive critical care, commencement of i.v. N-acetyl cysteine and vitamin K, and discussion with a specialist liver unit. Transient diabetes insipidus can occur because of the release of vasopressinase from the placenta leading to a four-fold increase in the rate of breakdown of arginine vasopressin. Intranasal DDAVP can be used to manage polyuria. Other causes of acute liver failure to consider include viral hepatitis (including HSV), drugs, and Budd-Chiari syndrome.

Human placental lactogen promotes insulin resistance in pregnancy. Starvation ketosis is not uncommon, and ketones should always be measured when metabolic acidosis is present.³¹ Diabetic ketoacidosis is considered a medical emergency during pregnancy. The current recommendation is to discuss and refer for high dependency unit care. A multidisciplinary approach is required, with prompt management according to local guidelines for diabetic ketoacidosis. The aim is not to deliver the baby at this time but stabilise and correct the metabolic acidosis, identify and manage the precipitating factors.

Women may be vulnerable to water intoxication in the peripartum period: excessive fluid intake, prolonged labour, and the use of syntocinon intrapartum are contributory factors.³² Acute onset of hyponatraemia and associated cerebral oedema can lead to cortical irritation and seizures in both the mother and neonate. The first line of treatment is restriction of fluids. Hypertonic saline is an effective treatment for seizures caused by cerebral oedema. Once seizures are terminated, aim for a sodium correction of <1 mmol every 2 h and no more than 10 mmol L⁻¹ over 24 h to avoid central pontine myelinolysis.

In summary, as obstetric patients are increasingly becoming more complex, the management in critical care should be seamless and needs a multidisciplinary approach.

MCQs

The associated MCQs (to support CME/CPD activity) will be accessible at www.bjaed.org/cme/home by subscribers to BJA Education.

Table 6 A Description of the Swansea Criteria for the diagnosis of acute fatty liver of pregnancy³⁰. APTT, activated partial thromboplastin time; ALT, alanine aminotransferase; AST, aspartate transaminase; PT, prothrombin time.

Six of the following 14 features are required:

History	Abdominal pain Polydipsia/polyuria Vomiting
Examination	Encephalopathy
Biochemistry	Ammonia >47 µmol L ⁻¹ AST/ALT >42 IU L ⁻¹ Bilirubin >14 µmol L ⁻¹ Creatinine >150 µmol L ⁻¹ Glucose <4 mmol L ⁻¹ Urate >340 µmol L ⁻¹
Haematology	Leucocytes >11 × 10 ⁹ L ⁻¹ PT >14 s or APTT >24 s
Imaging	Ascites or bright liver echotexture
Histology	Microvesicular steatosis

Declaration of interests

The authors declare that they have no conflicts of interest.

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