# Underlying medical conditions

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**Abstract**

More than half of maternal deaths in the UK are due to pre-existing medical conditions, and medical emergencies often mimic acute obstetric conditions. An acute flare of systemic lupus erythaematosus, a thyroid storm or a phaeochromocytoma have many of the signs and symptoms of imminent eclampsia. Similarly, severe postpartum haemorrhage can result in diagnostic difficulties of medical conditions such as Addison's syndrome or acute renal failure. An acute collapse can be due to a pulmonary embolus, myocardial infarction, tachyarrhythmia or myasthenia gravis. These conditions are rare; however, unless they are considered in a differential diagnosis, they will not be diagnosed, to the detriment of the woman and her infant. This chapter deals with acute medical conditions occurring uncommonly in pregnant women. An obstetrician should know the initial steps to take in the emergency management of these cases.

**Key words:** pre-existing medical conditions in pregnancy; emergency care

Pre-existing medical conditions play an important role in maternal mortality, contributing to 5.6% of maternal deaths in South Africa (a developing country[1]). As the tide of readily preventable maternal deaths recedes, the importance of pre-existing medical conditions increases, such that more than half of maternal deaths in the UK are due to pre-existing medical conditions.[2] In South Africa, 25% of maternal deaths due to pre-existing medical conditions had a significant delay in diagnosis, and a further 25% had delayed referral or an incorrect diagnosis.[1] Quoting from the latest Saving Mothers' Lives report, 'the assessors were struck by the number of health care professionals who appeared to fail to be able to identify and manage common medical conditions or potential emergencies outside their immediate area of expertise'.[2] Forty percent of indirect maternal deaths had substandard care, of which 69% had major substandard care (substandard care that contributed significantly to the death of the mother). In many, but not all, cases, correct treatment may have altered the outcome.[2] A diagnosis can only be made if the condition is considered; in other words, 'if you do not think of it, you cannot diagnose it'. This was often the case with avoidable maternal deaths due to pre-existing medical conditions.

This chapter will evaluate the management of medical emergencies that can occur in the different categories of underlying medical conditions. Few randomized controlled trials or meta-analyses are available to guide management in these conditions because of their acute nature and rarity. The best-available evidence will be used to guide management, but evidence is often anecdotal.

## ENDOCRINE DISORDERS

### Diabetes mellitus emergencies

Diabetes mellitus is fast becoming a new medical pandemic and the associated problems were highlighted at the 4th International Symposium on Diabetes and Pregnancy. Data from both the Netherlands and England illustrated the increase in prevalence. According to the Confidential Enquiry into Maternal and Child Health report, 50% of diabetic pregnant women experienced hypoglycaemia and 23% required treatment.[3] In a study by Nielsen et al, 45% of patients experienced hypoglycaemia, with most incidents occurring in the first trimester and the fewest incidents occurring in the third trimester.[4] In another study, pregnant diabetic women accounted for 2.2% of all admissions to adult intensive care units (ICUs).[5] Nielsen et al also found that a history of hypoglycaemia in the preceding year and impaired awareness or unawareness were independent risk factors for severe hypoglycaemia.[4] Hypoglycaemia is an important differential diagnosis in an unconscious pregnant woman, and rapid diagnosis and treatment of hypoglycaemia is an essential skill for obstetricians. Estimating the blood glucose level using Dextrostix at the woman's bedside confirms the diagnosis.

*Management of hypoglycaemia*

Common causes of hypoglycaemia in pregnancy are as follows.[6 and 7]

• Drug induced: this can be due to insulin and often results from strict glycaemic control. Drug-induced hypoglycaemia may also be caused by sulphonylureas, β-blockers and quinine used in the treatment of malaria.
• Organ failure: gluconeogenesis may be affected in pregnant women with HELLP (haemolysis–elevated liver enzymes–low platelets) syndrome, resulting in hypoglycaemia.
• Certain neoplasms: insulinoma and certain non-islet-cell tumours such as adrenocortical tumours and carcinoid tumours.
• Anti-insulin and anti-insulin receptor antibodies present in some auto-immune diseases may cause hypoglycaemia.

The management of hypoglycaemia includes the following[8], depending on level of consciousness and ability to swallow. If awake and orientated, the patient should be treated with two to six dextrose tablets followed by long-acting carbohydrates. If not well orientated and intravenous access is not possible, a 30% glucose gel can be applied to the oral mucosa. If the oral route is unsafe, a 50-mL intravenous bolus of 50% dextrose or glucagon 1 mg by intramuscular injection is administered and both should be followed with a 5% dextrose infusion. The underlying cause will then need to be addressed and response to treatment monitored.

*Management of diabetic ketoacidosis*

Diabetic ketoacidosis has the same effects in both pregnant and non-pregnant patients: dehydration, hypotension and acidosis that can lead to multi-organ dysfunction[9] and possibly death. It is another important aetiology to consider in an unconscious pregnant woman. The fetus may also be affected by acidosis and hypoxia, and Doppler studies have shown redistribution of fetal blood flow during maternal ketoacidosis.[9] Table 1 summarizes the symptoms, signs and laboratory findings of ketoacidosis.[9]

The management of ketoacidosis consists of three aspects: fluid resuscitation; insulin therapy; and management of electrolytes.[9]

**Table 1. Symptoms, signs and laboratory findings in diabetic ketoacidosis.**

| Symptoms | Signs | Laboratory findings |
|---|---|---|
| Malaise | Hypotension | Hyperglycaemia >13 mmol/L |
| Dehydration | Tachypnoea | Acidosis: arterial pH < 7.3 |
| Polydipsia | Tachycardia | Low bicarbonate: <15 mEq/L |
| Polyuria | Kussmaul respiration | Anion gap >12 mEq/L |
| Nausea | Drowsiness | Elevated base deficit >4 mEq/L |
| Vomiting | Mental status change | Ketonaemia > 1:2 dilution |
| Abdominal pain | Lethargy | Urinary ketones > +1 |
| Fruity breath odour | Coma | Venous pH 0.003 below arterial pH |
| Ileus | Shock | |

*Fluid resuscitation*[9, 10 & 11]

To replace fluid, use the formula of 100 mL/kg body weight. Administer 1 L of 0.9% normal saline over the first hour and replace 75% of the fluid deficit over the first 24 h by giving 250–500 mL/h of 0.45% saline over the next 12 h. If the glucose is <14 mmol/L, change to 5% dextrose/0.45% saline and continue fluids for 24–48 h to correct the deficit.

*Insulin therapy*[9, 10 & 11]

Prepare a short-acting insulin and normal saline solution (1 U insulin/10 mL fluid). It is important to flush the tubing with the solution before the infusion is started. Start immediately with a bolus of 0.1 U/kg intravenously and follow up with 0.1 U/kg/h. Check serum glucose, arterial blood gas and ketones every 2 h. Increase the insulin infusion if serum glucose does not decrease by at least 5 mmol/h. When the serum glucose drops below 14 mmol/L, decrease the infusion rate by half and adjust the insulin infusion based on serum glucose every 2 h according to Table 2.

**Table 2. A two-hourly sliding scale for managing diabetic ketoacidosis**.[10]

| Capillary glucose | Adjust intravenous Actrapid or Humulin R |
|---|---|
| <5.6 mmol/L | Reduce by 10 mL/h and give 25 mL of 50% dextrose |
| 5.6–8.9 mmol/L | Reduce by 10 mL/h |
| 9–12.2 mmol/L | No change |
| 12.3–15.6 mmol/L | Increase by 10 mL/h |
| >15.6 mmol/L | Increase by 10 mL/h and give an 8 U intravenous bolus of Actrapid or Humulin R |

*Management of electrolytes*[9, 10 &11]

If pH is between 6.9 and 7.0, add $NaHCO_3$ 50 mmol/L to 200 mL of 0.45% saline and give over 1 h. If pH is <6.9, add $NaHCO_3$ 100 mmol/L to 400 mL of 0.45% saline and give over 1 h and repeat two-

hourly until pH is >7.0. Potassium depletion is common and needs to be monitored two-hourly if the level is >5 mmol/l. If potassium is between 3.0 and 5.0 mmol/L, add KCl 20 mmol to each litre of fluid, attempting to keep potassium between 4 and 5 mmol/L. If potassium is <3 mmol/L, add KCl 40 mmol to the initial fluid and delay insulin therapy until potassium is >3 mmol/L.

**Acute adrenal insufficiency**

Adrenal insufficiency is rare in pregnancy and can cause a diagnostic dilemma. The clinical picture includes the following.[12 and 13]

• sudden weakness;
• altered consciousness that can cover the spectrum from delirium to coma;
• hypotension;
• bradycardia;
• fatigue;
• hypothermia or cold intolerance;
• hypoglycaemia;
• hyponatraemia;
• slow respiration;
• malaise and sometime nausea and vomiting; and
• inability to lactate.

The condition should be suspected in the following clinical scenarios[13, 14 & 15]:

• severe postpartum haemorrhage that leads to pituitary necrosis (Sheehan syndrome);
• a patient with a suppressed axis due to prolonged corticosteroid use;• other causes: surgical removal, tuberculomas and encephalitis; and
• lymphocytic hypophysitis.

*Management*

Once the diagnosis is suspected, it is important to establish the cause and determine the adrenal hormones affected, since some of the deficiencies can be life threatening.[12] An early morning cortisol blood sample of <100 nmol/L is diagnostic of adrenal insufficiency, whereas a cortisol value of >500 nmol/l is normal.[14] The cortisol level in a pregnant woman is about three to five times that of a non-pregnant female.[12]

Management consists of replacing the affected hormones[13]; for example, thyrotropin deficiency needs l-thyroxin. In women with severe pan-hypopituitary crisis, it is important to give intravenous hydrocortisone initially with a high dosage of 100–150 mg hydrocortisone three times daily. Intravenous hydrocortisone is also indicated to cover surgery. Intravenous hydrocortisone is gradually tapered after surgery until oral treatment of 5 mg hydrocortisone twice daily can be started.[12 and 14]

**Phaeochromocytoma**

The combination of hypertension and the classical triad of paroxysmal headache, sweating and palpitations has a sensitivity of 91% and a specificity of 94% for diagnosing phaeochromocytoma.[16] The condition can mimic imminent eclampsia. Women can also present with tachycardia, seizure disorders, anxiety attacks, chest pain, dyspnoea, nausea, vomiting and flushing, and an abnormal glucose tolerance test.[17] Undiagnosed cases have high maternal (4–17%) and fetal (28%) mortality rates.[17] The diagnosis is based on increased levels of catecholamines and the metabolites vanillylmandelic acid, metanephrine and normetanephrine in a 24-h urine collection.[16 and 17] Patient management consists of α-blockade with either prazosin or phenoxybenzamine at a dose of 1 mg/kg infused over a minimum of 2 h, followed by β-blockade for tachycardia.[8, 16, 17 and 18] Prazosin is a Class C drug and can be used in pregnancy.[19] It appears from reported cases that labetolol is safe in the management of phaeochromocytoma.[18] The aim of therapy is to create a relative circulating volume expansion with mild postural hypotension.[16 and 17] Surgery during the second trimester of pregnancy is possible if early and correct diagnosis is made.

### Thyroid storm

A thyroid storm is a life-threatening event that arises in patients with underlying thyroid disease, and can be fatal in 20–50% of untreated cases. It is usually the result of either undertreatment or infection[20], but it may be associated with labour.[21] It can mimic imminent eclampsia. The diagnosis is made on clinical grounds supported by laboratory findings of hyperthyroidism.

The clinical presentation of a patient with thyroid storm includes the following[20, 21 and 22]:

• excessive sweating;
• severe pyrexia;

• cardiac failure;
• tachycardia;
• atrial fibrillation;
• tachyarrhythmias;
• hypertension;
• altered mental status (from agitation to coma);
• vomiting;
• diarrhoea;
• hyperglycaemia;
• acute abdominal pain; and
• hypovolaemia.

*Management*[20, 22 and 23]

• Propylthiouracil: initial dose of 600–1000 mg administered orally, via nasogastric tube or rectally, followed by 200–250 mg every 4 h.
• Iodine preparations should be given at least 1 h after propylthiouracil. This is given to prevent the release of formed hormone. The treatment is iopanoic acid 1 g every 8 h for the first 24 h then 500 mg twice daily, or four to eight drops of Lugol's iodine or saturated solution of potassium iodide every 6–8 h.
• Propanolol 60–80 mg orally every 4 h is given to block the peripheral effect of thyroxine. The effect starts 1 h after oral administration. Alternatively, propanolol 0.5–1 mg intravenously every 15 min for the first hour. The dosage for esmolol is a loading dose of 0.25–0.5 μg/kg followed by an infusion of 0.05–0.15 μg/kg/min. Careful cardiac monitoring is mandatory. Contra-indications for β-blocker therapy must be noted, and include a history of asthma, current bronchospasm or congestive cardiac failure. In these cases, one of the following alternatives can be considered: guanethidine 30–40 mg orally every 6 h or reserpine 2.5–5 mg intramuscularly every 4 h. The safety of both of these drugs during pregnancy has not been established.[24 and 25]
• High-dose corticosteroids: dexamethasone 4 mg intravenously every 6 h.[8]
• Supportive care includes intravenous fluid to correct dehydration and lower body temperature. Intravenous fluid should contain 5–10% dextrose to prevent hypoglycaemia. In cases of severe hyperthermia, extra measures should be taken to cool the patient, such as tepid sponging. Salicylates should be avoided in the management of these patients.
• The cause of the thyroid storm should be identified and treated.

## CARDIAC CONDITIONS

### Myocardial infarction

The incidence of myocardial infarction in pregnancy is between one in 10 000 and one in 30 000, and is most common in multigravid women over 33 years of age.[26, 27, 28 and 29] Other associated risk factors for the development of myocardial infarction in pregnancy are: cigarette smoking; hypertension; diabetes; obesity; hypercholesterolaemia; antiphospholipid syndrome; proteins S and C deficiency; antithrombin III deficiency; hyperhomocysteinaemia; and increased use of combined oral contraceptive

pills.[26, 27, 28, 29 and 30] The normal physiological changes in the cardiovascular system in pregnancy predispose women with risk factors to myocardial infarction. This condition is associated with a maternal mortality rate of 50% and a fetal mortality rate of 17% in surviving mothers.[30]

The differential diagnosis of chest pain in pregnancy includes the following conditions[26 and 28]:

• pulmonary embolism;
• hypertensive crisis;
• pericarditis;
• aortic dissection;
• sickle cell crisis; and
• acute coronary syndrome.

*Diagnosis of acute myocardial infarction*

• Electrocardiogram with ST segment elevation, usually in leads V1–V4, and aVL because most infarctions are anterior or antero-lateral.[28]
• Full blood count and platelets to help exclude other possible causes.[26]
• Cardiac enzymes: cardiac troponin I and troponin T. These enzymes are not increased during normal pregnancy, delivery, prolonged labour, caesarean section or obstetric anaesthesia.[30]
• Transthoracic echocardiography is safe, easily obtainable and allows visualization of decreased wall motion due to ischaemia.
• Cardiac catheterization can be done safely if there is proper shielding of the fetus. This helps with the diagnosis and location of the infarct.[26]

*Treatment*

Once a diagnosis of acute myocardial infarction has been made, resuscitation is the same as for the non-pregnant patient. Several case reports have confirmed that percutaneous coronary intervention is a treatment option.[26, 27, 28, 29, 30 and 31] Fetal exposure to radiation is calculated at approximately 80 Gy/cm$^2$, and the advantage of this procedure over conventional thrombolytic agents is that it is associated with less risk of bleeding for the patient.[27, 28 and 30] Coronary bypass is associated with an increased fetal loss rate of 19%, but is indicated under the following circumstances[30] and has been performed in pregnancy[28]:

• left main dissection of the aorta;
• multivessel disease; and
• failure of interventional treatment.

Thrombolytic therapy is regarded as first-line treatment in non-pregnant individuals but is relatively contra-indicated in pregnancy because of the theoretical risk of bleeding. Streptokinase and urokinase have been used in pregnancy and cases have been reported with successful outcomes.[26, 28 and 30] Neither streptokinase nor urokinase cross the placenta, so the risk of bleeding is only maternal.[30]

*Anticoagulation therapy*[28, 30 and 32]

Heparin does not cross the placenta so can be used safely in pregnancy. Low-molecular-weight heparin is a safe alternative to unfractionated heparin, but patients should be changed over to unfractionated heparin close to delivery to avoid peripartum haemorrhage because of the shorter half-life. Aspirin 150–300 mg can be used in the acute setting[32], but care needs to be taken in the third trimester as the drug is associated with premature closure of the ductus arteriosus.[29] After the acute setting, aspirin 75–150 mg is safe in pregnancy.[26 and 32] The safety of clopidrogel has not been proven in pregnancy, and it is classified by the US Food and Drug Administration as a Category B drug.[30] However, several case reports have reported successful outcomes of pregnancies where it was used.[27, 28 and 31] β-blockers and nitrates can both be used in the management of these patients.[25, 29 and 31] The administration of nitrates in a pregnant patient needs to be monitored carefully as they may cause hypotension and tachycardia.[30] β1-selective blockers should be used in pregnant women, but it is important to remember that β-

blockers are associated with intra-uterine growth restriction and hypoglycaemia.[30] Angiotensin-converting-enzyme inhibitors and statins should both be avoided in pregnancy because of proven adverse effects on the fetus.[32, 33 and 34] If possible, delivery should be postponed until 2 weeks after the infarct to reduce the risk for both mother and fetus.[35] The route of delivery is debatable. Caesarean section has the advantage of a planned labour, but carries the risks associated with anaesthesia and possible bleeding. Vaginal delivery, on the other hand, is associated with a prolonged period of increased cardiac output but avoids surgical intervention. Studies undertaken to date have failed to show any difference in outcome between the two methods.[30] If vaginal delivery is planned, the patient should be monitored intensively with repeated cardiac enzyme assays.[30]

## RESPIRATORY CONDITIONS

### Asthma

Severe asthma accounted for between 0.1% and 2.9% of ICU admissions in Mumbai and Texas, respectively.[5] A difficulty with the presentation of asthma in pregnancy is that 70% of normal pregnant women experience a degree of dyspnoea due to the physiological changes of pregnancy.[5] A recent study in the UK found that asthma was associated with an increased risk of preterm delivery[36], and was probably associated with an increase in abruptio placentae.[35 and 37] Asthma in pregnancy is further associated with complications such as hypoxaemia, fetal death, pregnancy-induced hypertension, eclampsia, gestational diabetes, intra-uterine growth restriction, preterm delivery and caesarean section.[38, 39 and 40]

The presentation of acute asthma includes the following symptoms and signs[41]:

- cough;
- breathlessness;
- wheezing;
- tightness over the chest;
- increased respiratory rate;
- use of accessory respiratory muscles;
- inability to speak;
- tachycardia > 110 beats/min;
- respiratory rate > 25 breaths/min; and
- peak expiratory flow rate = 33–50% of the patient's best or predicted.

Patients who present with severe life-threatening asthma (SLTA) have usually had an incident in the previous year[42], and it is therefore important to ask in the history about previous episodes. The features of SLTA are[41]:

- peak expiratory flow rate < 35% of predicted;
- arterial oxygen saturation of < 8 kPa;
- normal or raised $pCO_2$ > 4.6 kPa;
- silent chest;
- cyanosis;
- feeble respiratory effort;
- bradycardia;
- arrhythmia;
- hypotension;
- exhaustion;
- confusion; and

- coma.

*Management*

The management of an acute asthmatic attack is the same as in the non-pregnant individual.[39, 43 and 44] None of the standard management steps should be avoided, since poorly managed asthma can have

serious consequences for both mother and child. The implementation of asthma care maps can help in the emergency department to increase the prescription of bronchodilator therapy.[44] This can be especially useful in the labour ward where SLTA is a rare occurrence.

*Management of SLTA*[41, 44 and 45]

• Oxygen especially high flow, start at 8 L/min.
• Intravenous fluid if patient is dehydrated; otherwise, a line for intravenous access.
• Patients should be managed in a monitored setting and with fetal assessment.
• β2 agonist, e.g. salbutamol 5 mg via nebulization for 1 h.
• Ipratropiumbromide 0.5 mg via nebulization for 1 h.
• Intravenous corticosteroids: hydrocortisone 100 mg or solumedrol 125 mg intravenously.
• Magnesium sulphate 2 g infusion over 20 min, intravenously if peak flow rate < 30%.

• Intravenous aminophylline (safe in pregnancy) or intravenous salbutamol.[46]
• Intravenous salbutamol 250 μg. Dilute 0.25 mL of 1 mg/mL to 1 mL and administer over 2 min, thereafter 5 μg/min and increase every 15 min by 5 μg/min.
• If $PaCO_2$ rises or fetal distress is noted, transfer to the ICU.[43]
• A progressive increase in maternal $PCO_2$ or $PaCO_2$ > 40–45 mmHg associated with maternal exhaustion are indications for intubation and ventilation.[43]
• To prevent dynamic hyperinflation with subsequent volu- and barotraumas, the following steps should be taken[44]:

○ small tidal volumes 6–8 mL/kg;
○ high peak inspiratory flow rates 100–120 L/min;
○ low respiratory rates 8–12 breaths/min; and
○ the use of square wave inspiratory flow pattern.

• Admission to ICU with a viable preterm infant requires corticosteroids for lung maturation.
• In severe non-responsive cases, caesarean section delivery should be considered in the ICU.[44]
• Patients who develop acute asthma during labour should be managed as described above. In the case of caesarean section, care should be taken with the choice of anaesthetic drugs.

## NEUROLOGICAL DISORDERS

### Status epilepsy

Seizures due to epilepsy are the most common form of seizures in pregnancy, and affect 0.5% of childbearing women. The differential diagnoses of seizures in pregnancy include the following[47, 48, 49 and 50]:

• epilepsy;
• eclampsia;
• cerebral parenchymal disorders;
• metabolic abnormalities;
• intracranial haemorrhage;
• cerebral venous thrombosis;
• cerebral malaria and other infections such as toxoplasmosis;
• pseudostatus epilepticus;
• mitochondrial myopathy, encephalopathy, lactic acidosis, stroke-like syndrome;
• thrombotic thrombocytopenic purpura;
• drug and alcohol withdrawal; and
• postdural puncture.

Forty percent of women with epilepsy experience a convulsion during pregnancy[51], and in 30% of epileptic women, there is worsening of seizures during pregnancy.[47] and [48] The following are possible reasons[47 and 48]:

• increased psychological stress;
• women stop treatment out of fear of the effect on the fetus;
• oestrogen reducing the seizure threshold; and
• decreased serum levels of the drugs due to: increased plasma volume; reduced plasma protein binding; increased clearance by the liver and kidneys; and impaired absorption.

*Management*[52, 53 and 54]

Initial management is the same as for any other very ill patient. Care should be taken to maintain the airway, and the patient should be placed on the left lateral side to keep the airway open and prevent postural hypotension. Intravenous access should be established and arterial blood gas values obtained. Serum blood glucose should be checked and if hypoglycaemia is present, treatment is 50 mL of 50% dextrose and 100 mg of thiamine intravenously. An oral airway may be needed to protect the airway, and a nasogastric tube will help to empty the stomach contents. Other investigations include electrocardiogram, pulse oximetry, electrolytes and full blood count. If the maternal temperature rises >40 °C, the patient should be cooled. The patient should be well hydrated to protect the kidneys against renal failure caused by myoglobinuria. If myoglobinuria is present or the creatine kinase level is between 5000 and 10 000 U/L, forced saline diuresis and urinary alkalinization should be started.

Initial pharmacotherapy is as follows.[52 and 54]

• Lorazepam 0.1 mg/kg intravenously and repeated if the seizures continue for >10 min. Several studies have confirmed that lorazepam is superior to diazepam in the control of seizures. There are possible risks to the fetus with long-term use of lorazepam in the first and third trimesters, but use in the acute setting is probably justified.[55]
• Phenytoin 15–20 mg/kg intravenously should be administered after lorazepam. The advantages of phenytoin for the mother outweigh the fetal risk. Vitamin K should be given to the mother during labour.[56]

If the seizure continues, the following drugs can be used.[52], [53] and [54]

• Rocuronium 1 mg/kg for intubation. This should be used instead of succinylcholine.
• Thiopental 100–200 mg bolus over 20 s followed by 50 mg boluses every 2–3 min until the seizures stop. This is then followed with an infusion of 3–5 mg/kg/h for 24 h.
• Pentobarbital 10–20 mg/kg bolus followed by an infusion of 0.5–1 mg/kg/h, increased to 1–3 mg/kg/h if necessary. There are limited data on the use of pentobarbital in pregnancy, but it is probably safe with the advantages outweighing possible harm. Haemorrhagic disease is a possibility in the newborn.[57]
• Midazolam 0.2 mg/kg followed by 0.1–2.0 mg/kg/h infusion.
• Propofol 3–5 mg/kg loading dose followed by an infusion of 30–100 μg/kg/min. The dose is titrated against electroencephalogram (EEG) activity. After 12 h of seizure suppression, the dose is halved over the next 12 h and then gradually decreased over the second 12 h.
• All patients should receive continuous EEG monitoring.
• Once the patient's seizure has been controlled, fetal well-being should be monitored and delivery should be considered.
• After control, the patient's therapy should be tailored to their needs.

## AUTO-IMMUNE DISORDERS

### Systemic lupus erythaematosus

When managing a patient with systemic lupus erythaematosus (SLE) in pregnancy, the following considerations are important. The patients should be in remission (inactive disease for >6 months and prednisone dosage <10 mg/day) when they consider pregnancy. Preconception counselling is therefore most important.[58] and [*59] Management should be undertaken by a multidisciplinary team.[59]

*Effect of SLE on pregnancy*[58 and 59]

SLE leads to increased risk of the following conditions:

• spontaneous abortion;
• intra-uterine growth restriction;
• premature rupture of membranes;
• premature delivery;
• pre-eclampsia; and
• fetal death.

*Effect of pregnancy on SLE*

There is an increased risk of flare up during pregnancy.[58 and 59] It can be misdiagnosed as pre-eclampsia, and diagnosis may not be considered for a considerable period of time.

*Diagnosis of lupus flare up*[59, 60, 61, 62 and 63]

• A new or worsening photosensitivity or discoid or any other lupus-associated cutaneous rash.
• Acute sinovitis.
• Pericarditis.
• Pleuritis.
• Nasopharyngeal ulcers.
• Fever in the absence of infection.
• Thrombocytopenia usually $<100 \times 10^9$/L.
• Leucopenia $<4 \times 10^9$/L.
• Haemolytic anaemia.
• Worsening of proteinuria >2 g/24 h or a 30% increase above baseline.
• Recurrence of active urinary sediment.
• An increase of >0.25 in the Lupus Activity Index in Pregnancy Scale.

The diagnosis is made by decreased serum compliment, a rise in the anti-DNA antibody titre, and red cell or cellular casts in the urine. It is differentiated from severe pre-eclampsia because the serum compliment is normal in pre-eclampsia but the final diagnosis is by renal biopsy.[59]

*Treatment*

Once a flare up has been diagnosed, oral prednisolone should be started. The dose ranges between <0.5 mg/kg/day for moderate flare and >0.5 mg/kg/day for severe flare.[60 and 64] Women already on prednisolone need hydrocortisone 100 mg every 6 h.[64] Other drugs that should be added in cases of severe renal flare up include cyclophosphamide, azathioprine, mycophenolate mofetil and cyclosporine A.[59, 60 and 62] Cyclophosphamide is contra-indicated in pregnancy, but in life-threatening disease, it can be used in late pregnancy but should be avoided in the first trimester.[59 and 65] The associated embryopathy includes abnormalities of face, fingers and toes, as well as growth impairment.[65] There are limited data on the use of mycophenolate mofetil in pregnancy, and data suggest possible risk to the fetus. This should be weighed up against possible harm to the mother if treatment is withheld. It is classified as a Class C drug.[66] Azathioprine can be used in pregnancy if the maternal condition does not respond to steroids alone, but there are data suggesting possible fetal harm.[67] There are limited data on the use of cyclosporine A in pregnancy but it appears to be safe.[68] Patients experiencing a discoid rush can be treated with hydroxychloroquine (safe in pregnancy[69]) added to prednisolone.[59] In cases of severe flare up with deteriorating renal function despite treatment, termination of pregnancy should be considered before using drugs such as methotrexate that are contra-indicated in pregnancy.

**Myasthenic crisis**

This is an emergency precipitated by infection, inadequate treatment or initial high-dose steroids.[70] There is a rapid deterioration in the normal symptoms with changes in speech, dysphagia, inability to

swallow saliva and a cranial caudal progression of weakness.[70 and 71] Respiratory muscles weaken with subsequent respiratory failure.[70] It is important to monitor lung function with forced vital capacity a few times daily.[70] Plasmapheresis and intravenous immunoglobulin are the best treatment options.

*Diagnosis of myasthenia*[70 and 72]

• Tensilon test: administration of edrophonium chloride produces a short improvement in muscle strength.
• Electromyography.
• Acethylcholine receptor antibodies in 90% of patients and a thymoma in 10%.

*Management*

The following drugs are used in the chronic management of myasthenia gravis.[70, 71and 72]

• Pyridostigmine bromide is an anticholinesterase inhibitor. The initial dose is 30–60 mg every 4–8 h. Neonatal effects include respiratory distress and microcephaly if the dose exceeds 600 mg/day. It is regarded as safe in pregnancy. During labour, it is better to change to intramuscular injections.
• Prednisone is safe in pregnancy. Thirty percent of patients will experience deterioration of symptoms in the first 7–10 days. The starting dose is 10 mg/day, increasing slowly to 60 mg/day.
• Azathioprine should be avoided but it can be used if required.[67] The initial dose is 1 mg/kg/day, increasing slowly to a maximum of 2.5 mg/kg/day. It takes 3–6 months to show effect.
• Cyclosporine A is probably safe in pregnancy.[68]
• Mycophenolate mofetil should be avoided in pregnancy if possible.[66]
• Methotrexate should be avoided in pregnancy.
• Cyclophosphamide is used in refractory cases of myasthenia gravis. It should not be used in the first trimester and should only be considered in life-threatening cases.[65]

*Management of crisis*[70 and 71]

• Plasmapheresis is safe in pregnancy and is used for rapid control in crisis situations. The improvement is only effective for 4–10 weeks.
• Intravenous immunoglobulin is used for the same indications as plasmapheresis. The dose is 400 mg/kg/day for 5 days and is probably safe in pregnancy.

## SUMMARY

Some acute medical crises can mimic common obstetric emergencies. If these medical conditions are not considered, the diagnosis may be delayed critically, leading to disastrous consequences. The obstetrician must be able to diagnose and coordinate the management of these conditions with the relevant specialists.

***Hypoglycaemia***

• start early with 50 mL of 50% dextrose
• treat the underlying cause

***Diabetic ketoacidosis***

• adequate fluid management; fluid needs: 100 mL/kg body weight
• 1 L of 0.9% saline over 1 h
• replace 75% of estimated fluid loss over 24 h
• insulin initial bolus 0.1 U/kg
• maintenance 0.1 U/kg/h
• potassium replacement if potassium <5 mmol/L
• potassium before insulin if potassium <3 mmol/L

***Acute adrenal insufficiency***

• correct any electrolyte imbalances
• establish exactly which hormones are affected and replace
• treat with oral hydrocortisone
• need intravenous cortisone when delivering

***Phaeochromocytoma***

• circulating volume expansion
• α-blockade to control hypertension
• β-blockade to prevent arrhythmias
• carefully planned surgery – do not palpate tumour

***Thyroid storm***

• propylthiouracil to reduce thyroxine secretion
• iodine preparation to reduce thyroxine secretion
• β-blockers to control blood pressure, reduce pulse rate and prevent tachyarrhythmias
• dexamethasone
• supportive care

***Myocardial infarction***

• keep on the differential diagnosis list
• ABC of resuscitation
• percutaneous coronary intervention is an option
• streptokinase and urokinase can be used
• patients should be placed on low-dose aspirin
• β-blockers and nitrates can be used in management

***Asthma***

• oxygen
• nebulization with β2 stimulants
• intravenous corticosteroids
• in severe cases, intravenous aminophyline
• may need intubation and ventilation
• delivery may help with ventilation of the patient

***Status epilepsy***

• lorazepam followed by phenytoin
• rocuronium for intubation
• thiopental for prolonged seizures after intubation
• pentobarbital, midazolam or propofol are alternatives

***Systemic lupus erythaematosus***

• steroids are the first line of treatment for an SLE flare up
• for cutaneous problems, hydroxychloroquine can be used
• azathioprine can be used during pregnancy
• for severe renal flare up, more toxic drugs need to be considered
• the place of glucagon in the treatment of pregnant women
• cardiac drug safety in pregnancy
• management of SLE flare up in pregnancy
• safety of SLE drugs in pregnancy

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