Acute medical disorders in **pregnancy**

This Update explores the commonly occurring acute conditions associated with pregnancy and offers advice regarding their investigation and management.

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Conflicts: nothing to declare

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The Duchess of Cambridge, Kate Middleton, experienced hyperemesis gravidarum with both her pregnancies.

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Introduction

UP TO one in five pregnancies are complicated by some type of medical disorder.

For many women, this is a longstanding chronic medical condition, which may predate the pregnancy by months or years. The course of these medical problems may be altered during pregnancy, and they themselves may increase the risk of obstetric complications.

The management of pregnant women with chronic medical problems was covered in a previous Update (4 September 2015). This Update will focus on medical problems unique to pregnant women and which only arise for the first time during pregnancy.

By definition, women with a pregnancy-related medical problem will not have required medication periconceptually, thus risks regarding teratogenic effects are not a concern. Additionally, although disorders such as pre-eclampsia are very familiar, some pregnancy-specific conditions are quite uncommon and may only be appreciated by experienced clinicians.

Hyperemesis gravidarum

While all pregnant women are familiar with morning sickness, true hyperemesis gravidarum (HG) is far rarer. Although there is no standardised definition, most agree it implies protracted nausea and vomiting in early pregnancy resulting in fluid and electrolyte disturbance.

True HG affects less than 1% of pregnancies, and the widely accepted theory is that it is related to maternal serum hCG levels, which peak at 6-8 weeks before plateauing after 14 weeks.[1]

True HG always requires referral and admission to hospital, and many affected women bounce in and out of hospital several times in early pregnancy until the symptoms naturally begin to improve.

Abdominal pain is not generally a feature of HG and women with nausea and vomiting associated with abdominal pain or tenderness need investigation for other diagnoses.

WHAT INVESTIGATIONS ARE ADVISED IN WOMEN WITH HYPEREMESIS?

Hyperemesis gravidarum is a diagnosis of exclusion.

An early pregnancy ultrasound is mandatory to exclude conditions associated with high hCG levels – multiple pregnancies and (more importantly) hydatidiform mole. UEC typically reveals hypokalaemia, hyponatraemia and a metabolic alkalosis.

Up to half of women with HG have moderately raised liver transaminases, which are a result of the disorder rather than a cause. Serum Ca2+ helps to exclude rare cases of hyperparathyroidism.

As hCG has TSH-like activity, HG is commonly accompanied by a transient gestational hyperthyroid state, which is generally self-limiting and rarely requires antithyroid medication.

Urinalysis frequently shows ketonuria. Treatment should be directed at symptomatic improvement rather than eradication of urinary ketones.

MSU should be sent to exclude UTI, which can exacerbate underlying hyperemesis.

WHAT IS THE RECOMMENDED MANAGEMENT OF HYPEREMESIS?

There is no cure; treatment aims to improve nausea, maintain hydration and limit nutritional depletion.

Hydration is paramount, and women with dry mucous membranes who cannot tolerate oral fluids require admission to hospital for IV hydration.

Women with HG should avoid heavy, greasy foods and concentrate on bland foods which are more easily tolerated, such as dry crackers, toast and high-protein snacks.

HG in women with pre-pregnancy diabetes requires hospital admission and management by experienced endocrinologists and maternal-fetal medicine specialists.

WHAT COMPLICATIONS CAN ARISE IN WOMEN WITH HYPEREMESIS?

HG does not increase the risk of fetal congenital abnormalities.

Often HG is associated with maternal weight loss (indeed, some experts include 5% weight loss in the definition). A weight loss of ≥15% of pre-pregnancy weight is considered severe.

While not all studies agree, true HG does appear to increase rates of low birth weight/preterm labour.[2]

The dehydration associated with HG is a risk factor for DVT and women admitted to hospital require T.E.D. stockings and consideration of prophylactic LMWH.

Rarely, HG leads to thiamine deficiency, which can precipitate Wernicke's encephalopathy.

Severe HG can have a significant impact on psychosocial health, which must not be forgotten.

WHAT MEDICATIONS ARE APPROPRIATE IN TREATING HYPEREMESIS?

Many women find natural remedies – including ginger and chamomile – to be beneficial, although hard scientific evidence is lacking.

Vitamin B_6 (pyridoxine 25-50mg tds-qds) may improve nausea and can be safely used.

Oral iron preparations can exacerbate gastrointestinal upset and should be temporarily discontinued.

A variety of antiemetic agents can be safely prescribed in early pregnancy and may help minimise hospital admissions (Table 1).

PO/IV thiamine 100mg/day is recommended for hyperemesis to prevent Wernicke's disease.

Steroid treatment should be reserved for women with severe, treatment-refractory hyperemesis gravidarum.

TABLE 1. ANTIEMETICS

Antiemetic medication		Brand name	Dose (PO)
First line	Doxylamine	Restavit	12.5–25mg nocte
	Promethazine	Phenergan	10–25mg tds
Second line	Metoclopramide	Maxolon	10mg tds
	Prochlorperazine	Stemetil	10mg tds
	Cyclizine	–	50mg tds
Third line	Ondansetron	Zofran	4mg bd–tds

Pre-eclampsia and pregnancy-induced hypertension

Pre-eclampsia (previously pre-eclamptic toxaemia, PET) is the most well-known pregnancy-specific medical disorder, affecting 5% of pregnant women. PET is a multisystem disorder defined as new-onset hypertension (BP ≥ 140/90mmHg) occurring after 20 weeks' gestation, with evidence of end-organ dysfunction.[2]

PET is more common in first pregnancies, in twin/triplet pregnancies and in women with diabetes, renal disease or thrombophilia.

Additionally, women with pre-pregnancy hypertension may develop PET 'superimposed' on their chronic disease.

Pregnancy-induced hypertension (PIH) is a related hypertensive disorder which is distinguished from PET by the absence of end-organ dysfunction.

PIH (also called gestational hypertension) may evolve into pre-eclampsia as the pregnancy progresses and always resolves by three months postpartum.

WHAT 'END-ORGAN DYSFUNCTION' IS REQUIRED TO MAKE A PET DIAGNOSIS?

Significant proteinuria is defined as a spot urine protein:creatinine ratio (PCR) of ≥30mg/mmol in the absence of urinary tract infection.

Dipstick urinalysis is not sufficient to make a diagnosis.

Twenty-four-hour urine collections to quantify proteinuria are rarely needed in contemporary obstetrics.

Proteinuria is no longer a *sine qua non* for diagnosing PET; it is possible to meet a diagnosis of PET if there is hypertension without proteinuria but some other evidence of end-organ dysfunction.

Evidence of 'end-organ dysfunction' in a hypertensive pregnant woman include:

- Newly abnormal serum biochemistry (Table 2). Although serum uric acid is often increased in PET, it is not sufficient for making a new diagnosis.
- Evidence of fetal growth restriction on ultrasound scan.
- Pulmonary oedema.
- Typical clinical features suggestive of PET (see below).

WHAT ARE THE CLASSIC CLINICAL FEATURES OF PET? [3]

Mild pre-eclampsia is frequently asymptomatic and without physical signs.

Symptoms include headache, visual disturbance, nausea and vomiting, epigastric/RUQ pain.

Signs include hyperreflexia, clonus, epigastric/RUQ tenderness, severe peripheral oedema.

For newly hypertensive pregnant women with any of these features, blood and urine work-up (Table 2) and referral for hospital assessment are mandatory.

HOW IS PRE-ECLAMPSIA TREATED?

The only cure for PET is delivery of the baby and placenta.

For women with confirmed PET at ≥37 weeks, delivery is indicated, usually by induction of labour.

Women with PET who are preterm (<37 weeks) are usually managed expectantly with antihypertensive medication, regular blood testing and close fetal monitoring.

The oral anti-hypertensive agents of choice for women with PET are labetalol (100-400mg tds-qds) and methyldopa (250-750mg bd-tds).

Oral hydralazine 25-50mg tds may be added as a second line agent.

WHAT ARE THE POTENTIAL COMPLICATIONS FROM PET?

HELLP syndrome (syndrome of **H**aemolysis, **E**levated **L**iver enzymes and **L**ow **P**latelets): This is really a variant of severe PET rather than a complication. Women with HELLP syndrome have severe biochemical derangement and a risk of maternal liver rupture.

Eclampsia: A convulsive episode in women with PET is an obstetric emergency. IV magnesium sulphate (4g loading followed by 1g/h infusion) is the treatment of choice. About 40% of eclamptic seizures occur in the first 24 hours postpartum. In antenatal eclampsia, delivery of the baby is indicated following maternal stabilisation, irrespective of gestation (assuming the diagnosis is clear).

Haemorrhagic stroke: the risk increases when maternal systolic BP exceeds 160mmHg.

Placental abruption: maternal hypertension can cause sudden retroplacental bleeding which can be massive and life-threatening.

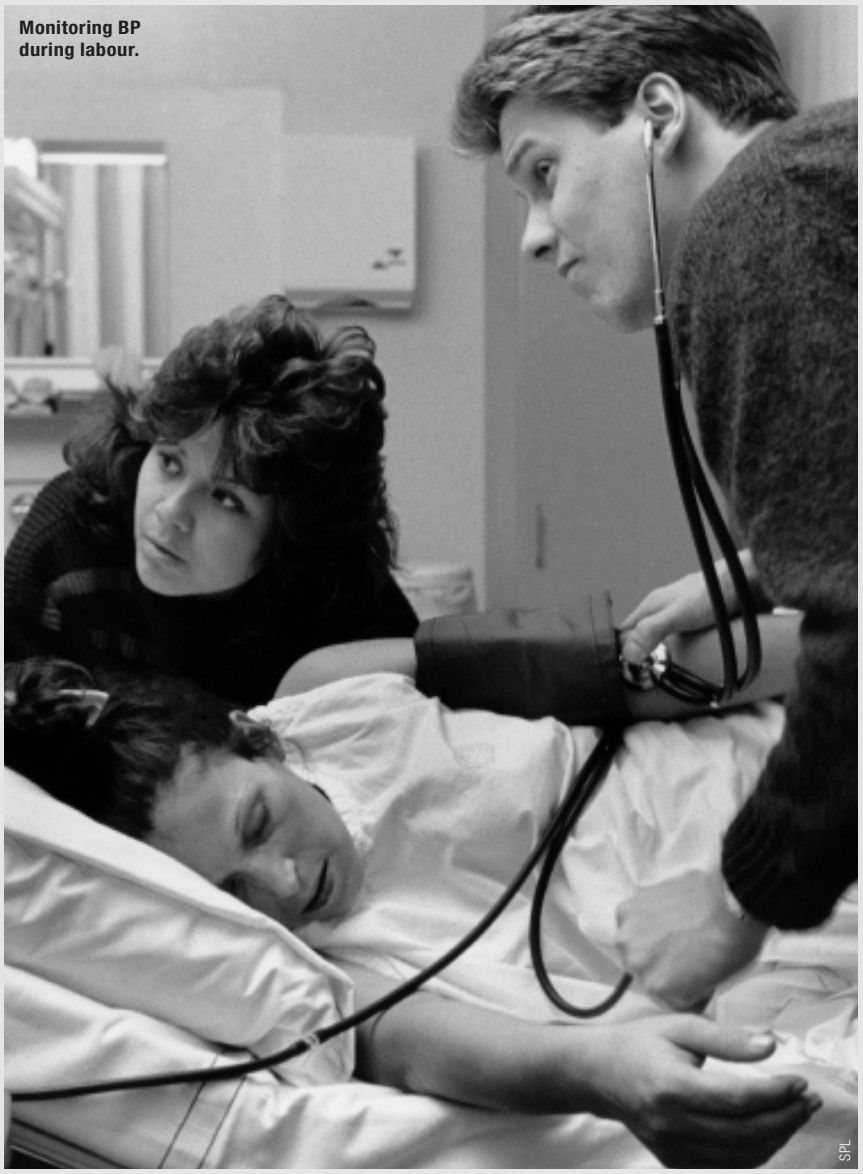
Stillbirth: growth-restricted babies have a significantly higher risk of in utero fetal demise.

DVT/PE: large degrees of proteinuria and peripheral oedema increase the risk of thromboembolism.

Long-term sequelae: a history of PET, particularly recurrent cases, is now known to be a significant risk factor for long-term atherosclerotic morbidity and renal disease.[4]

TABLE 2. ROUTINE TESTING IN WOMEN WITH SUSPECTED PET

Routine tests	Evidence of 'end-organ dysfunction' needed to meet a diagnosis of PET
	BP ≥ 140/90mmHg plus one of the following:
Full blood count	Platelet count < $100x10^9$/L
Urea, electrolytes, creatinine	Newly raised serum creatinine > 90umol/L
Liver function tests	Newly raised AST or ALT (≥70U/L)
Uric acid	Evidence of intravascular haemolysis: - Schistocytes on blood film - Serum LDH > 600mIU/L - Serum haptoglobin < 0.2g/L
+/- Haemolysis screen	
Urine for protein : creatinine ratio	Urine PCR ≥ 30mg/mmol without UTI


Monitoring BP during labour.
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Acute fatty liver of pregnancy

WHAT IS ACUTE FATTY LIVER OF PREGNANCY?

Acute fatty liver of pregnancy (AFLP) is a very rare but serious cause of abnormal liver function during pregnancy.

Even experienced obstetricians in busy units will only see a handful of AFLP cases during their careers.

The classic presentation is of new onset nausea and vomiting in late pregnancy; indeed AFLP should be considered in any woman presenting with nausea and vomiting after 30 weeks.

It is a very serious condition, associated with fetal and maternal mortality if not diagnosed promptly.

Transaminases are raised and there may be hypoglycaemia and/or DIC.

Occasionally, AFLP may co-exist with pre-eclampsia, and distinguishing AFLP from HELLP syndrome can be challenging in such cases.

AFLP has the potential to cause fulminant hepatic failure and encephalopathy.

Management is delivery once the woman is stable. Post-op care is likely to require ICU admission and involvement from an experienced liver specialist.

Obstetric cholestasis

Obstetric cholestasis (OC), also called intrahepatic cholestasis of pregnancy, is a relatively common medical complication of pregnancy which is poorly understood by many clinicians.

It is a multifactorial disorder with a strong ethnic component. While the overall prevalence is approximately 0.5–1%, Indian, Pakistani, Chilean and Scandinavian women are at especially high risk.

OC is also more common in multifetal pregnancies, women with previous OC and carriers of hepatitis C.

The typical presentation of OC is a woman presenting with intense pruritus in the third trimester, with no skin rash and with abnormal liver function tests.

The itch classically affects the palms and soles but may present anywhere on the body. By definition, a skin rash is absent but there may be evidence of skin excoriation from prolonged scratching.

HOW IS OC DIAGNOSED?

The disorder is usually initially suspected in a pregnant woman complaining of pruritus, who is then found to have abnormal liver function testing.

OC is a diagnosis of exclusion. Other causes of abnormal liver function must be excluded, specifically:

- Viral hepatitis: hepatitis A, B and C serology
- Other viral infections: cytomegalovirus and Epstein-Barr serology
- Biliary obstruction such as gallstones: liver ultrasound
- Autoimmune liver conditions: anti-smooth muscle/anti-mitochondrial antibodies
- Pre-eclampsia/HELLP syndrome: check blood pressure, platelet count, proteinuria
- Dermatological causes of itching, such as eczema, should also be considered.

OC may cause elevated transaminases (usually ALT), elevated bile acids or both.

While alkaline phosphatase is secreted by the placenta and therefore not very helpful during routine pregnancy, it may be raised beyond pregnancy levels with OC.

Testing of serum bile acids (ideally fasting) is popular, as elevated bile acids may precede raised transaminases and bile acids are more specific to OC than transaminases (but not pathognomonic).

Although different laboratories use different normal ranges for bile acids in pregnancy, a threshold of >10umol/L is commonly used in the Australian setting.

Women with symptoms of OC but normal LFTs should have their bloods rechecked after 1–2 weeks.

WHAT PREGNANCY COMPLICATIONS ARE ASSOCIATED WITH OC?

The most feared complication is stillbirth. Internationally it is accepted that the rate of stillbirth is slightly higher in women with OC, possibly a fetal cardiotoxic effect from elevated maternal bile acids.

Stillbirth risk increases in women with severe OC (defined as serum bile acids ≥40umol/L).[5]

Rates of preterm delivery (both spontaneous and iatrogenic), meconium-stained amniotic fluid and emergency caesarean section are increased.

In OC with severely deranged liver function, a maternal coagulation screen should be checked; oral vitamin K (10mg daily) is only required for women with a prolonged prothrombin time (PT).

ANTENATAL MANAGEMENT OF WOMEN WITH OC

Women with confirmed OC should be cared for by a maternal-fetal medicine specialist.

Oral ursodeoxycholic acid (Ursofalk) is usually prescribed to reduce pruritus. It increases bile flow and reduces serum bile acid levels; a dose of 500mg bd is commonly used (maximum 15mg/kg/day).

Although the LFTs often improve, it is unclear whether the risk to the fetus is altered.

To relieve bothersome night-time itching, 25mg oral promethazine (nocte) may help.

While no strategy of antepartum surveillance has been shown to reduce the rate of stillbirth with OC, in practice fetal surveillance (using ultrasound, CTG or both) is usually increased.

Other treatments, such as dexamethasone, cholestyramine or rifampicin are occasionally used.

DELIVERY AND POSTPARTUM IN WOMEN WITH OC

Induction of labour is usually recommended at 37–38 weeks to minimise the risk of stillbirth.

Following OC, women should avoid the COCP, as the oestrogen component may cause cholestasis.

Resolution of symptoms and abnormal LFTs are required postnatally to confirm the diagnosis. Women with persistently abnormal LFTs six weeks postnatally require referral to a hepatology clinic.

OC has a very high (70–90%) risk of recurrence in future pregnancies.

Gestational diabetes mellitus

Gestational diabetes mellitus (GDM) is the most common pregnancy-specific medical disorder, affecting some 10–15% of women.

The Australian Carbohydrate Intolerance Study in Pregnant Women published in 2005 provided convincing evidence that identifying and treating GDM improved perinatal outcomes.[6] As a result of the landmark 2008 Hyperglycaemia and Adverse Pregnancy Outcome study, many countries have lowered the diagnostic thresholds for GDM, with a consequent rise in population prevalence.[7] The Australian Diabetes in Pregnancy Society has recently published consensus guidelines on the screening and diagnosis of GDM in Australia.[8]

HOW SHOULD WE SCREEN FOR GDM IN PREGNANCY?

Older screening models based solely on maternal risk factors missed a high proportion of GDM cases. Universal screening for GDM using a 75g oral glucose tolerance test (GTT) is now recommended for all pregnant women at 24-28 weeks' gestation (Table 3).[8]

The one-hour 50g glucose challenge test lacks sensitivity and should no longer be used.[8]

In addition, women with extra risk factors for GDM should also be screened early in pregnancy, ideally with a two-hour 75g GTT (or occasionally with HbA_{1c}). Typically, this is done at around 16 weeks. If this early screen is negative, routine 24-28 weeks screening is still required.

Women who benefit from early GDM screening in pregnancy include:

TABLE 3. DIAGNOSTIC CRITERIA FOR GDM

Two-hour 75g oral glucose tolerance test (GTT)
Fasting 5.1–6.9mmol/L
One hour ≥ 10.0mmol/L
Two hour 8.5–11.0mmol/L
Only one abnormal result is required to diagnose GDM
Fasting glucose level ≥ 7.0mmol/L or a two-hour glucose level ≥ 11.1mmol/L diagnoses overt diabetes

- Previous GDM or previous baby ≥ 500g without GDM
- Ethnicity: Asian, Indian, Aboriginal, Pacific Islander, Maori, Middle Eastern
- First degree relative with diabetes mellitus
- Maternal BMI > 30kg/m^2
- Maternal age ≥ 40 years old
- Polycystic ovarian syndrome (PCOS)
- Current use of steroid medication.

HOW SHOULD WOMEN WITH GDM BE MANAGED DURING PREGNANCY?

Women with confirmed GDM need multidisciplinary input from an endocrinologist, dietitian and maternal-fetal medicine specialist.

Targets for weight gain during pregnancy should be discussed (Table 4). Self blood glucose monitoring 4–5 times daily using a glucometer is essential. Although not universally agreed, reasonable glucose targets for women with GDM are:

- Fasting glucose ≤ 5.0mmol/L
- Two-hour postprandial glucose ≤ 6.7mmol/L.[8]

Women with two or more high glucose levels per week should be considered for further treatment.

Insulin is the mainstay of therapy for women with high glucose readings despite lifestyle and dietary modification.

Depending on the thresholds used, 10–20% of all women with GDM will require insulin to keep their glucose levels within target levels.

Although glyburide has been used in the management of GDM for many years in the US, it is not used in Australia.

There may be a role for oral metformin for a subset of women with GDM, but this is not yet standard clinical practice.

A repeat 75g GTT at six weeks postpartum is essential to exclude underlying type 2 diabetes.

WHAT OBSTETRIC RISKS ARE ASSOCIATED WITH GDM?

There is an increased risk of fetal macrosomia (birth weight ≥ 4.5kg), which results in higher risks of caesarean section, operative vaginal delivery, shoulder dystocia and brachial plexus injury.

The risk of stillbirth is increased, predominantly in women with poor glycaemic control. As such, women with GDM are typically delivered between 38 and 40 weeks.

Given the associations with fetal growth problems and stillbirth, additional third trimester growth ultrasounds are warranted in women with GDM.

Rates of pre-eclampsia are increased in women with GDM.

Neonatally, there are higher rates of hypoglycaemia, respiratory distress and jaundice.

TABLE 4. TARGET WEIGHT GAIN IN PREGNANCY

Pre-pregnancy weight	Target weight gain
Underweight (BMI < 18.5kg/m^2)	13–18kg total
Normal weight (BMI 18.5–24.9kg/m^2)	12–16kg total
Overweight (BMI 25–29.9kg/m^2)	7–12kg total
Obese (BMI 30–39.9kg/m^2)	5–7kg total
Morbidly obese (BMI ≥ 40kg/m^2)	≤5kg total

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Fetal macrosomia is a risk in gestational diabetes.

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Peripartum cardiomyopathy

Peripartum cardiomyopathy (PPCM) is a rare but serious disorder uniquely associated with pregnancy. Although most clinicians will rarely see a case of PPCM, it must be considered in women with a suggestive history, or the diagnosis will be missed entirely.

The rate of PPCM in Australia is about one in 3500 pregnancies.

PPCM is defined as heart failure developing towards the end of pregnancy or in the months following delivery, with LV ejection fraction nearly always <45%, in the absence of other identifiable causes.[9] PPCM is a diagnosis of exclusion and other causes of heart failure, including pre-existing dilated and familial cardiomyopathies, congenital heart disease (either previously known or unmasked in pregnancy), acute MI and pulmonary embolus must be considered. Although the cause remains unknown, one theory proposes a link with prolactin levels.

CLINICAL FEATURES OF PPCM

PPCM presents with symptoms and signs of heart failure (Table 5), typically in late pregnancy or in the first postpartum month.

An urgent echocardiogram is indicated in suspected PPCM.

ECHO demonstrates an LV ejection fraction <45%, fractional shortening <30% and global dilatation.

Input from a cardiologist experienced in obstetric medicine is mandatory.

MANAGEMENT OF PPCM

In cases presenting antenatally, delivery is indicated. Caesarean section is not universally required and vaginal delivery with close monitoring can be considered in stable patients.

Beta-1-blockade, (e.g. metoprolol) is indicated if tolerated; ACE inhibitors are commenced postpartum.

TABLE 5. PERIPARTUM CARDIOMYOPATHY

Risk factors	Clinical features
Multifetal pregnancies	Shortness of breath
Hypertensive disorders	Orthopnoea + PND
African ethnicity	Non-specific fatigue
Age >30 years	Haemoptysis
Drug use: cocaine, tocolysis	Pedal oedema

Anticoagulation is important if the LV function is severely impaired or if there is arrhythmia.

PROGNOSIS WITH PPCM

PPCM is a very serious disorder. Even in contemporary practice, maternal mortality approaches 10%.

Only 50% of women make a full recovery – with normal ECHO – at six months postpartum.

PPCM has a high recurrence risk (25– 50%) in future pregnancies.

Women with previous PPCM should have pre-pregnancy counselling by a maternal-fetal medicine specialist.

If there is residual LV dysfunction, then future pregnancy should be discouraged.

Gestational thrombocytopenia

Low-level thrombocytopenia (platelet count < 150 x 10^9/L) is common in pregnancy, affecting 5% of women. As such, further testing is usually only indicated for platelet counts < 100 x 10^9/L, which is only seen in 1% of pregnant women. By far the most common cause of platelet counts < 100 x 10^9/L during pregnancy is gestational thrombocytopenia.

WHAT IS GESTATIONAL THROMBOCYTOPENIA?

Gestational thrombocytopenia is an idiopathic disorder of uncertain pathophysiology, unique to pregnancy.

Three-quarters of all low platelet counts in pregnancy are due to gestational thrombocytopenia.

It is a diagnosis of exclusion; there is no diagnostic test other than normalisation of the platelet count after delivery, which can only be ascertained in retrospect.

The typical presentation is of a moderately low platelet count in the third trimester in a woman with a normal platelet count in early pregnancy.

Gestational thrombocytopenia virtually never causes platelet counts < 50–70 x 10^9/L. Importantly, there is no risk of fetal or neonatal thrombocytopenia.

WHAT OTHER CONDITIONS SHOULD BE CONSIDERED IN THROMBOCYTOPENIA IN PREGNANCY?

The main differential is autoimmune idiopathic thrombocytopenic purpura (ITP).

A low platelet count pre-pregnancy or in the first trimester suggests ITP as the cause.

The main distinction is that ITP has the potential to cause neonatal thrombocytopenia (5–10%) and, in rare cases, neonatal intracranial haemorrhage.[10]

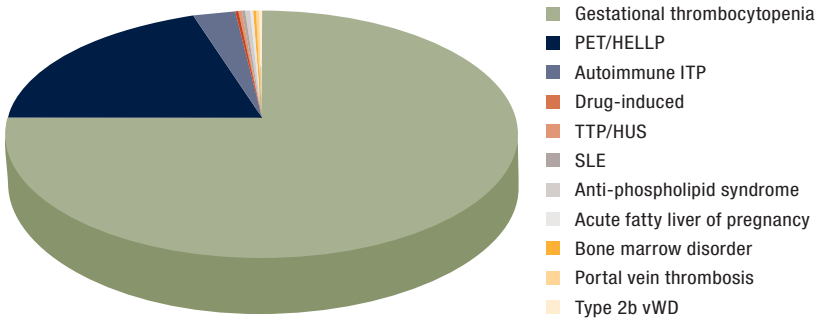
Testing for antiplatelet antibodies to diagnose ITP in pregnancy is not recommended, as the antibodies are not sensitive or specific.

Other potential causes for thrombocytopenia in pregnancy include:

- Pre-eclampsia/HELLP syndrome
- Thrombotic thrombocytopenic purpura (TTP)
- Secondary ITP from SLE, HIV, viral hepatitis or anti-phospholipid syndrome
- Rarely, haematological malignancy.

Together, gestational thrombocytopenia, PET/HELLP and ITP account for more than 95% of cases of low platelets in pregnancy (Figure 1).

FIGURE 1: THROMBOCYTOPENIA IN PREGNANCY



TREATMENT OF THROMBOCYTOPENIA IN PREGNANCY

Women with platelet counts < 100 x 10^9 in pregnancy require referral to a maternal-fetal medicine specialist or a haematologist experienced in obstetric haematology.

Gestational thrombocytopenia is a benign condition associated with mildly low platelet counts, and specific treatment, other than regular full blood counts, is not indicated.

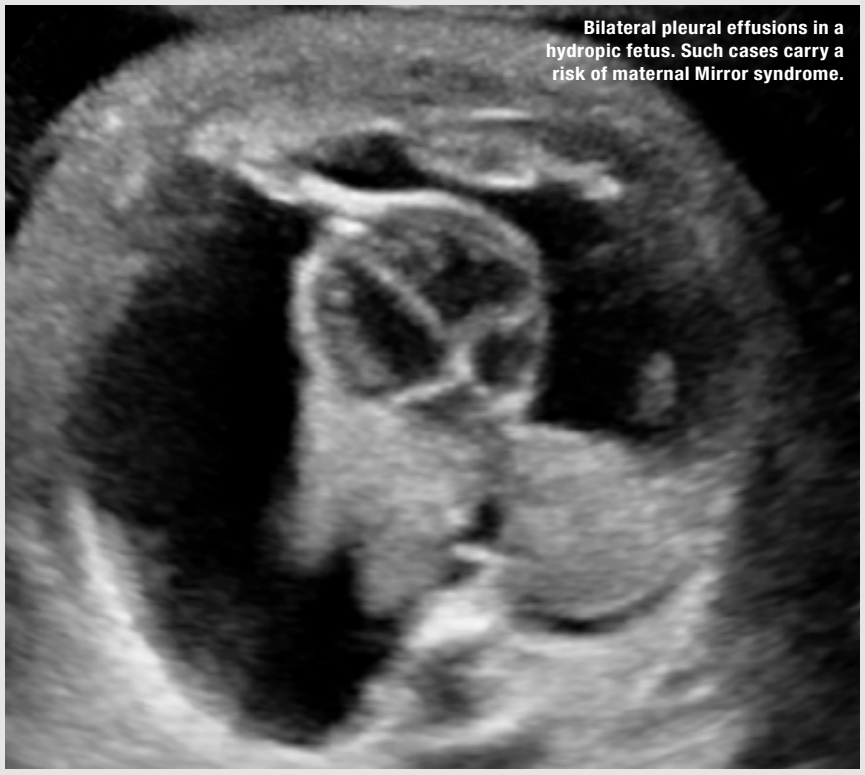
For women with ITP, bleeding is very uncommon at platelet counts > 50 x 10^9/L.

Treatment is indicated if the woman has abnormal bleeding, for platelet counts < 20 x 10^9/L (irrespective of bleeding), or for platelet counts <50 x 109/L near delivery.

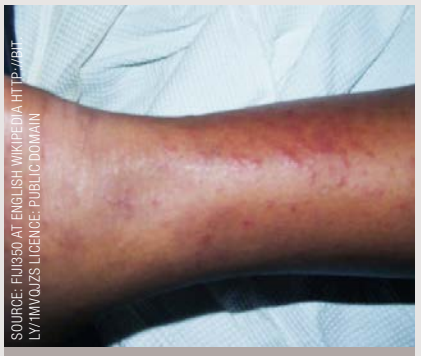
Oral prednisolone is the first-line treatment. Increasingly, IV immunoglobulin is used if a rapid response is needed.

Platelet transfusions will be rapidly consumed by women with ITP but may provide effective perioperative short-term cover for women requiring caesarean section.

Other pregnancy-specific medical disorders



Bilateral pleural effusions in a hydropic fetus. Such cases carry a risk of maternal Mirror syndrome.



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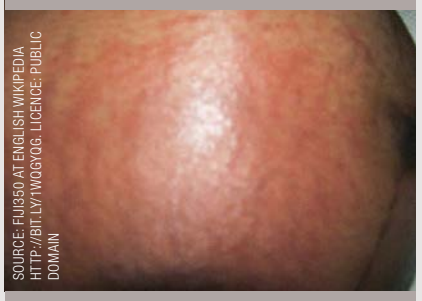
Feet and ankle view of pruritic urticarial papules and plaques of pregnancy.



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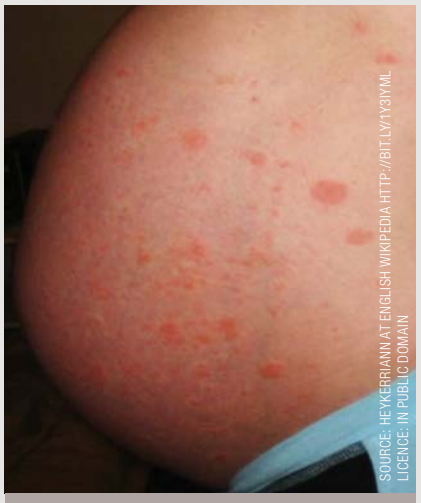
Abdomen with pruritic urticarial papules and plaques of pregnancy.



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Left side view of the abdomen at nearly 36 weeks' gestation, on which the papules and plaques of PUPPP can clearly be seen. The patient's fundal height is above the 95th percentile, and the skin is quite distended (stretched). Skin distension is a common factor in PUPPP, which is more common in mothers carrying large babies, twins and triplets. Although no stretch marks can be seen in this patient, the papules and plaques often first appear within stretch marks.

PUERPERAL (POSTPARTUM) PSYCHOSIS

The first month postpartum is the period in a woman's life with the highest risk of psychosis.

The overall prevalence of puerperal psychosis is 1:500 to 1:1000. The typical presentation is of hallucinations, delusions and bizarre behaviour, with associated mania or depression, within weeks of birth.

Half of affected women will have a previous psychiatric history. Substance use, as well as metabolic and endocrine causes for the symptoms should be excluded.

Suspicion of puerperal psychosis is considered a psychiatric emergency and urgent input from an experienced psychiatrist should be sought.

DERMATOSES OF PREGNANCY

The most common skin problem unique to pregnancy is PUPPP (pruritic urticarial papules and plaques of pregnancy), affecting about one in 200 women. It is much more common in twin pregnancies and in first-time mothers. It typically presents with intense itching around the abdominal region, and erythematous papules develop within the striae. Characteristically, the periumbilical region is spared. There is no association with adverse fetal outcome and therapy is directed at symptomatic relief.

The main differential diagnosis, which is far less common but more serious, is pemphigoid gestationis (PG, also called herpes gestationis). A very rare disorder, pruritic abdominal plaques coalesce to form large bullae and the periumbilical area is involved. Suspected PG needs prompt referral to a dermatologist for skin biopsy and diagnosis.

As there is a risk of fetal growth restriction and preterm delivery with PG, women should also be referred to a maternal-fetal medicine specialist for ongoing care.

MIRROR (BALLANTYNE) SYNDROME

This is a rare variant of pre-eclampsia which affects pregnant women whose pregnancies are complicated by fetal hydrops. Hydrops is essentially fetal heart failure and can be due to a variety of causes. Occasionally the mother of a hydropic fetus can develop severe peripheral oedema, shortness of breath, hypertension and proteinuria. Effectively, the maternal signs 'mirror' those of the hydropic fetus. Treatment is usually expeditious delivery, which leads to resolution of the maternal condition.

CHOREA GRAVIDARUM

This is characterised by random, jerky body movements beginning for the first time during the pregnancy. The diagnosis should only be made by an experienced neurologist after excluding other potential causes, including antiphospholipid syndrome, heart disease, hyperthyroidism and Wilson's disease.[11] The abnormal movements usually resolve within a few weeks of delivery.

References at medobs.com.au