Renal disease in pregnancy

Kate Wiles
Priscilla Smith

Abstract

Diagnosis of acute kidney injury (AKI) in pregnancy is complicated by haemodynamic and urinary tract changes in pregnancy, and non-pregnant references intervals for serum creatinine should not be used. Pre-eclampsia is the most common cause of AKI in pregnancy. Although pregnancy-associated haemolytic mircoangiopathies are rare, it is useful for physicians and nephrologists to be aware of potential clinical discriminators.

Pregnancy is successful for most women with chronic kidney disease (CKD). There is, however, an increased risk of adverse maternal and neonatal outcomes at all stages of CKD, including pre-eclampsia, growth restriction, preterm delivery, low birthweight, neonatal unit admission and postpartum loss of maternal renal function. Women with CKD should therefore have access to pre-pregnancy counselling in order to be able to make an informed decision about proceeding with pregnancy. A multidisciplinary team with expertise in obstetric nephrology should coordinate the care of pregnant women with CKD, which includes obstetric and renal surveillance, monitoring of immunosuppression, and the initiation and/or intensification of haemodialysis if required. The diagnosis of superimposed pre-eclampsia remains clinically challenging.

Keywords Acute kidney injury; chronic renal insufficiency; dialysis; pre-eclampsia; pregnancy; renal transplantation

Key points

- Acute kidney injury in pregnancy can be missed if non-pregnancy reference ranges for serum creatinine are used
- Pregnancy is successful for most women with chronic kidney disease (CKD)
- An increased risk of adverse pregnancy outcomes mandates prepregnancy planning and multidisciplinary expertise in pregnancy
- Pre-eclampsia is a common cause of acute kidney injury in pregnancy, and making the diagnosis in women with CKD can be difficult

Acute kidney injury (AKI)

Clinicians often miss pregnancy-associated-AKI, which can be masked by the use of non-pregnant reference intervals. The normal haemodynamic changes of pregnancy lead to a fall in serum creatinine during the first trimester from a mean pre-pregnancy concentration of 60 micromol/litre, to a nadir of 47 micromol/litre in the second trimester, before climbing back to prepartum levels at term. Systematic review shows that upper reference limits for creatinine in pregnancy are 85%, 80% and 86% of the non-pregnant limits in the first, second and third trimesters, respectively. Therefore, if the upper reference limit for non-pregnant creatinine is 90 micromol/litre, values >77 micromol/litre in pregnancy should trigger investigations to exclude AKI (or undiagnosed chronic kidney disease (CKD)).

Definitions of AKI in non-pregnant women are based on threshold levels of rise in creatinine and urine output. These cannot, however, be used in pregnancy as gestational variations in serum creatinine mean there is no baseline against which relative change can be reliably assessed. In addition, physiological postpartum oliguria impacts on urine output parameters.

Causes of AKI in pregnancy mirror those in non-pregnant populations, with the addition of pregnancy-specific aetiologies (Table 1). Initial management of AKI is an assessment of maternal and fetal well-being. Volume status should be optimized, nephrotoxic medications stopped, acute obstruction excluded with appropriate imaging, urinary tract infection treated and investigation of primary renal disease determined by urinary sediment, systemic features and stage of gestation. Maternal indications for renal replacement mirror those in non-pregnant women and include refractory hyperkalaemia/acidosis and oligoanuric fluid overload. The fetotoxicity of urea is a factor in initiating dialysis in CKD (see below), but there are no data on the maternal serum urea concentration at which dialysis confers clinical benefit in AKI.

Renal biopsy is difficult to perform in the prone position with increasing gestation, and the risk of bleeding is higher (around 7%) in the late second and third trimesters compared with the postpartum period (around 1%). Renal biopsy is therefore advocated only where a histological diagnosis will lead to a change in management during pregnancy.

The most common cause of AKI in pregnancy is pre-eclampsia. Standard diagnostic criteria for pre-eclampsia are the development of *de novo* hypertension (>140/90 mmHg) after 20 weeks' gestation in conjunction with either *de novo* proteinuria (urinary protein:creatinine ratio (uPCR) >30 mg/mmol), maternal organ dysfunction (AKI, transaminitis, neurological symptoms, thrombocytopenia) or evidence of uteroplacental dysfunction (fetal growth restriction, abnormal umbilical artery Doppler recordings).[1] Fluid balance in pre-eclampsia is complicated by hypoalbuminaemia, endothelial dysfunction and capillary leak. Women with pre-eclampsia are therefore vulnerable to pulmonary oedema with even small volumes of fluid, leading to

Kate Wiles *MSc MRCP is a Doctoral Research Fellow in Obstetric Nephrology at Guy's and St. Thomas' NHS Foundation Trust and King's College London, UK. Competing interests: none declared.*

Priscilla Smith *MB BS is a Senior Clinical Fellow in Renal Medicine at Guy's and St Thomas' NHS Foundation Trust, London, UK. Competing interests: none declared.*

Causes of AKI in pregnancy

Gestation	Diagnosis	Clinical features
Early	Hyperemesis gravidarum	Vomiting, ptyalism
	Septic abortion/ miscarriage	Abdominal pain, vaginal bleeding, sepsis
Mid to late	Pre-eclampsia (see text)	*De novo* hypertension after 20 weeks' gestation with any of: • *De novo* proteinuria (uPCR >30 mg/mmol) • Maternal organ dysfunction • Evidence of uteroplacental insufficiency
	HELLP (see text)	Haemolysis, elevated liver enzymes, low platelets. A serious manifestation of pre-eclampsia
	Bladder outflow obstruction	Consider risk factors: single kidney, neuropathy, polyhydramnios, multiple pregnancy, obstructed labour Physiological dilatation of the urinary tract in pregnancy can mimic hydronephrosis. Look for failure of decompression in the prone position and an absence of urinary jets
	Placental abruption	Abdominal pain, uterine tenderness, vaginal bleeding
	Acute fatty liver of pregnancy	Elevated transaminases, hypoglycaemia, lactic acidosis. New nausea/vomiting in the third trimester
	Microangiopathic haemolytic anaemia (TTP/aHUS) (see text)	Platelet consumption leading to haemolysis and end-organ damage including renal failure (aHUS) and neurological symptoms (TTP)
Peripartum	Chorioamnionitis	Sepsis, uterine tenderness, abnormal lochia, prolonged rupture of membranes
	Postpartum haemorrhage	Intravascular volume depletion, hypotension
	Ureteric injury	Operative delivery, fever, leucocytosis, pain, persistent ileus
	Non-steroidal anti-inflammatory drugs	Most commonly used postpartum analgesia
Any	Urosepsis	Dysuria, back/flank pain, renal angle tenderness, sepsis
	Lupus nephritis	Proteinuria ± haematuria, malaise, joint pain, hair loss, rash, pancytopenia. Positive ANA/dsDNA, low C3/C4
	Glomerulonephritis	Persistent proteinuria (uPCR >30 mg/mmol) before 20 weeks should be investigated to exclude primary renal disease
	Interstitial nephritis	New drug exposure
	Renal stone disease	Renal colic

ANA, antinuclear antibodies; dsDNA, anti-double-stranded DNA; HELLP, haemolysis, elevated liver enzymes and low platelets; uPCR, urinary protein:creatinine ratio.

Source: Adapted from Wiles KS and Banerjee A. Acute kidney injury in pregnancy and the use of non-steroidal anti-inflammatory drugs. *Obstetr Gynaecol* 2016. doi.org/10.1111/tog.12257.

Table 1

increased maternal morbidity and mortality, with no evidence of improved uteroplacental perfusion. Pre-eclampsia is therefore managed with replacement of insensible losses (30 ml/hour) along with anticipated urinary losses (0.5–1 ml/kg/hour), while restricting overall intake to 80–100 ml/hour to avoid risk of pulmonary oedema.[1]

Although rare, thrombotic thrombocytopenic purpura (TTP) and atypical haemolytic–uraemic syndrome (aHUS) can present in pregnancy or the postpartum period because of a gestational fall in a disintegrin and metallopeptidase with thrombospondin type 1 motif 13 (ADAMTS13) and peripartum activation of the alternative complement pathway respectively. Such conditions have phenotypic overlap with haemolysis, elevated liver enzymes and low platelets (HELLP) syndrome, which is a severe manifestation of pre-eclampsia; however, rapid clinical distinction is required to commence timely treatment with plasma exchange and/or eculizumab, which therapeutically blocks complement activation in aHUS (Table 2).

CKD and pregnancy

An overview of the management of CKD in pregnancy is shown in Figure 1.

Before pregnancy

Fertility and contraception: CKD is associated with mechanistic effects on fertility via disruption of the hypothalamic–pituitary–ovarian axis.[2] However, unintended pregnancies can and do occur in women with CKD, including women on dialysis. It is therefore vital that all reproductive-age women with CKD have access to contraceptive counselling, and that safe and effective contraceptive methods are offered to women who are taking teratogenic medication, are within the first year after renal transplantation, have active vasculitis or for all who not wish to conceive.

Progesterone-only contraceptives avoid the risks of venous thromboembolism, arterial thrombosis and cervical cancer that are associated with oestrogen-containing agents, adverse effects of particular relevance to women with nephrotic syndrome, with advanced CKD or on long-term immunosuppression. The progesterone-only pill (desogestrel), subdermal progesterone implant (Nexplanon®) and the progesterone-containing

Possible distinguishing features of aHUS/TTP and HELLP

Feature	HELLP	HUS/TTP
Incidence	Around 1% of pregnancies	1 in 25,000 pregnancies
Gestation	Prepartum (70%) Postpartum (30%) Occurs after 20 weeks' gestation	TTP: second and third trimester and postpartum period HUS: >75% postpartum Can occur before 20 weeks' gestation
Blood pressure	>160/110 mmHg	Evidence of haemolysis in the absence of hypertension
Platelet count	$<100 \times 10^9$/litre, but $<10 \times 10^9$/litre is rare	Usually $<70 \times 10^9$/litre; $<10 \times 10^9$/litre is suggestive of aHUS/TTP
Haemoglobin	>11 g/litre favours HELLP	<80 g/litre in the absence of another cause favours aHUS/TTP
Abnormal liver function	Transaminitis. LDH:AST <10:1 suggests HELLP	Normal liver function, unconjugated hyperbilirubinaemia
LDH	Typically <1000 U/litre	Typically >1000 U/litre
AKI	3–15% of HELLP Serum creatinine typically <180 micromol/litre	Serum creatinine typically >180 micromol/litre in aHUS
Coagulopathy	20% of HELLP	None. Elevated antithrombin and fibrinogen may be seen
Postpartum progress	Clinical improvement by 48–72 hours postpartum	Persistent laboratory abnormalities >72 hours after delivery
ADAMTS13	Deficiency may be found in HELLP	Activity <10% in TTP
Complement abnormalities	Described in HELLP	Detected in >80% pregnancy associated aHUS
Treatment	Delivery, supportive	Plasma exchange, eculizumab

ADAMTS13, a disintegrin and metalloproteinase with a thrombospondin type 1 motif 13; aHUS, atypical haemolytic uraemic syndrome; AST, aspartate aminotransferase; HELLP, Haemolysis Elevated Liver enzymes and Low Platelets; LDH, lactate dehydrogenase.

Table 2

intrauterine system (Mirena®) are considered safe and effective in women with CKD.[2] In addition, emergency contraceptives in the UK act via progesterone (levonorgestrel, ulipristal) and can be safely prescribed within 72 hours of unprotected intercourse to women with CKD.

Pre-pregnancy counselling: this provides an individualized, evidence-based assessment of the risk of adverse pregnancy outcomes, prevents exposure to teratogenic medication and offers an opportunity to optimize maternal health for pregnancy.

The risk of adverse pregnancy outcomes including pre-eclampsia, fetal growth restriction, preterm delivery, low birthweight, neonatal unit admission and loss of maternal renal function increase with a higher stage of CKD pre-pregnancy (Table 3). However, even women with stage 1 CKD and preserved glomerular filtration have an increased pregnancy risk compared with women without renal disease, even after correction for systemic disease, proteinuria, hypertension and late referral, suggesting that CKD per se confers risk in pregnancy.[3] Chronic hypertension and proteinuria are independent risk factors for adverse pregnancy events, and evidence from observational cohorts shows that optimizing these parameters in advance of pregnancy is associated with improved pregnancy outcomes.

Known teratogens should be discontinued before pregnancy and converted to pregnancy-safe alternatives. The most commonly prescribed teratogen in women with CKD is mycophenolate, which should be discontinued at least 6 weeks before pregnancy; however, a longer time might be needed to ensure disease/transplant stability on a pregnancy-safe alternative, commonly azathioprine.

Angiotensin-converting enzyme (ACE) inhibitors are toxic to the fetal kidney if exposed in the second and third trimesters; however, population data demonstrate no additional exposure risk in the first trimester when data are corrected for maternal comorbidities including diabetes mellitus and chronic hypertension. In women with proteinuric CKD, ACE inhibitors can therefore be continued for nephroprotection provided regular pregnancy testing is undertaken to ensure early confirmation of pregnancy. There are limited data on angiotensin receptor antagonist exposure in pregnancy, so these agents are discontinued in advance of attempts to conceive. Medication considerations for pregnancy and lactation are shown in Table 4.

Active lupus nephritis confers a risk of maternal hypertension and preterm delivery; thus women should be advised to delay pregnancy until their disease has been quiescent on stable treatment for at least 6 months. Hydroxychloroquine is advocated for women with lupus nephritis in pregnancy to minimize corticosteroid exposure, prevent flare and reduce fetal growth restriction. The antibodies SSA (Ro) and SSB (La) can undergo placental transfer, conferring a 15% risk of self-limiting neonatal cutaneous lupus and 2–5% risk of congenital heart block, which requires fetal heart rate and echocardiography surveillance. Hydroxychloroquine reduces the risk of congenital heart block in women with previously affected infants, and these data are generalized for primary prevention.

Women with diabetic nephropathy should be supported in optimizing blood glucose as any reduction in prepregnancy glycated haemoglobin (HbA_{1c}) towards an optimum of 48 mmol/litre (6.5%) confers a decrease in the risks of miscarriage, congenital malformation, stillbirth and neonatal death.

Meta-analysis data examining transplant-to-pregnancy intervals have shown that an interval <2 years from transplant to conception is associated with a higher live birth rate and lower

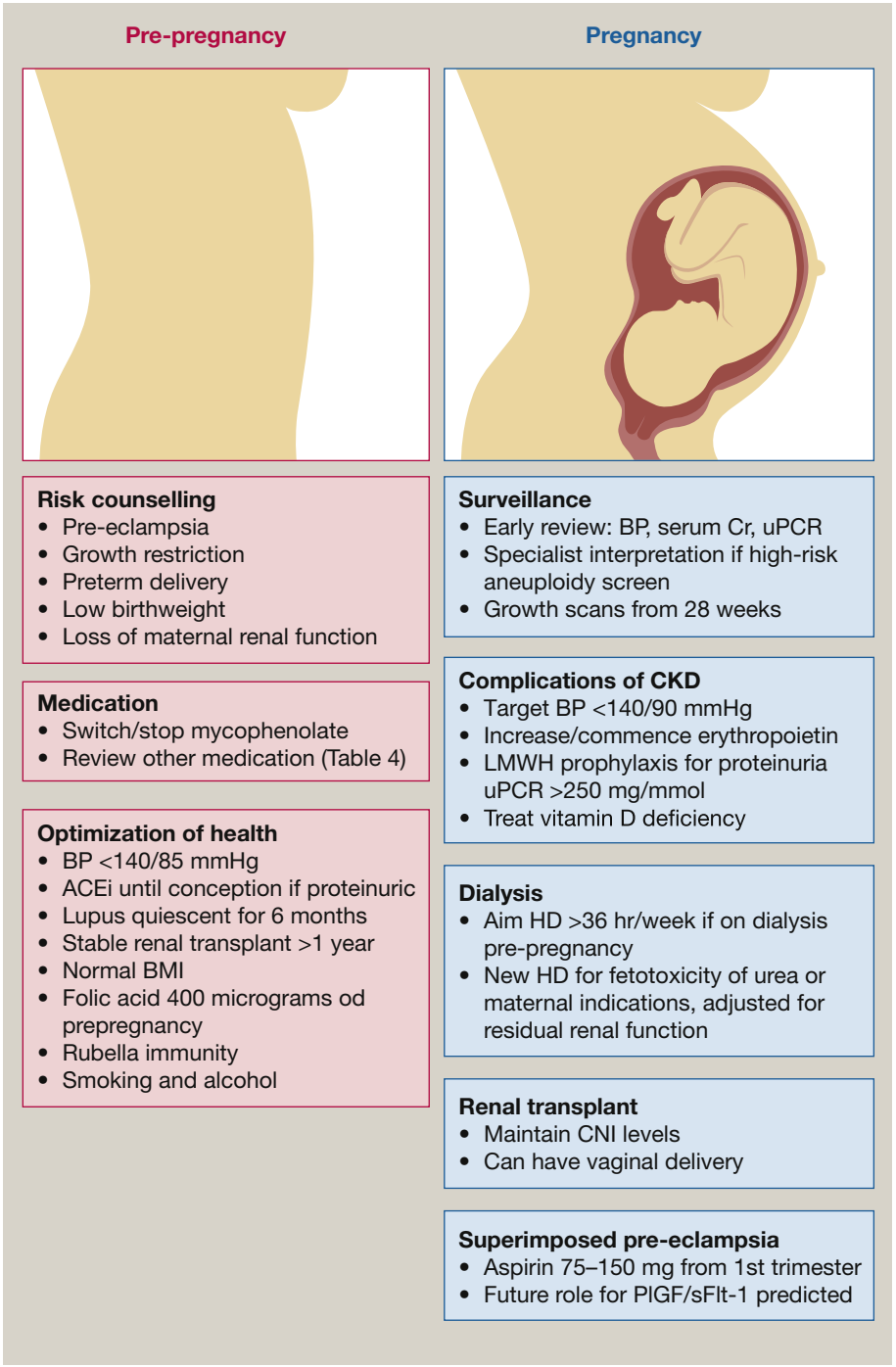

Figure 1 An overview of the management of chronic kidney disease (CKD) in pregnancy. ACEi, ACE inhibitor; BMI, body mass index; BP, blood pressure; CNI, calcineurin inhibitor (tacrolimus/ciclosporin); Cr, creatinine; HD, haemodialysis; LMWH, low-molecular-weight heparin; PlGF, placental growth factor; uPCR, urinary protein: creatinine ratio; sFlt-1, soluble fms-like tyrosine kinase-1.

miscarriage rate, although pre-eclampsia, gestational diabetes mellitus, caesarean delivery and preterm birth are common.[4] Standard immunosuppression regimens routinely include mycophenolate in the first year after transplantation; therefore a delay of at least a year after transplantation is advised before attempting to conceive provided a switch to azathioprine is feasible and subsequent graft function remains stable. Additional considerations include previous transplant rejection, graft function, cytomegalovirus infection, hypertension and proteinuria, although direct evidence regarding their impact on pregnancy and transplant outcomes is limited.

Pregnancies in women on dialysis are high risk, both in absolute terms and in comparison to pregnancy after successful kidney transplantation. Delaying pregnancy until after

Geometric mean rates (%) of adverse pregnancy outcomes from contemporaneous prospective cohorts according to CKD stage[2,3]

Outcome	Control	CKD stage			
		1	2	3	4-5
eGFR (ml/minute/1.73 m^2)		>90	60–89	30–59	<30
n	915	426	118	65	17
Preterm delivery <37/40 weeks	6	23	42	61	77
Preterm delivery <34/40 weeks	1	8	16	28	44
Birthweight <5th centile	5	6	9	5	38
Neonatal unit admission	2	10	23	33	65
Pre-eclampsia	6	18	13	21	43
CKD stage shift	NA	8	13	16	20
25% reduction in eGFR at 6 months postpartum	NA	8	15	23	67

CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; NA = not applicable.

Table 3

transplantation is therefore advocated for women who are established on or approaching dialysis, if renal transplantation is anticipated during reproductive age.

Pregnancy

There is no evidence specifically addressing the schedule of care for pregnancy in women with CKD. Expert consensus is that all women with CKD should have a plan for pregnancy made by a multidisciplinary team with expertise in obstetric nephrology; including appropriate surveillance and plans for delivery. Clinical review in early pregnancy is important to establish baseline levels of serum creatinine and proteinuria, to optimize blood pressure control (<140/85 mmHg) and to ensure that low-dose aspirin (75–150 mg) is commenced as prophylaxis against later pre-eclampsia. Renal function in pregnancy is monitored by serum creatinine as eGFR variably underestimates renal function and is not valid.

Women with CKD should be offered routine trisomy screening, which includes quantification of β-human chorionic gonadotrophin (β-HCG). This can be elevated in women with CKD as a result of reduced renal clearance, so positive screening based on a high β-HCG concentration requires specialist interpretation, with

Safety of medication used in CKD in pregnancy and lactation

Prepregnancy		Pregnancy		Lactation	
Considered safe	**Unsafe or unknown**	**Considered safe**	**Unsafe or unknown**	**Considered safe**	**Unsafe or unknown**
Labetalol	ARB	Labetalol	ACE inhibitors[b]	Labetalol	Methyldopa[d]
Nifedipine	Mycophenolate	Nifedipine	ARB	Nifedipine	ARB
Methyldopa	Cyclophosphamide	Methyldopa	Mycophenolate	Enalapril/captopril	Mycophenolate
ACE inhibitors	Allopurinol	Corticosteroids	Cyclophosphamide	Corticosteroids	Cyclophosphamide
Corticosteroids	Cinecalcet	Azathioprine	Warfarin	Azathioprine	Allopurinol
Azathioprine	HIF stabilisers	Ciclosporin	Allopurinol	Ciclosporin	Cinecalcet
Ciclosporin		Tacrolimus	Cinecalcet	Tacrolimus	HIF stabilisers
Tacrolimus		Hydroxychloroquine	HIF stabilisers	Hydroxychloroquine	
Hydroxychloroquine		Aspirin		Aspirin	
Aspirin		LMWH		LMWH	
LMWH		Colchicine		Colchicine[c]	
Colchicine		Iron[a]		Iron	
Iron		Erythropoietin		Erythropoietin	
Erythropoietin					

Limited data:

- Rituximab: no evidence of teratogenicity. Placental transfer in the second and third trimesters. Treatment pre/early pregnancy minimizes risk of neonatal B cell depletion, but live vaccines should be appropriately delayed. Trace amounts in breast milk, but neonatal absorption is unlikely. No long-term outcome data.
- Eculizumab: no evidence of teratogenicity ($n = 20$). Benefit likely to be greater than theoretical risk given the morbidity of the underlying maternal condition. Trace amounts in breast milk, but neonatal absorption is unlikely. No long-term outcome data.

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blockade; HIF, hypoxia inducible factor; LMWH, low-molecular-weight heparin.

[a] No data on intravenous iron in the first trimester.
[b] Toxic to the fetal kidney in the second and third trimesters.
[c] Estimated 10% of maternal dose is received by the infant.
[d] Risk of postnatal depression.

Source: Adapted from Wiles et al. (2018).[2]

Table 4

consideration of non-invasive prenatal testing using circulating fetal DNA if available. Ultrasound assessment of fetal growth and well-being should occur every 4 weeks from 28 weeks' gestation, and more frequently if there is any clinical concern.

Physiological adaptation to pregnancy results in an increase in proteinuria and circulating erythropoietin concentration. Although the threshold concentration of proteinuria that confers a clinically significant maternal thromboembolic risk remains unknown, expert consensus is to commence low-molecular-weight heparin prophylaxis if uPCR is >300 mg/mmol, or >100 mg/mmol if there are other risk factors. The need to initiate and/or titrate synthetic erythropoietin should be anticipated.

Renal transplants: women with renal transplants require regular monitoring of tacrolimus/ciclosporin levels; as these fall in pregnancy, doses should be increased to maintain prepregnancy levels. Antibiotic prophylaxis should be considered after confirmed and treated urinary tract infection, including asymptomatic bacteriuria. Women with renal transplants can have a vaginal delivery, with transplant team input into a surgical plan made in advance, in case obstetric indications for caesarean delivery arise.

Dialysis: women established on dialysis before pregnancy should be given increased haemodialysis in pregnancy, with >36 hours/week shown to improve neonatal outcomes.[4] New haemodialysis in pregnancy is most commonly initiated because of concern about the fetotoxicity of urea, rather than for standard indications including refractory fluid overload, hyperkalaemia or acidosis. The optimum urea threshold at which dialysis should be commenced in pregnancy remains unknown as limited historical data fail to reflect contemporary obstetric practice. Discussion should begin at a urea concentration >15 mmol/litre so haemodialysis can be commenced at a concentration >17–20 mmol/litre depending upon the trajectory of decline of renal function, stage of gestation and risks of iatrogenic preterm delivery. Residual renal function is thought to be important in women who are new to dialysis in pregnancy, which begins 'gently' (e.g. for 2 hours three times per week) and titrated according to maternal parameters; no additional benefit has been shown for long, frequent dialysis in a single small cohort of women newly commencing dialysis in pregnancy.[4]

Pre-eclampsia: the risk of pre-eclampsia is increased in women with CKD, and increments with CKD stage (Table 3). Low-dose aspirin (75–150 mg) is given from the first trimester to reduce the risk of pre-eclampsia, based on high-quality evidence from pregnant women without CKD. For women with hypertension and/or proteinuria before pregnancy, there are no standard diagnostic criteria for superimposed pre-eclampsia. Relative changes in blood pressure and proteinuria after 20 weeks' gestation are difficult to interpret in the context of gestational change, but should trigger increased surveillance for uteroplacental and maternal organ dysfunction.[1]

It can be challenging to distinguish between pre-eclampsia and lupus flare because of overlapping phenotypes that include proteinuria, hypertension and thrombocytopenia. Systemic features, double-stranded DNA titres and complement levels can help. There is emerging evidence that placental growth factor and soluble fms-like tyrosine kinase-1 (sFlt1) concentrations can be used for the prediction and prognosis of pre-eclampsia in women without CKD. The validation of these tests in CKD is eagerly awaited.

Postpartum

Women with CKD should be supported in breastfeeding. Drug regimens can and should be made safe for lactation (Table 4).

Numerous studies have shown that the lifespan of a renal transplant is not significantly different in transplant recipients undertaking pregnancy compared with those who do not.[5] However, for women with prepregnancy CKD stages 3–5, there is a step-loss in renal function during pregnancy that equates to 1–3 years of background disease; this potentially brings forward the need for dialysis and transplantation. ◆

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TEST YOURSELF

To test your knowledge based on the article you have just read, please complete the questions below. The answers can be found at the end of the issue or online here.

Question 1

A 32-year-old woman was reviewed 3 days after the delivery of a female infant weighing 2.4 kg. This was her first child. She had been seen initially at 37 weeks gestation with a 24-hour history of headache, blood pressure was 162/110 mmHg and platelet count was 75 x10^9 per litre. Labour had been induced. Following delivery her blood pressure had been treated with labetalol and enalapril. On clinical examination, blood pressure was 152/105mmHg, there were no other abnormal findings.

Investigations

Haemoglobin 90 g/litre (115–145)
Platelets 140 x10^9/litre (150–400)
Serum creatinine 125 micromol/litre (45–90)
Alanine aminotransferase 55 U/litre (<45)
Lactate dehydrogenase 480 U/litre (10–250)

What is the most likely diagnosis?

A. Atypical haemolytic–uraemic syndrome.
B. Immune thrombocytopenic purpura (ITP)
C. Haemolysis, elevated liver enzymes and low platelet (HELLP) syndrome
D. Autoimmune haemolytic anaemia
E. Thrombotic thrombocytopenic purpura (TTP)

Question 2

A 27-year-old woman attended for routine transplant clinic review. She had been given a cadaveric renal transplant 4 years previously. She was well and had had no episodes of transplant rejection.

On clinical examination, her blood pressure was 127/78 mmHg on her current medication. She was taking erythropoietin, mycophenolate, nifedipine, azathioprine and tacrolimus, She and her partner were considering pregnancy.

Investigation

- Creatinine 137 micromol/litre (45–90)

Which of her medications should be discontinued in advance of pregnancy?

A. Erythropoietin
B. Mycophenolate mofetil
C. Nifedipine
D. Azathioprine
E. Tacrolimus

Question 3

A 34-year-old woman was seen at 13 weeks' gestation in a multidisciplinary obstetric nephrology clinic. She had been found to have chronic kidney disease caused by immunoglobulin (Ig)A nephropathy.

On clinical examination, her blood pressure was125/85 mmHg.

Investigations

- Haemoglobin 109 g/litre (non-pregnant 115–155)
- Urinary protein:creatinine ratio 50 mg/mmol (<30)
- Creatinine 95 micromol/litre (non-pregnant 45–90)

Which one of the following medications should be offered?

A. Folic acid 400 micrograms daily
B. Aspirin 75 mg daily
C. Ramipril 2.5 mg daily
D. Hydroxychloroquine 200 mg 12-hourly
E. Erythropoietin weekly.