Medical consultations on the labour ward: assessment and management of common medical presentations

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Abstract

Pregnancy and the postpartum period are a time of significant physiological adaptations that can impact on the presentation, assessment and diagnosis of common medical problems. Physicians can have limited postgraduate obstetric experience and yet are called upon to assess a pregnant woman in the context of new or pre-existing medical disease on the labour ward. This article reviews the evaluation and management of breathlessness, chest pain, palpitations, seizures, headache, acute kidney injury and sepsis. Through comprehensive assessment that is cognisant of normal pregnancy physiology, and by using the excellent resources now available from specialist organizations, physicians can offer a unique and valuable perspective for the multidisciplinary care of pregnant individuals.

Keywords

Acute kidney injury; maternal mortality; maternal sepsis; pregnancy; pulmonary embolism; seizure

Key points

- Pregnancy should be viewed as a stress test capable of unmasking unknown cardiovascular and/or metabolic vulnerabilities – older women, those with pre-existing medical co-morbidities and those carrying twin or higher multiple pregnancies can be particularly at risk
- While pregnancy and the postpartum period incur an increased risk of thromboembolic disease, the possibility of cardiac disease must always be considered in pregnant or recently pregnant patients presenting with chest pain, breathlessness and/or palpitations
- Troponin, brain natriuretic peptide and C-reactive protein testing retain their validity in evaluating pregnant and postpartum patients; D-dimer testing is not accurate in pregnancy and should not be used to risk stratify women with possible pulmonary embolism
- Do not unnecessarily withhold imaging or treatment from pregnant or breastfeeding women – although some interventions can carry proven or theoretical risks of harm to the pregnancy, fetus or infant, these must be balanced against the known risks of inadequate treatment and investigation

Introduction

The most common causes of maternal death in UK during the 2020–2022 triennium were thromboembolism, COVID-19 and cardiac disease. Recurring themes in Mothers and Babies: Reducing Risk through Audits and Confidential Enquires across the UK (MBRRACE-UK) include delays and omissions in the care of pregnant women because of fears that treatment or imaging could harm the fetus, and knowledge gaps in how the physiology of pregnancy can alter clinical assessment.

Labour wards can feel unfamiliar, but physicians can offer specialist expertise and contribute to constructive multidisciplinary team (MDT) discussions regarding investigation, management, place of care and timing of delivery. Excellent resources exist to support physicians, including the Acute Care Toolkit 15,[1] specialist prescribing resources (Table 1), and the adoption of a national Maternity Early Warning Score across England for the early recognition of deteriorating mothers.

Maternal Medicine Networks, established across England in 2021, provide additional specialist pre-pregnancy, antepartum and postpartum care, routes of referral and provision of education to ensure parity in access to obstetric medicine services.

Normal physiology of pregnancy

Physiological adaptations to pregnancy are evident within weeks of conception to support implantation, fetal growth and development, and to prepare for childbirth. Clinically, these changes manifest as the following: increased heart rate; dilutional changes in haemoglobin, platelets, sodium and albumin; increased tidal volume with a compensated respiratory alkalosis; and a fall in serum creatinine concentration.

Increased turnover of the coagulation and fibrinolytic systems in pregnancy can lead to false-positive D-dimer results, while reduced neutrophil apoptosis leads to a mild neutrophilia. Other tests, such as for C-reactive protein, troponin and brain natriuretic peptide (BNP), retain their validity in evaluating pregnant patients.

The physiological adaptations of pregnancy are more pronounced in those carrying twins or higher multiples, and can be less well tolerated by individuals with an advanced maternal age, or with pre-existing cardiometabolic or respiratory disease – all increasingly common demographics as a result of societal changes, effective contraception and assisted reproduction technologies.

A particularly high-risk period is delivery and the immediate postpartum period. In this period blood loss during labour and compensatory prothrombotic adaptations combine with a reversion to non-pregnant physiology with an increased incidence of aortic dissection, spontaneous coronary artery dissection, peripartum cardiomyopathy, thromboembolic disease and stroke.

Breathlessness

Breathlessness is a common symptom of pregnancy. This physiological breathlessness arises initially secondary to progesterone-induced increases in tidal volume, and later in pregnancy from diaphragmatic splinting by the gravid uterus. It is typically mild, gradual in onset and without associated ‘red flags’ (e.g. chest pain, syncope). Observations, including oxygen saturations and respiratory rate, are normal – individuals breathe deeper, not faster.

Contributory factors that can worsen breathlessness such as iron deficiency anaemia should be considered by measuring haemoglobin (normal value ≥110 g/litre first trimester, ≥105 g/litre second and third trimesters, ≥100g/litre postpartum) and ferritin (target in pregnancy >30 microgram/litre), and any abnormalities corrected.

If assessment suggests another cause, the breathlessness should be appropriately investigated. It is safe to perform chest radiographs (CXRs) in pregnancy (Table 2), and they can evaluate for consolidation, infiltrates including oedema, pneumothoraces and masses. A CXR should always be

performed before more detailed imaging such as computed tomography (CT) or ventilation/perfusion (V/Q) scans.

Because of the increased thrombotic risk in pregnancy and the postpartum period, pulmonary embolism (PE) is an important differential diagnosis. Low molecular weight heparins do not cross the placenta and should be started while investigations are continuing, with an appropriate MDT discussion of dosing in the event of recent/imminent neuraxial anaesthesia or other obstetric concerns.

CXR and electrocardiography (ECG) are the key initial tests, and if there is a clinical suspicion of deep vein thrombosis (DVT), compression duplex ultrasonography (US) should also be performed. If US demonstrates a DVT, no further investigations are required as DVT treatment addresses any concomitant PE.

If there is a continuing suspicion of PE, further imaging is required with CT pulmonary angiography (CTPA) or V/Q scanning. As outlined in Table 2, CTPA and V/Q differ with regard to the radiation dose to the fetus and maternal breasts, but both techniques can be safely performed in pregnancy. CTPA allows a broader differential diagnosis to be ruled in and out, versus the binary assessment on a V/Q scan of whether or not a PE is present.

D-Dimer testing and the use of probability tests, including the existing pregnancy-adapted algorithms, are not yet sufficiently accurate and should not be used.

The possibility of a cardiac aetiology for breathlessness should always be considered in pregnancy. Pregnancy can trigger *de novo* disease (e.g. peripartum cardiomyopathy) and unmask pre-existing disease (e.g. undiagnosed dilated cardiomyopathy). Features of cardiac failure should thus be explicitly sought on history and examination, and BNP measurement with or without echocardiography be considered.

Chest pain and palpitations

Chest pain in pregnancy has a broad differential diagnosis from gastro-oesophageal reflux causing dyspepsia, gallstones causing biliary colic, and PE as described above. There are also specific entities to be aware of; for example, pneumothorax or pneumomediastinum can develop during the second stage of labour.

In addition, while events such as coronary artery or aortic dissection and acute coronary syndrome are rare in seemingly healthy young women, pregnancy should be viewed as a stress test for the cardiovascular system that can unmask as yet unappreciated vulnerabilities. This is exemplified by cardiac disease now being a leading cause of maternal death in the UK, and most women who die having acquired rather than congenital cardiac disease.

Aortic dissection can develop spontaneously or in the context of a bicuspid aortic valve or collagen vascular disorder (e.g. Marfan syndrome, vascular Ehlers–Danlos syndrome, Loeys-Dietz syndrome). Acute coronary syndrome can develop secondary to atherosclerosis, thrombosis or spontaneous coronary artery dissection. Physicians must therefore consider ischaemia when assessing chest pain in pregnancy, even in the absence of established traditional risk factors.

Troponin, ECG and CXR should be requested in all those in whom an acute aortic or coronary syndrome is suspected. Possible ECG changes in pregnancy include premature atrial and ventricular complexes, mild left axis deviation and T wave inversion in leads III and aVF, occasionally accompanied by non-specific ST depression or small Q waves. Over 10% of healthy women record heart rates >100 beats per minute from 18 weeks of pregnancy and up to 40% have a small pericardial effusion on echocardiography.

Palpitations are common in pregnancy; exacerbating factors including anaemia and thyrotoxicosis should be considered and corrected if present. Women should be evaluated for red flags (e.g. syncope, chest pain, family history of sudden cardiac death), and rarer differential diagnoses including PE and phaeochromocytoma considered. An ECG and/or Holter monitor, ideally when symptomatic, is essential in identifying the underlying rhythm and determining the need for specialist cardiology interventions.

Supraventricular tachycardias are the most common pathological rhythms in pregnancy, and all aspects of Resuscitation Council UK guidelines can be safely used if required, including adenosine, β-adrenoceptor blockers, verapamil and DC cardioversion.

Seizures and headache

MBRRACE-UK has highlighted a doubling in maternal deaths secondary to sudden unexplained death in epilepsy in recent years. Nocturnal seizures or an increasing seizure frequency are red flags warranting an urgent neurology review.

Subtherapeutic antiseizure medication (antiepileptic drug (AED)) concentrations can occur secondary to poor adherence, sudden discontinuation of teratogenic AEDs, or pregnancy-induced reductions in AED concentration, as is recognized for lamotrigine. Measuring AED concentrations can be a useful adjunct, but AED dose escalation should be guided first and foremost by the clinical assessment. If a woman has already presented with seizures without another correctable explanation, AEDs should be up-titrated and timely follow-up arranged while the results are pending.

Eclampsia, which can be accompanied by posterior reversible encephalopathy syndrome or intracerebral haemorrhage, is often an early consideration in a pregnant woman with seizures. Assessment should, however, consider the broad differential diagnosis including metabolic (e.g. hyponatraemia, hypoglycaemia), thrombotic (e.g. cerebral venous sinus thrombosis) and vascular (e.g. ischaemic or haemorrhagic stroke, subarachnoid haemorrhage) triggers.

Many intracerebral events can cause hypertension (blood pressure ≥140/90 mmHg) but seizures can also cause hypotension. Consequently, normal/low blood pressure at the time of seizure does not exclude eclampsia, and up to 40% of women with eclampsia do not have a record of hypertension and proteinuria before the seizure.

Key investigations include fingerprick glucose, blood tests (including for electrolytes), urine dipsticks for proteinuria, and ECG to exclude cardiac arrythmia. Supporting evidence of placental insufficiency in keeping with eclampsia can be obtained from a bedside fetal US for fetal growth restriction, and blood testing of the sFlt-1:PlGF ratio (soluble fms-like tyrosine kinase 1:placental growth factor) or standalone PlGF test according to local availability.

Stabilization, intravenous magnesium sulphate, control of hypertension and delivery are the treatment for eclampsia, while other causes may require AEDs and further tailored testing including intracranial imaging.

Peripartum seizures should prompt a consideration of hyponatraemia. Oxytocin concentrations surge during labour, and its synthetic forms can be used to augment contractions. Oxytocin has an antidiuretic effect, and when combined with the dilutional state of pregnancy (pregnancy normal range of sodium 130–140 mmol/litre, blood osmolarity 280 Osm/litre) and fluids taken orally or administered intravenously during labour, significant hypotonic hyponatraemia can develop.

Clinically, this can manifest as neonatal seizures, or maternal disorientation, nausea, headache and seizure. Review the medications and fluid balance charts, and consider a further evaluation of hyponatraemia including thyroid function tests, early morning cortisol and paired urine and serum osmolarities.

Headache in pregnancy has an overlapping differential diagnosis with that of seizures, including pre-eclampsia and cerebral venous sinus thrombosis. Other considerations include common conditions such as migraine and medication overuse headache, specific entities such as post-dural puncture headache after neuraxial anaesthesia, and rarities such as reversible cerebral vasoconstriction syndrome and arterial dissection. As outlined in Table 2, CT of the head, including CT venogram, is safe in pregnancy.

Magnetic resonance imaging (MRI) is also a safe modality in all three trimesters. However, gadolinium contrast has been rarely associated with stillbirth and infant rheumatic and inflammatory diseases; as such, it should be reserved for select cases, after MDT discussion and appropriate maternal counselling.

Acute kidney injury (AKI)

An increase in glomerular filtration rate means that a creatinine concentration ≥77 micromol/litre is abnormal at any stage in pregnancy.[2] As a creatinine concentration of 77–90 micromol/litre falls within the normal, non-pregnant adult range, this is not flagged by most electronic result systems. Clinicians must therefore closely examine trends and appreciate that if a woman's baseline creatinine in pregnancy is 30–40 micromol/litre, a concentration of 80 micromol/litre could indicate a significant AKI.

AKI should be approached similarly to that in non-pregnant adults, exploring the pre-renal, intra-renal, and post-renal differential diagnoses, and considering life-threatening consequences that could require urgent temporizing measures and renal replacement therapy. Renal tract US and Doppler scanning can help identify structural causes of AKI; however, it can demonstrate hydronephrosis resulting from ureteric compression by the gravid uterus in almost 30% of asymptomatic pregnant women.

AKI around the time of delivery is often multifactorial as blood loss combines with other renal insults including pre-eclampsia, nephrotoxic medications (e.g. non-steroidal anti-inflammatory drugs) and/or sepsis. The possibility of specific intra-renal pathologies must, however, be remembered; this should include AKI as an end-organ manifestation of a thrombotic microangiopathy (e.g. thrombotic thrombocytopenic purpura, atypical haemolytic uraemic syndrome, catastrophic antiphospholipid antibody syndrome) as pregnancy and delivery can act as triggering events.

Sepsis and fever

The immunological adaptations to pregnancy result in a vulnerability to more severe infection with a variety of microorganisms including intracellular (e.g. *Listeria*) and viral infections (e.g. influenza, COVID-19, hepatitis E). Structural changes also predispose to infection in particular anatomical sites, including urinary tract infections, mastitis, endometritis and surgical site infections.

Physicians may be called to assess women with persistent and/or unexplained fever. Differential diagnoses include occult infection (e.g. endocarditis), non-bacterial infection (e.g. tuberculosis, malaria, herpes simplex virus), toxin-mediated infections (e.g. group A *Streptococcus*), resistant organisms and non-infective pathologies including autoimmunity and malignancy. Specialist advice from rheumatology, haematology and infectious disease clinicians should therefore be sought early if the patient is deteriorating despite broad-spectrum antimicrobials.

If cytopenias or 'inappropriately normal' cell counts are identified in this setting, hyperinflammation should also be considered. Haemophagocytic lymphohistiocytosis (HLH) can be screened for using the 3 Fs of Fever, Falling cell counts and hyperFerritinaemia as described in the 2023 HLH consensus guideline.[3] If present, more specific tests should be requested including fibrinogen, triglycerides (triacylglycerols), cross-sectional imaging and bone marrow biopsy. A probability of HLH can be assigned using the *H*-score; the case can be referred to a local or national HLH MDT, and early institution of immunosuppression (e.g. anakinra, corticosteroids) is recommended.

Table 1

Prescribing resources for pregnancy

1. Best Use of Medicines in pregnancy (BUMPS) (medicinesinpregnancy.org)
2. United Kingdom Teratology Information Service (UKTIS): Evidence-based safety information about medication, vaccine, chemical and radiological exposures in pregnancy (https://uktis.org). UKTIS monographs are also available via the TOXBASE app

Prescribing resources for lactation

1. The Breastfeeding Network (BfN) (www.breastfeedingnetwork.org.uk)
2. United Kingdom Drugs in Lactation Advisory Service (UKDILAS): Breastfeeding Medicines Advice service –Specialist Pharmacy Service
3. Drugs and Lactation Database (LactMed): NCBI Bookshelf (nih.gov). LactMed monographs are also available within the LactRx app, by MotherToBaby

Table 2 Radiological imaging in pregnancy

	Typical fetal dose (mGy)	Comments
Radiation in context		
10 days of natural background radiation	0.001-0.01	
Natural background radiation dose during pregnancy	1	
Dose above which there is a concern for serious *in utero* harm[a]	100	
Imaging modality		
Any MRI Any MRI with contrast	0	MRI is safe in all three trimesters and during lactation MRI contrast should be avoided in pregnancy, but is safe in lactation
Extremity X-ray CXR CT of the head	Very low dose 0.001–0.01	
CTPA[b,c]	Low dose 0.01–0.1	CT contrast is safe in all three trimesters and during lactation CTPA maternal breast dose: 10–70 mGy
V/Q scan[b,c] Pelvic X-ray Hip X-ray	Low to moderate dose 0.1–1	V/Q maternal breast dose: 0.22–0.28 mGy
CT of the abdomen and pelvis PET CT whole body	Moderate dose 10–50	

[a] Dependent on dose and gestation of fetal exposure; includes risks of fetal death, malformation, growth restriction or abnormal brain development.[5]

[b] Background childhood cancer risk of 1:500; additional risk of childhood cancer 1:280,000 for V/Q scan and 1:1,000,000 for CTPA.[5]

[c] Background risk of breast cancer in the next 10 years in a 30-year-old woman is 1:200; additional risk of breast cancer 1:7000 for CTPA.

PET, positron emission tomography.

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