Management of new-onset hypertension in pregnancy

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Abstract

Hypertensive disorders affect approximately 8–10% of all pregnancies and include pre-eclampsia, gestational hypertension and pre-existing chronic hypertension, which may be primary or secondary. New onset hypertension in pregnancy is defined as a sustained systolic blood pressure (sBP) ≥140 mmHg *and/or* diastolic blood pressure (dBP) ≥90 mmHg, and severe hypertension diagnosed when sBP ≥160 mmHg *and/or* dBP ≥110 mmHg. Gestational hypertension and pre-eclampsia are most common, affecting 4.2–7.9% and 1.5–7.7% respectively. Chronic hypertension affects 0.6–2.7% of pregnancies but may be under-reported due to early physiological adaptations in pregnancy lowering blood pressure or unknown preconception blood pressure. New onset hypertension developing at any stage of pregnancy requires a full history, examination, and investigations to delineate an underlying cause, assess for target organ damage and the presence of pre-eclampsia to assign risk. Developing a hypertensive disorder in pregnancy is associated with increased life-long cardiometabolic risk and other cardiovascular risk factors should be minimised to improve a woman's long-term health.

Keywords Blood pressure; cardiovascular; hypertension; pre-eclampsia; pregnancy

Introduction

Hypertensive disorders affect approximately 8–10% of all pregnancies and include pre-eclampsia (PET) and gestational hypertension, which are specific to pregnancy, and pre-existing chronic hypertension which may be primary or secondary. All these forms of hypertension cause significant morbidity and mortality. The most recent MBRRACE-UK (Mothers and Babies reducing risk through audits and confidential enquiries across the UK) report found a 4.75-fold increase in deaths from pre-eclampsia and eclampsia over the last 6 years. Despite ongoing education of healthcare professionals, warning signs of new onset hypertension are still frequently missed and thus informing women of the signs and symptoms of hypertensive disease in pregnancy is paramount to ensure that they seek clinical review urgently. This is most important in women of black ethnicity who are at higher risk of hypertensive disorders and its associated morbidity and mortality. The *5timesmore* campaign, has encouraged patient-education and implemented a "Six steps" guide to support black women to advocate for themselves alongside investigating disparities in maternity care for black women.

Hypertension in pregnancy is defined as a sustained systolic blood pressure (sBP) of 140 mmHg or more, *or* diastolic blood pressure (dBP) of 90 mmHg or more and severe hypertension diagnosed when sBP ≥160 mmHg *and/or* dBP ≥110 mmHg. Gestational hypertension and pre-eclampsia are most common, affecting 4.2–7.9% and 1.5–7.7% respectively. Chronic hypertension affects 0.6–2.7% of pregnancies but may be under-reported due to a combination of early maternal physiological adaptations to pregnancy lowering BP, and unknown preconception blood pressure.

Women who are affected by hypertensive disorders in pregnancy have an increased risk of developing hypertension in the immediate postpartum period, and as a group also develop hypertension up to 10 years earlier than women who had a normotensive pregnancy. They are also more likely to suffer from hypertension in future pregnancies. Additionally, they have a 2–3 fold higher risk of future ischaemic heart disease, stroke and diabetes. Finding appropriate pathways to educate affected women about their long-term risk alongside prospective BP/CV screening and management postpartum may help to reduce the overall incidence of subsequent cardiovascular events.

Definition of hypertension and severe hypertension

Hypertension in pregnancy is defined as a systolic blood pressure (sBP) of ≥140 and/or a diastolic (dBP) ≥90 mmHg and a sBP ≥160 mmHg and/or dBP ≥110 mmHg defined as severe hypertension. This should be diagnosed based on at least two readings, averaged to reflect the BP in clinic. If BP values have a >10 mmHg difference, a third measurement should be taken, and the second and third measurements used for the average. Although the diagnostic threshold of hypertension in pregnancy is internationally recognised, there is still debate and differences in guidelines worldwide regarding the BP threshold required for treatment of hypertension in pregnancy. The classification of hypertensive disorder of pregnancy can be seen in Figure 1.

In the UK, hypertensive thresholds in non-pregnant people remain sBP ≥140 mmHg and/or a dBP ≥90 mmHg from clinic blood pressure measurements, or ≥135/85 mmHg on home or ambulatory BP. Whilst the American Heart Association guidelines have reduced diagnostic targets for a diagnosis of stage 1 hypertension in non-pregnant people to sBP of 130–139 mmHg and/or dBP of 80–89 mmHg, these definitions have not been adopted for pregnancy in the UK or US. Identifying women with stage 1 hypertension may indicate those at increased risk of adverse pregnancy outcomes including PET. However, Community-Level Interventions for Pre-eclampsia (CLIP) study, which measured blood pressure in low-middle income countries, showed no benefit in reducing the hypertension diagnostic threshold in pregnancy to 130/80 mmHg. Stage 1 hypertension in this study was not associated with maternal, fetal, or neonatal morbidity or mortality. A recent UK prospective longitudinal

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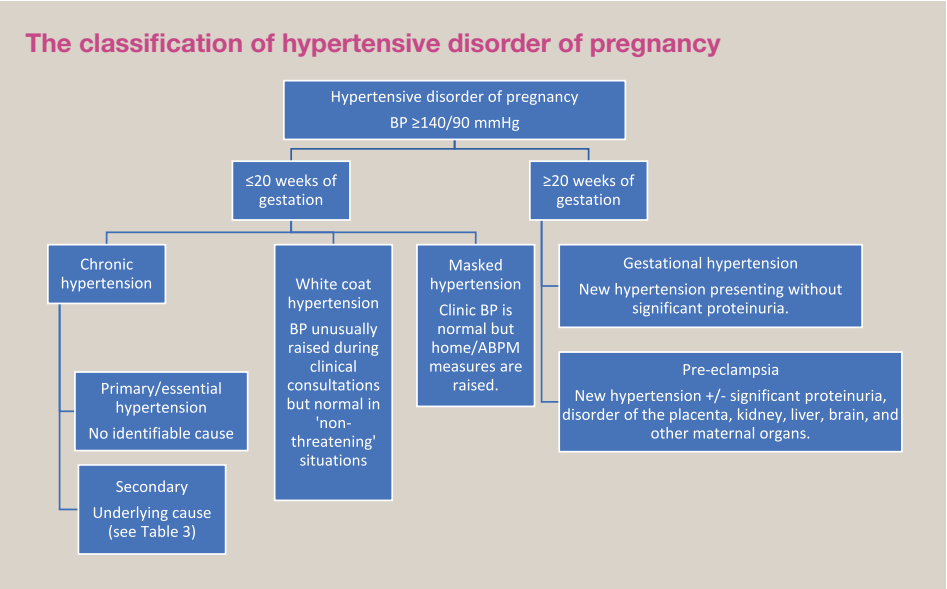

Figure 1

cohort study supported ≥140/90 mmHg as the hypertension threshold as women with normal BP values in the 97th centile at 40 weeks of gestation had a blood pressure of 143/94 mmHg. This was slightly higher in nulliparous women (146/96 mmHg) and lower in parous women (140/92 mmHg) at 40 weeks of gestation.

Severe hypertension, defined as a diagnostic threshold of sBP ≥160 mmHg and/or ≥dBP 110 mmHg, is internationally recognised and is lower than the severe hypertension diagnosis in non-pregnant populations due to the increased risk of haemorrhagic stroke and aortic dissection in pregnant populations at sBP ≥160 mmHg. Women presenting with severe hypertension require admission and urgent assessment in hospital.

If severe hypertension occurs in the first 20 weeks of pregnancy it is usually due to chronic hypertension which can have either a primary (90% of cases) or secondary cause. Assessment should include understanding the trajectory of blood pressure rise to ensure appropriate response to managing acute-onset hypertension. Severe hypertension occurring at 20 weeks or more of gestation should be considered an emergency due to the likely development of PET and risk of end organ damage.

It is important to recognise in the non-pregnant population, diagnostic and treatment thresholds for hypertension and severe hypertension are different. This is due to physiological haemodynamic changes that occur in pregnancy which cause a reduction in systemic vascular resistance and expected lowering of blood pressure in pregnancy. Endothelial dysfunction and the loss of cerebral autoregulation that occurs in PET, exacerbates the effect of hypertension in pregnant women, leading to an increased risk of cerebrovascular injury at lower blood pressure values compared to those with hypertension without PET. It is vital that pregnant women's observations are documented on a specific Maternity Early Warning Score, such as the new National MEWS score which is being introduced across all maternity units in England. This identifies women with hypertension in pregnancy early, whereas a non-pregnant National early warning score (NEWS) would miss BP rising above pregnancy diagnostic thresholds.

Prevention of hypertension before and during pregnancy

Preconception health has a significant impact on the health of a pregnancy as well as impacting on health across generations within the life-course. This benefit is also seen in relation to the development of new hypertension in pregnancy and its outcomes. Women with or without chronic hypertension that have a higher BMI and baseline systolic/diastolic blood pressure, larger waist circumference and/or raised lipid levels are more likely to develop hypertensive disorders in pregnancy. In addition, the long-term CV risk from a pregnancy affected by hypertension is associated with pre-existing CV risk factors. Therefore reducing a woman's CV risk profile prior to pregnancy may reduce their long-term risk of CV disease independent of the development of a hypertensive disorder in pregnancy.

Risk stratifying and managing women with chronic hypertension preconception by investigating for persistent proteinuria, chronic kidney disease and/or left-ventricular hypertrophy is helpful to identify those with multiple risk factors for developing a hypertensive disorder and in whom more intensive antenatal care and monitoring is required. The choice of antihypertensive should be changed to one accepted in pregnancy e.g. labetalol, *modified release* nifedipine or methyldopa. Although treating chronic hypertension in pregnancy is beneficial to reduce the risk of severe hypertension, this does not reduce the risk of proteinuria or developing pre-eclampsia. Women who lose weight prior to pregnancy have reduced rates of hypertensive disorders of pregnancy, and this is equally important in-between pregnancies. The POPPY study (Preconception to Postpartum cardiometabolic study of primigravid pregnancy), a large observational preconception study, aims to investigate the impact of preconception cardiometabolic health on the development of hypertensive disorders in pregnancy and the long-term CV effects of this.

Exercise during pregnancy has been shown to be a safe, effective, and inexpensive way to reduce the risk of developing pre-eclampsia in pregnancy. A systematic review involving 3322 women showed exercise during pregnancy (at least 140 minutes of moderate-intensity per week), reduced the risk of pre-eclampsia by 40% without adverse fetal effects. The definition of moderate intensity was exercise that raised the heart rate but still allowed women to talk (although not sing) throughout. A systematic review and meta-analysis of 5939 women also showed a reduction in hypertensive disorders in those who undertook yoga or structured training.

The ASPRE (Combined Multimarker Screening and Randomised Patient Treatment with Aspirin for Evidence-Based Pre-eclampsia Prevention) study identified a high-risk group of women using a multi-variable first trimester screen and randomly assigned women to 150 mg of aspirin at night or placebo. There was a 62% reduction in the incidence of preterm pre-eclampsia (odds ratio, 0.38; 95% CI, 0.20 to 0.74) when aspirin was taken from 11 to 13 weeks' gestation until 36 weeks compared to placebo. As such, NICE guidance now advises those who are at increased risk of pre-eclampsia in pregnancy (see Table 1) to start aspirin therapy at 11–13 weeks' gestation.

Approach to new onset hypertension occurring at ≤20 weeks of gestation

Case 1

A 36-year-old female presented to her midwife at 18 + 4 weeks of gestation with a history of headache, vomiting, intermittent sweating, and dizziness. Her blood pressure was 220/110 mmHg initially with a repeat measure of 196/101 mmHg. She was transferred to the labour ward and given oral labetalol 200 mg, at which time her blood pressure became 90/60 mmHg and she felt faint. Her blood pressure was persistently labile with associated symptoms of headache, nausea, sweating and pallor. A secondary screen for hypertension was undertaking including plasma and urine metanephrines which were raised, and an abdominal MRI showed a large 10 cm paraganglioma.

Box 1

Hypertension occurring in the first 20 weeks of gestation is usually due to chronic hypertension, which affects 0.6–2.7% of pregnancies. 90% are idiopathic i.e. primary hypertension and the remainder have an underlying cause (secondary hypertension (Box 1)). The prevalence of chronic hypertension in pregnancy may be under representative due to the early physiological reduction in blood pressure, which can mask pre-existing hypertension. This physiological adaptation may explain why some women develop "new" persistent hypertension postpartum without a prior diagnosis of chronic hypertension if it had not been picked up in the preconception period. Secondary causes of hypertension are usually due to an underlying renal parenchymal disease e.g. reflux nephropathy or glomerulonephritis, and less commonly fibromuscular dysplasia, primary hyperaldosteronism, or undiagnosed monogenic hypertension. PET is rare in the first 20 weeks of gestation although can occur and should be considered a differential diagnosis in women presenting with new onset hypertension with or without proteinuria and multi-organ involvement.

Risk factors for pre-eclampsia

Women are at high risk of pre-eclampsia if they have

One of the following high-risk factors:	**Two or more of the following moderate-risk factors:**
A history of hypertensive disease during a previous pregnancy.	First pregnancy.
Chronic kidney disease.	Aged 40 years or older.
Autoimmune disease, such as systemic lupus erythematosus or antiphospholipid syndrome.	Pregnancy interval of more than 10 years.
Type 1 or type 2 diabetes.	Body mass index (BMI) of 35 kg/m^2 or greater at the first visit.
Chronic hypertension.	Family history of pre-eclampsia.
	Multiple pregnancy.

Table 1

All women with new onset hypertension in pregnancy should have a full history, examination and set of investigations (Table 2), considering CV risk factors, signs, and symptoms of end organ damage and evidence for secondary causes (Table 3). Women with new onset severe hypertension in pregnancy should be managed in hospital regardless of the timing of its onset in pregnancy.

The underlying pathophysiology of hypertension occurring in the first 20 weeks of pregnancy is likely to be different to those presenting in the last 20 weeks of pregnancy and this should be considered when treatment is chosen and commenced. Additional ultrasound scans should be requested for fetal growth and amniotic fluid volume assessment, and umbilical artery Doppler velocimetry at 28 weeks, 32 weeks and 36 weeks or as per local guidance.

Approach to new onset hypertension occurring at ≥20 weeks of gestation

Case 2

A 28-year-old woman at 33 weeks' gestation in her second pregnancy presents to the obstetric unit with a severe headache, new onset peripheral swelling and visual disturbance. Her BP is 180/110 mmHg and a urine dipstick showed protein 3+. Initial investigations showed a thrombocytopenia (platelets 89 × 10^9/L), acute kidney injury (creatinine 98 μmol/L) and a growth restricted fetus on the 6th centile. She was diagnosed with pre-eclampsia and treated with IV labetalol and Magnesium sulphate and delivery expedited due to deteriorating symptoms despite initial therapy.

Box 2

New onset hypertension occurring at ≥20 weeks' gestation is usually a pregnancy-induced disorder i.e. gestational hypertension or pre-eclampsia, or chronic hypertension not previously diagnosed. However, diagnostic curiosity should remain for

History, examination, and initial investigations for a patient presenting with new onset hypertension in pregnancy

HPC	Headaches/migraines, chest pain, shortness of breath, visual symptoms.
	Secondary HTN signs: palpitations, sweating, postural symptoms, weight changes, nocturia.
	PET signs: peripheral oedema, epigastric pain, headache, N+V, visual disturbance.
PMH	Hypertension, diabetes, obesity, hypercholesterolaemia, obstructive sleep apnoea (OSA), stroke/TIA, antiphospholipid syndrome (APS), connective tissue disorder.
	Previous pregnancies — PET/HELLP/gestational hypertension
	Current pregnancy — same partner?
DHx	Review medications as a secondary cause of hypertension (see Table 3).
	Aspirin PET prophylaxis
FHx	Hypertension, diabetes, premature CV disease
	PET, connective tissue disorder, APS, T1DM.
SHx	Smoking/vaping
	Recreational drugs: cocaine and amphetamines
	Over the counter medications/herbal remedies/patches/supplements
	Exercise
	Occupation
Examination (including end organ damage review)	Body habitus and BMI
	BP in both arms (×3) and postural BP
	Palpation and auscultation for heart and carotid arteries — heat murmur or bruit of aortic coarctation.
	Palpation of peripheral arteries - radio-radial delay, radio-femoral delay.
	Neurological examination and cognitive status — clonus, reflexes.
	Fundoscopy for hypertensive retinopathy
	Secondary hypertension signs: rash (vasculitis) or café-au-lait (associated neurofibromatosis and phaechromocytoma)
	Abdominal examination - enlarged kidneys/liver, renal bruit.
	Features of endocrine disease — Cushings or thyroid disease
Investigations	Urine: Dipstick, urine PCR/ACR
	Bloods: FBC, U+Es, LFT, HbA1C, VBG, glucose, bone (Ca^{2+}), clotting
	Troponin/BNP
	Secondary causes: Plasma and urine metanephrines, TSH, T3+T4, PTH
	Toxicology screen — if concerned
	Other: ECG, Renal USS, Fetal scan — growth and dopplers.

Table 2

those women who present atypically or in whom antihypertensive treatments are not working. In addition to pregnancy-specific causes, BP physiologically rises in the second half of pregnancy towards term back to pre-pregnancy levels and blood pressure in women with pre-existing chronic hypertension may trend upwards. If not frequently monitored and treated, this BP rise may supersede the hypertension and/or severe hypertension threshold unnoticed. Women with hypertension, and more so with severe hypertension, are at risk of maternal and fetal morbidity. The additional purpose of diagnosing pre-eclampsia is to identify women who are at a higher risk of end-organ damage such as haemorrhagic stroke (PET OR 10.4 vs chronic hypertension 2.6) (Box 2).

PET is defined as new-onset hypertension (sBP ≥140 mmHg and/or dBP ≥90 mmHg) occurring at ≥20 weeks' gestation with either 1 or more new-onset condition below:

- proteinuria (≥0.3 g/24 hour or uPCR ≥30 mg/mmol)
- haematological involvement: thrombocytopenia (platelet count $<150 \times 10^9$/L), DIC or haemolysis.
- acute kidney injury (creatinine >90 μmol/L).
- liver involvement (elevated transaminase e.g. alanine aminotransferase >40 IU/L or epigastric/right upper quadrant pain.
- neurological complications: eclampsia, altered mental status, blindness, stroke, clonus, severe headache or persistent visual disturbance (scotomata).
- Uteroplacental dysfunction e.g. fetal growth restriction, abnormal umbilical artery [UA] Doppler wave form, or stillbirth)

The pathophysiology is complex and involves defective trophoblast invasion and incomplete spiral artery remodelling in the placenta although the causative element remains unknown and on-going research is required. In-turn leading to endothelial dysfunction with multi-organ involvement and the clinical features of pre-eclampsia. Approximately 70% of women will present with late-onset pre-eclampsia (≥34 weeks' gestation) which appears less severe with a lower risk of developing end-organ damage and fetal growth restriction compared to those with early-onset disease (≤34 weeks' gestation).

Alongside the clinical and biochemical features of pre-eclampsia seen above, angiogenic factors can now be measured to help diagnose pre-eclampsia. This is particularly helpful in women who have pre-existing hypertension and/or proteinuria where baseline clinical and biochemical results cross with pre-eclampsia diagnostic thresholds. PlGF and sFLt-1 have been incorporated into NICE guidelines for the diagnosis of pre-eclampsia in pregnancy

Secondary causes of hypertension with investigations and implications in pregnancy

Secondary cause	History, signs, and symptoms	Diagnosis	Implication for pregnancy
Renovascular: Chronic kidney disease, chronic pyelonephritis diabetic nephropathy PCKD, Glomerulonephritis, RCC, obstetric uropathy	History of recurrent UTI/ pyelonephritis.	Urine dipstick Urine protein:creatinine ratio (uPCR)/urine albumin:creatinine (uACR) Renal USS — morphology and symmetry	↑Gestational hypertension and PET ↑Risk of renal dysfunction ↑UTI ↑Prematurity, low birthweight.
Coarctation of the aorta	Upper-limb hypertension ± interarm BP difference. Absent/weak femoral pulse, radio-femoral delay, suprasternal murmur radiating through to the back. Concomitant systolic murmur of bicuspid aortic valve Turner syndrome	Echo/MRI	BP monitoring and management - ↑risk of aortic rupture and rupture of cerebral aneurysm. Postpartum surgical correction. Echo-?concomitant bicuspid aortic valve.
Renal artery stenosis/ fibromuscular dysplasia	Peripheral vascular disease, abdominal bruit, resistant HTN	Renal USS with doppler	BP monitoring and management Postpartum surgical correction
Primary hyperaldosteronism	Hypokalaemia, alkalosis (elevated bicarbonate), plasma Na >140, nocturia, polyuria, muscle weakness.	Renal USS ± MRI for adrenal adenoma Renin/Aldosterone ratio difficult to interpret in pregnancy, wait until postpartum for accurate testing if BP allows	Monitor and replace K+. Spironolactone C.I. as antiandrogenic fetal effects. Limited data on eplerenone and amiloride in pregnancy.
Cushing's disease/syndrome	Truncal obesity, easy bruising, proximal weakness and striae. Development of gestational diabetes	Gestational increase in cortisol causes difficulty in interpretation — wait until postpartum	↑Risk of gestational diabetes
Phaeochromocytoma/ paraganglioma	Labile BP, intermittent hypertension, postural hypotension, headaches, sweating, palpitations, abdominal pain and anxiety	Plasma and urine metanephrines (unchanged in pregnancy)	Treat with α-blockade e.g. phenoxybenzamine/doxazosin until postural hypotension + liberal salt and fluid intake for re-expansion of plasma volume May require β-blockers for tachycardia but only after adequate α-blockade. Unopposed α-stimulation can lead to hypertensive crisis. Genetic testing
Hyper/hypothyroidism	Hyper — tremor, anxiety, sweating, weight loss, diarrhoea, heat intolerance. Hypo — increased diastolic BP, fatigue, weight gain, dry skin, hair loss, constipation, and muscle weakness.	Thyroid function tests: TSH, T3, T4, anti-TPO, TSH-R antibodies.	Hyper — PTU/carbimazole β-Blockers — (propranolol) symptomatic relief. Thyroidectomy Hypo — Levothyroxine If left untreated, associated with miscarriage, preterm birth, low birthweight, PET and/or stillbirth.
Hyperparathyroidism	Related to hypercalcaemia: nausea constipation, polydipsia, fatigue, depression, renal impairment and cardiac arrythmias.	Serum calcium and parathyroid hormone USS	Fluid replacement (oral/IV), low calcium diet and Vit D supplementation Calcitonin and cinacalcet - limited safety data and bisphosphonate therapy risk of

(*continued on next page*)

Table 3 (*continued*)

Secondary cause	History, signs, and symptoms	Diagnosis	Implication for pregnancy
			adverse effects on fetal skeletal development Associated with miscarriage, IUD, maternal pancreatitis, renal dysfunction and PET Parathyroidectomy during pregnancy. Genetic testing
Drugs	Liquorice, herbal medications, alcohol, cocaine/amphetamines Ciclosporin COCP Steroids Erythropoietin NSAIDs ADHD meds e.g. methylphenidate. Venlafaxine	History and urine Tox screen	Dependent on drug used: See Best Use of Medicine in Pregnancy (BUMPS)
Obstructive sleep apnoea	Daytime somnolence Snoring ± apnoeic episodes during sleep High BMI	Epworth sleep score. Sleep study	↑Pre-eclampsia ↑Diabetes

Table 3

and can be used to risk stratify women presenting with new hypertension in pregnancy. The diagnostic thresholds are dependent on the assay used.

Management of new onset hypertension in pregnancy

There is debate, and international differences, as to when hypertension in pregnancy should be treated. In the UK, NICE recommends pharmacological treatment of blood pressure ≥140/90 mmHg. The Control of Hypertension in Pregnancy Study (CHIPS) trial randomised women with non-severe pregnancy hypertension to a diastolic blood pressure (dBP) target of 100 mm Hg ("less tight" control) versus 85 mm Hg ("tight" control). Although this study showed no effect of tightly controlled BP (133/85 mmHg) versus less tightly controlled BP (139/90 mmHg) on rates of PET or maternal/perinatal mortality, it did show a reduction in severe hypertension (28% versus 41%). Post hoc analysis found that severe hypertension was associated with worse maternal and perinatal outcomes independent of PET including birth weight <10th percentile, PET, early delivery, thrombocytopenia, elevated liver enzymes and maternal length of stay. These results were similar to those of a systematic review of 3485 pregnant women across 31 trials which showed treatment of mild to moderate BP between 140 and 169/90−109 mmHg halved the risk of severe maternal hypertension importantly with no adverse fetal outcomes, but no impact on the risk of PET. The CHAP (Chronic Hypertension And Pregnancy) study showed targeting a blood pressure of less than 140/90 mmHg was associated with better pregnancy outcomes compared to only treatment for severe hypertension. Severe hypertension in pregnancy should always be managed and treated in hospital regardless of the timing of its occurrence in pregnancy due to the related maternal and fetal morbidity.

Most guidelines recommend the use of parenteral or oral antihypertensives including labetalol, modified-release nifedipine, hydralazine and methyldopa, alongside magnesium sulphate in those with pre-eclampsia for its anticonvulsant properties. Immediate-release nifedipine is a short-acting calcium channel blocker and should *not* be used for BP control due to the risk of CV events including myocardial infarction, arrhythmias, and stroke. Antihypertensive treatment options can be seen in Table 4. Once an antihypertensive has been started, the BP target varies in international guidelines. NICE guidelines recommend a tighter target of <135/85 mmHg for all hypertensive pregnancies regardless of the starting blood pressure and/or underlying pathophysiology. This may be appropriate for pregnant people whose pre-pregnancy BP was near normal but may be deleterious for those with pre-existing hypertension who blood pressure has been chronically higher unless affected by superimposed pre-eclampsia.

In pre-eclampsia with severe hypertension, due to the increased risk of stroke and end organ damage, treatment should be expeditious and aim to reduce BP to <160/110 mmHg within hours and maintain a target blood pressure of 110−140/70−85 mmHg. It is important to appreciate controlling blood pressure in pre-eclampsia does not impact on the multi-organ complications of pre-eclampsia e.g. thrombocytopenia or fetal involvement. For those with chronic hypertension, without superimposed pre-eclampsia, treatment can mirror that of the non-pregnant population i.e. reduce sBP by 25% at most in the

Antihypertensive therapy (oral or intravenous) for pregnancy and postpartum

Drug	Total dose	Women side effects	Baby side effects
Oral medications			
Labetalol	Total dose: 200—2400 mg Freq: TDS	Headaches (1:10) and shortness of breath. Avoid in asthmatics	Possible temporary low blood sugars after birth
Modified-release nifedipine	Total dose: 20—90 mg Freq: BD	Headache (1:10)	None known
Amlodipine	Total dose: 5—10 mg Freq: OD	Ankle swelling	Limited data for pregnancy but no evidence of harm
Methlydopa	Total dose: 500—3000 mg Freq: TDS	Low mood and tiredness. Avoid in postnatal period/ patients with depression	None known
Doxazosin	Total dose: 2—16 mg Freq: BD	Postural hypotension	Limited data for pregnancy but no evidence of harm
Intravenous medications			
Labetalol	Bolus: 20—50 mg over 1—2 minutes repeated every 10 mins to max. dose 200 mg. Infusion: 20 mg/hour titrated as required every 30 mins to a max. dose of 160 mg/hour until BP controlled	Avoid in asthmatics	Invasive BP monitoring Consider other PET therapy: IV Magnesium Sulphate Fluid restriction
Hydralazine	Bolus: 5 mg over 10 mins, repeated in 20—30 mins if required. Infusion in required: 5 mg/hour, titrated to blood pressure	Hypotension: consider 500 ml crystalloid before or at the same time as first IV dose.	Invasive BP monitoring
Post partum			
Enalapril	Total dose: 2.5—20 mg Freq: BD	Acute kidney injury Hyperkalaemia	Do not use in pregnancy as 1st trimester teratogenesis. Later oligohydramnios, fetal and neonatal renal failure.

Table 4

first hour, aiming for a reduction to <160/110 mmHg over 2—6 hours with recurrent clinical assessment.

The management of severe hypertension with a concomitant emergency e.g. subarachnoid haemorrhage, aortic dissection or myocardial infarction is not covered in this paper and further reading of hypertensive crises management can be found in the suggested reading.

Approach to new onset hypertension occurring postpartum

Towards the end of pregnancy, blood pressure gradually rises to pre-pregnancy values, and postpartum there is a blood pressure peak at day 3—6 in both normotensive women and those affected by hypertension in pregnancy. This peak on average is 6 mmHg systolic and 4 mmHg diastolic in normotensive women, but 12% will have a diastolic above 100 mmHg. This can be accounted for by the resolution of pre-pregnancy haemodynamic adaptations to pregnancy, but also transient causes include pain, medications and excess salt and fluid administration during delivery. Hypertension secondary to pre-eclampsia or HELLP may occur for the first time in the postpartum period and healthcare professionals caring for pregnant and postpartum women should be aware of this.

The hypertensive diagnostic threshold remains the same at ≥140/90 mmHg in the postnatal period. The choice of treatment is wider in the postpartum period and can include medications e.g. ACE-i, that would not be used during pregnancy. Treatment with a once daily regimen is preferred to help with concordance in the postnatal period.

The most recent MBBRACE-UK reported a case of maternal death from postpartum haemorrhagic stroke secondary to hypertension. She had no blood pressure measurements taken during her 5 postpartum visits, and although NICE Hypertension in pregnancy guidelines advise on BP measurement for women who have developed hypertensive disorders in pregnancy, assessment of new onset hypertension in the postnatal period is lacking. All women at post-natal discharge should be informed of the signs and symptoms of hypertensive disorders including pre-eclampsia and eclampsia, which can occur for the first time postnatally, to encourage early access for clinical review. Symptoms include persistent headache, visual disturbance such

as blurring, nausea and vomiting or increased/new swelling of the face, hands, or feet.

Postpartum care for all women with new onset hypertension in pregnancy

Women who develop hypertension in pregnancy have an increased risk of chronic hypertension in the postpartum period and lifelong increased risk of cardiometabolic disease. Most new onset hypertensive disorders of pregnancy resolve within the first few days to 1 week of delivery. However, those who required a longer duration of antihypertensive treatment, have higher sBP/dBP or BMI and/or those with previous chronic hypertension tended to have more sustained hypertension. Approximately 1 in 5 women with hypertension in pregnancy have persistently raised blood pressure requiring antihypertensive therapy.

Women should be informed, at an appropriate time, of their increased risk of long-term cardiometabolic disease and ideally would have prospective monitoring and treatment of any developing CV risk. Intensive blood pressure monitoring and management in the immediate postpartum period has a long-term positive impact on blood pressure control.

Conclusion

New onset hypertension in pregnancy remains a common complication in pregnancy and causes significant associated morbidity and mortality for both mother and baby. Due to increased multi-morbidity amongst women of child-bearing age and pregnant women, alongside reproductive techniques which make pregnancy possible, there is an increasing number of pregnancies affected by hypertension during pregnancy. The mainstay of treatment has been unchanged with antihypertensive therapy, magnesium sulphate and delivery of the baby for decades. Despite education and updates in guidelines, detection of hypertension and its complications remains difficult. Ensuring that women are informed of the signs and symptoms of new onset hypertension in pregnancy is paramount to aid detection and treatment. Improved postnatal care focussing on BP measurement alongside modification of CV risk will aid in reducing the long-term cardiometabolic disease related to these conditions. ◆

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Practice points

- New onset of hypertension in pregnancy requires a full history, examination, and investigations to delineate an underlying diagnosis, target organ damage and cardiovascular risk
- Hypertension occurring at ≤20 weeks' gestation is usually due to chronic hypertension, which may be primary (90% of cases) or secondary
- Hypertension occurring at ≥20 weeks' gestation is usually due to gestational hypertensive disorders such as gestational hypertension or pre-eclampsia
- Treatment of severe blood pressure should assess for presence or absence of pre-eclampsia to assign risk and speed of BP-lowering therapy
- Women who develop hypertensive disorders in pregnancy have an increased long-term cardiovascular risk. BP monitoring and reduction of CV risks should be undertaken to improve overall long term health