Hypertension And Pregnancy

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Introduction

- HDP are strongly associated with maternal and fetal/neonatal complications and are a leading cause of pregnancy-associated mortality. HDP are increasingly common with the highest prevalence experienced by women aged ≥35 years and women with obesity.

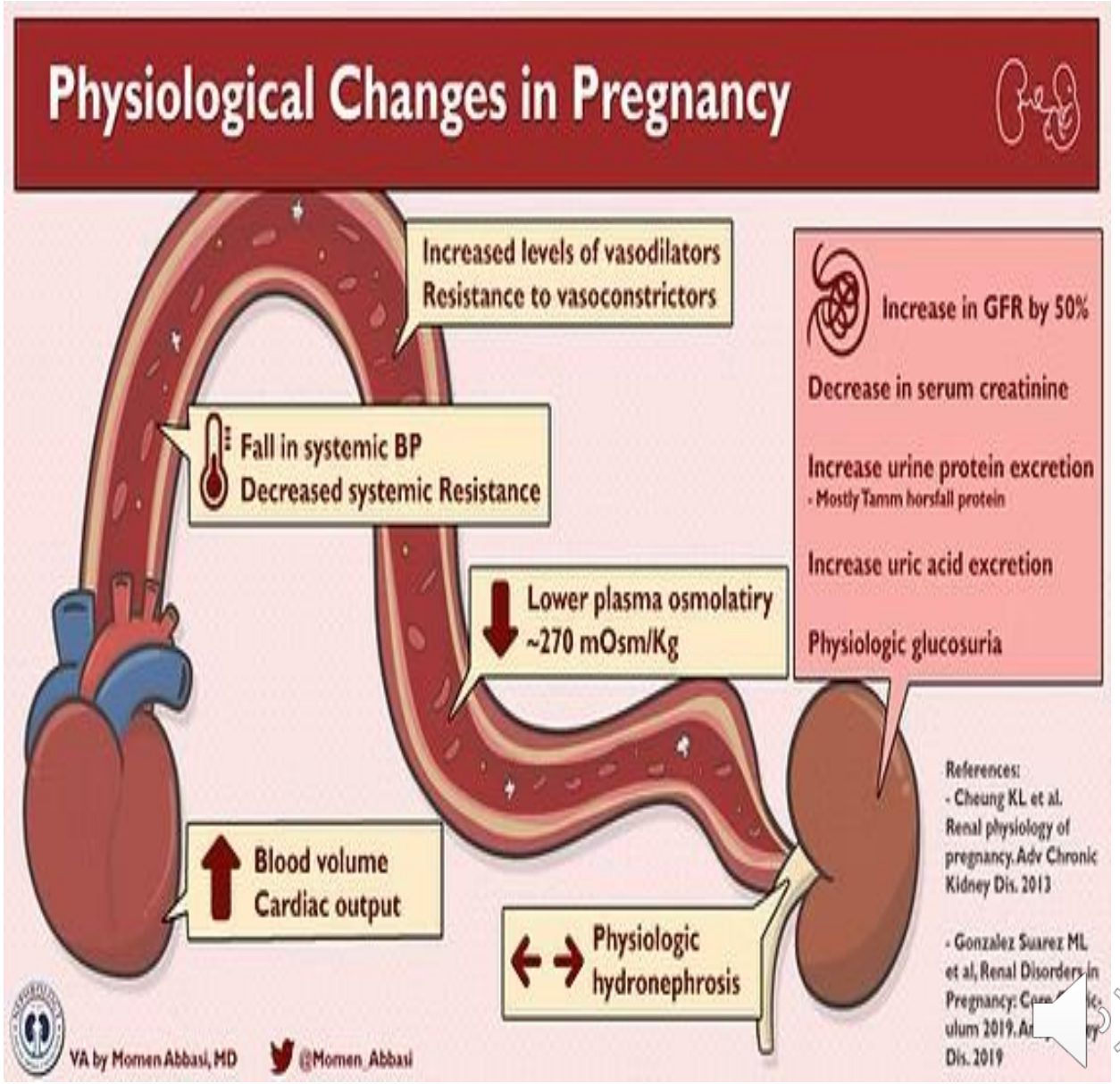
- The management of BP in pregnancy-capable individuals requires special considerations. The overarching goals of antihypertensive treatment during pregnancy are aimed at preventing severe hypertension and preeclampsia and optimizing maternal and fetal/neonatal clinical outcomes.
- Women with a history of hypertensive disorders during pregnancy are at increased risk of subsequent hypertension and CVD.

Physiologic changes in pregnancy

Table 58.1 Physiologic Changes in Pregnancy

Physiologic Variable	Change in Pregnancy
Hemodynamic Parameters	
Plasma volume	Increases 30%-50% above baseline due to RAAS activation, progesterone competitively inhibiting aldosterone, reduced set point for ADH release, and sodium retention of up to 1000 mmol total body sodium
Blood pressure	Decreases by approximately 10 mm Hg below prepregnancy level, with nadir in second trimester around 20 weeks
	Gradually increases toward prepregnancy levels from 35 weeks
Cardiac output	Increases 30%-50%
Heart rate	Increases by 15-20 beats/min
Renal blood flow	Increases to 50%-80% above baseline; afferent and efferent arteriole vasodilation
	Increased sensitivity to RAAS
Glomerular filtration rate	150-200 mL/min (increases to 40%-50% above baseline)
Structural changes	Physiologic collecting system dilation and mild hydronephrosis

Brenner Rectors the kidney



Serum Chemistry and Hematologic Changes

Hemoglobin	Decreases by an average of 2 g/L (from 13 to 11 g/L) owing to plasma volume expansion despite increase in red blood cell mass due to increase in erythropoietin
Serum creatinine	Decreases to a nadir of 0.4–0.5 mg/dL in the second trimester and rises back to nonpregnant levels by the third trimester; levels of 0.81–0.87 mg/dL in the second to third trimester represent kidney impairment
Uric acid	Decreases to a nadir of 2.0–3.0 mg/dL by 22–24 weeks due to reduced reabsorption, then increases back to nonpregnant levels toward term
pH	Increases slightly to 7.4–7.45
Partial pressure of carbon dioxide (pCO_2)	Decreases by approximately 10 mm Hg to an average of 27–32 mm Hg
Calcium	Increased calcitriol stimulates increases in both intestinal calcium reabsorption and increased urinary calcium excretion
Serum sodium	Decreases by 4–5 mEq/L below nonpregnancy levels despite increase in total body sodium
Serum osmolality	Decreases to a new osmotic set point of approximately 270 mOsm/kg with reduced ADH (vasopressin) release
Urinary protein	Physiologic proteinuria up to urine protein-to-creatinine ratio of 30 mg/mmol

Brenner Rectors the kidney

Definition

- Compared with the diagnostic criteria for hypertension in adults, the American College of Obstetricians and Gynecologists (ACOG) defines hypertension in pregnancy as an SBP ≥140 mm Hg or DBP ≥90 mm Hg on 2 occasions at least 4 hours apart.
- severe-range hypertension as sustained SBP ≥160 mm Hg or DBP ≥110 mm Hg with verification in 15 minutes to avoid treatment delays .

Condition	Definition
Hypertension in pregnancy	SBP ≥140 mm Hg and/or DBP ≥90 mm Hg
Severe-range hypertension	SBP ≥160 mm Hg and/or DBP ≥110 mm Hg

ACOG Diagnostic Criteria for Hypertension in pregnancy

- Pregnant individuals with elevated BP are further classified as having 1 of the HDP based upon gestational age at diagnosis and the presence of target organ involvement .

- Vascular and hemodynamic alterations in pregnancy result in a decline of BP by 10% in early pregnancy, reaching a nadir in the second trimester and slowly rising back to baseline by the end of the third trimester. It is because of these alterations that the classification of HDP depends on gestational age, and the use of BP monitors in pregnancy is advised.

Measuring blood pressure

- Monitoring BP during pregnancy is typically done at antenatal visits, which vary dependent on trimester (with increasing frequency towards term). BP tends to reach a nadir at 20–30 weeks of pregnancy before increasing towards term at 40 weeks.
- Only a small number of automated oscillometric BP monitors have been adequately validated in pregnancy and several have failed, usually due to providing BP values that are erroneously high.
- Auscultatory measurement with sphygmomanometry is consequently the clinical standard in pregnancy.
- Self-monitoring at home is not yet proven to be effective in gestational hypertension.

Classification of Hypertensive Disorders of pregnancy

Condition	Definition
Chronic hypertension	Diagnosis prior to pregnancy or at <20 wks' gestation
Gestational hypertension	De novo hypertension at ≥20 wks' gestation in the absence of proteinuria or other signs of preeclampsia
Preeclampsia	Gestational hypertension with proteinuria or other maternal end-organ dysfunction including neurologic findings, pulmonary edema, hematologic findings, acute kidney injury, hepatic dysfunction (Section 5.5.2 "Preeclampsia and Eclampsia, Including Preeclampsia Superimposed on Chronic Hypertension")
Preeclampsia superimposed on chronic hypertension	Preeclampsia in a woman with a history of hypertension before pregnancy or before 20 weeks' gestation

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Chronic Hypertension

- Chronic hypertension, defined as high BP that pre-dates pregnancy or is diagnosed before 20 weeks' gestation, persists for >6 weeks post-partum, and may be associated with proteinuria.

- it is associated with a high risk of developing preeclampsia.

Gestational hypertension

Gestational hypertension is the de novo development of hypertension after 20 weeks' gestation in the absence of new proteinuria or target organ damage.
IT usually resolves within 6 weeks post-partum

Gestational hypertension is associated with an increased risk of maternal and fetal/neonatal adverse events, and up to 30% of women with gestational hypertension ultimately develop preeclampsia.

Following delivery, individuals with gestational hypertension have an increased risk of future hypertension and CVD.

Pre eclampsia and preeclampsia superimposed chronic HTN

- Preeclampsia, a multiorgan system inflammatory syndrome, is an HDP characterized by hypertension, as well as proteinuria or target organ dysfunction.
- Preeclampsia also develops in 20% to 50% of individuals with chronic hypertension and is termed superimposed preeclampsia in that scenario,

  which often presents as an increase in baseline hypertension or proteinuria.

Diagnostic Criteria for Pre-eclampsia

Blood pressure	SBP ≥140 mm Hg or DBP ≥90 mm Hg on 2 occasions at least 4 h apart after 20 wks of gestation in a woman with previously normal BP *or* SBP ≥160 mm Hg or DBP ≥110 mm Hg (severe hypertension can be confirmed within a short interval [min] to facilitate timely antihypertensive therapy).
AND	
Proteinuria	≥300 mg per 24-h urine collection (or this amount extrapolated from a timed collection) *or* Protein/creatinine ratio ≥0.3 *or* Dipstick reading of 2+ (used only if other quantitative methods are not available)
OR in the Absence of Proteinuria, New Onset Hypertension With the New Onset of Any of the Following:	
Thrombocytopenia: Platelet count <100 × 10^9/L	
Renal insufficiency: Serum creatinine concentrations >1.1 mg/dL or a doubling of serum creatinine concentration in the absence of other renal disease	
Impaired liver function: Elevated blood concentration of liver transaminases to twice normal concentration	
Pulmonary edema	
New-onset headache unresponsive to medication and not accounted for by the alternative diagnoses or visual symptoms	

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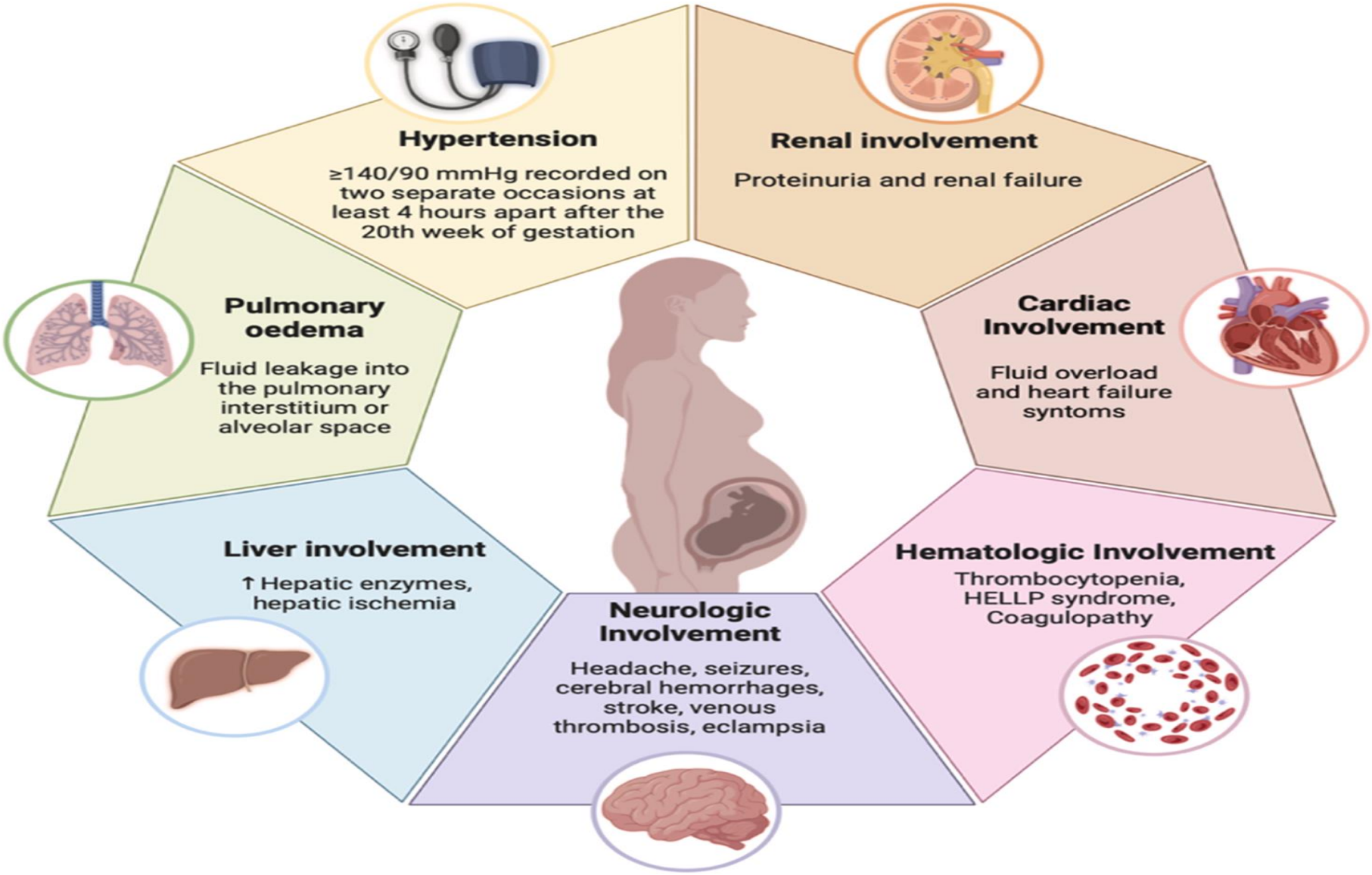
Clinical Feature



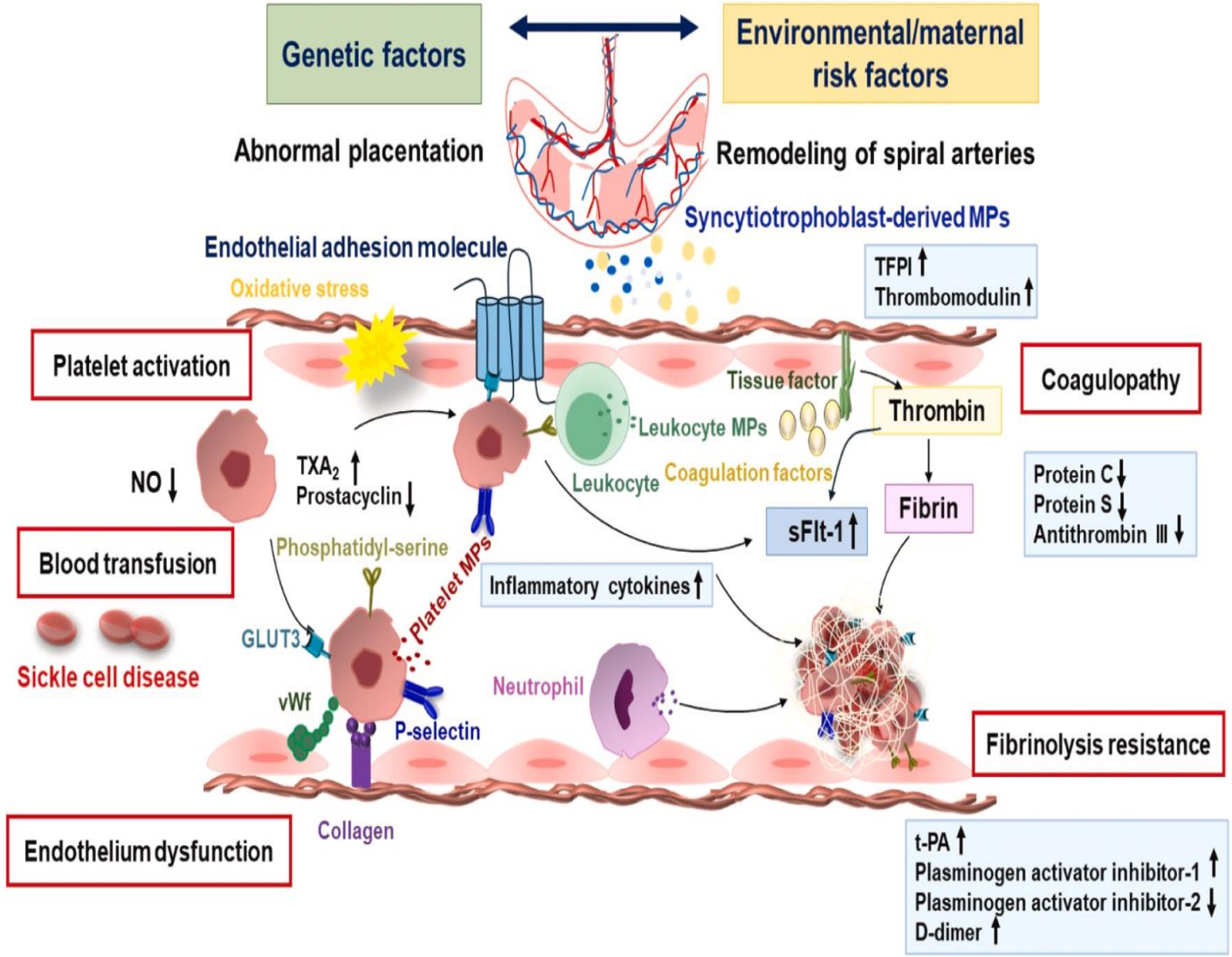
Preeclampsia: Insights into pathophysiological mechanisms and preventive strategies American Journal of Preventive Cardiology 23 (2025) 101054

- Of note, proteinuria is not mandatory for diagnosing preeclampsia but is present in about 70% of cases.
- Also, as proteinuria may be a late manifestation of pre-eclampsia, it should be suspected when de novo hypertension is accompanied by headache, visual disturbances, abdominal pain, or abnormal laboratory tests, specifically low platelets and/or abnormal liver function.

- In an individual with preeclampsia, the development of severe features or hemolysis, elevated liver enzymes, and low platelet count (HELLP) syndrome are associated with increased rates of maternal and fetal/neonatal morbidity and mortality.
- Eclampsia, the occurrence of convulsive seizures, is one of the most severe forms of preeclampsia.
- Both preeclampsia and eclampsia can occur before, during, or after delivery,

Pathophysiology of preeclampsia

- A complex combination of genetic and environmental/maternal risk factors are involved in the development of preeclampsia.
- Circulating syncytio trophoblast-derived MPs reveal placental injury and trigger hypercoagulability. Preeclamptic women exhibit high levels of MPs derived from platelets, endothelial cell, leukocytes, and monocytes. Endothelial dysfunction leads to platelet aggregation and adhesion.
- Elevated levels of activated platelets enhance platelet-leukocyte aggregation, resulting in a inflammatory and prothrombotic state.
- Transfusing erythrocytes promote thrombosis in preeclamptic women.



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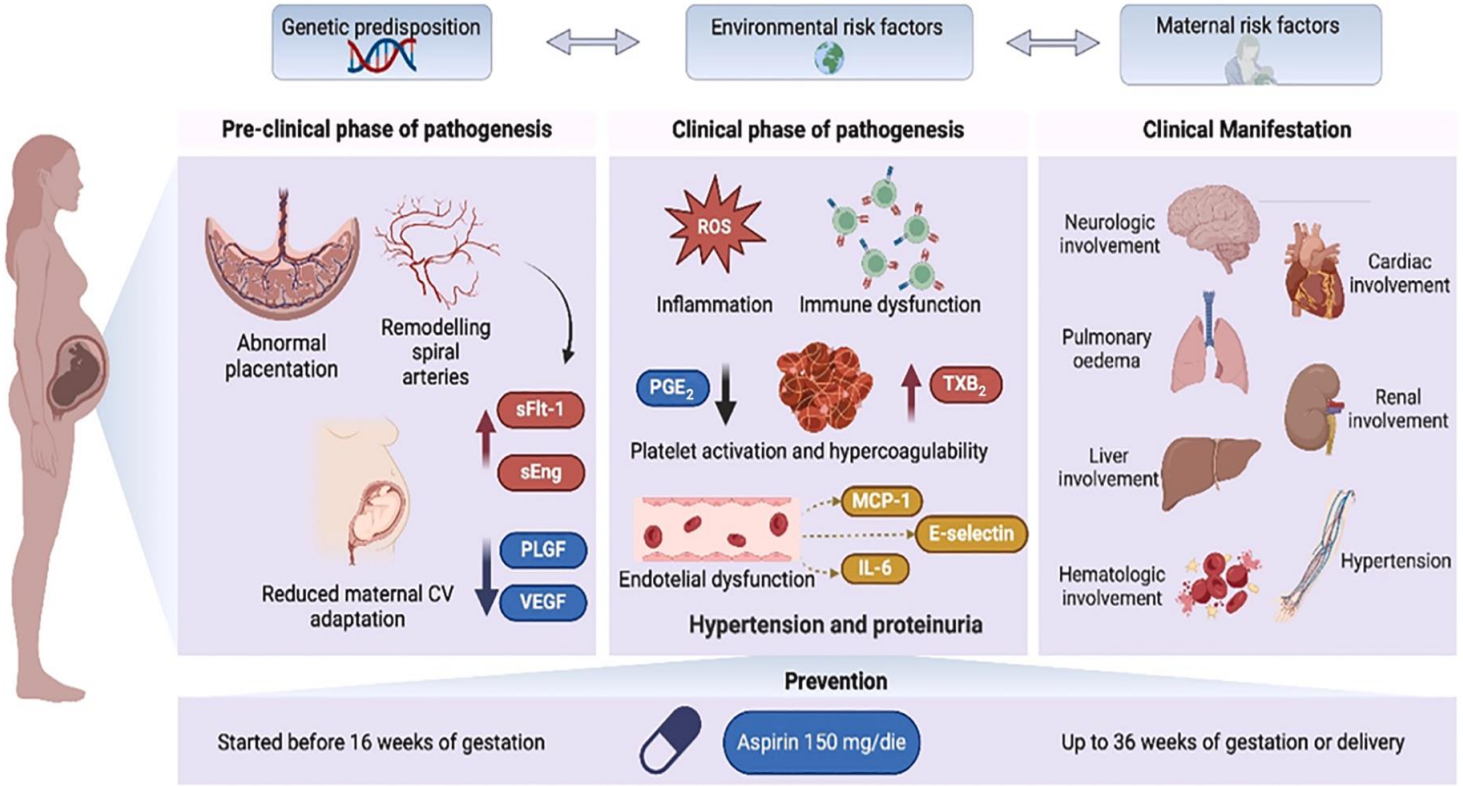
Pathogenesis

The release of antiangiogenic markers such as soluble fms-like tyrosine kinase-1 (sFlt-1) and soluble endoglin (sEng) in maternal circulation results in inflammation, immune dysfunction, hypercoagulability, platelet and endothelial dysfunction exerting a negative impact on maternal and fetus organ systems and causing hematological complications.



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Dx

- The measurement of the antiangiogenic markers soluble fms-like tyrosine kinase -1 (sFlt-1), placental growth factor (PlGF), and their ratio is emerging as a diagnostic test with high negative predictive value to rule out preeclampsia.
- To date, the heterogeneity in prospective studies limits definitive conclusions about their clinical utility for diagnosing preeclampsia.

Rx of pre -eclampsia

- The only cure for pre-eclampsia is delivery, which is recommended at 37 weeks' gestation, or earlier in high-risk cases.
- Most international societies, including the ESC, recommend an intensive approach to BP lowering in pre-eclampsia.
- In women with preeclampsia and severe hypertension, immediately reducing systolic BP to <160 mmHg and diastolic BP to <105 mmHg using i.v. labetalol or nicardipine (with administration of magnesium sulfate if appropriate and consideration of delivery if appropriate) was recommended in the 2018 ESC/ESH Guidelines and the 2022 ESC

Guidelines for management of cardiovascular disease in pregnancy.

- The objective of treatment is to lower BP within 150–180 min.

- magnesium sulfate in addition to antihypertensive medications are the mainstay of treatment.
- Magnesium sulfate [4 g i.v. over 5 min, then 1 g/h i.v.; or 5 g intramuscularly (i.m.) into each buttock, then 5 g i.m. every 4 h] is recommended for eclampsia treatment but also for women with pre-eclampsia who have severe hypertension and proteinuria or hypertension and neurological symptoms or signs.
- There is a risk of hypotension when magnesium is given concomitantly with nifedipine.
- If BP control is not achieved by 360 min despite two medications, consulting critical care is recommended for intensive care unit admission, stabilization, and delivery (if appropriate).
- Since plasma volume is reduced in preeclampsia, diuretic therapy should be avoided.

Recommendations for acutely managing blood pressure in patients with severe hypertension in pregnancy and pre-eclampsia

Recommendation	Class[a]	Level[b]
In pre-eclampsia or eclampsia with hypertensive crisis, drug treatment with i.v. labetalol or nicardipine and magnesium is recommended.[971]	I	C
In pre-eclampsia or eclampsia associated with pulmonary oedema, nitroglycerin given as an i.v. infusion is recommended.[242]	I	C
In severe hypertension in pregnancy: • drug treatment with i.v. labetalol, oral methyldopa, or oral nifedipine is recommended. Intravenous hydralazine is a second-line option.[666–668,969,971]	I	C

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Antihypertensive Agents Used for Urgent Blood Pressure Control in Pregnancy

Drug	Dose	Comments	Onset of Action
Labetalol	10-20 mg IV, then 20-80 mg every 10-30 min to a maximum cumulative dosage of 300 mg; *or* constant infusion 1-3 mg/min IV	Tachycardia is less common with fewer adverse effects. Avoid in women with asthma, preexisting myocardial disease, decompensated cardiac function, and heart block and bradycardia.	1-2 min
Hydralazine	5 mg IV or IM, then 5-10 mg IV every 20-40 min to a maximum cumulative dosage of 20 mg; *or* constant infusion of 0.5-10 mg/h	Higher or frequent dosage associated with maternal hypotension, headaches, and abnormal fetal heart rate tracings; may be more common than other agents.	10-20 min
Nifedipine (immediate release)	10-20 mg orally, repeat in 20 min if needed; then 10-20 mg every 2-6 h; maximum daily dose is 180 mg	May observe reflex tachycardia and headaches.	5-10 min

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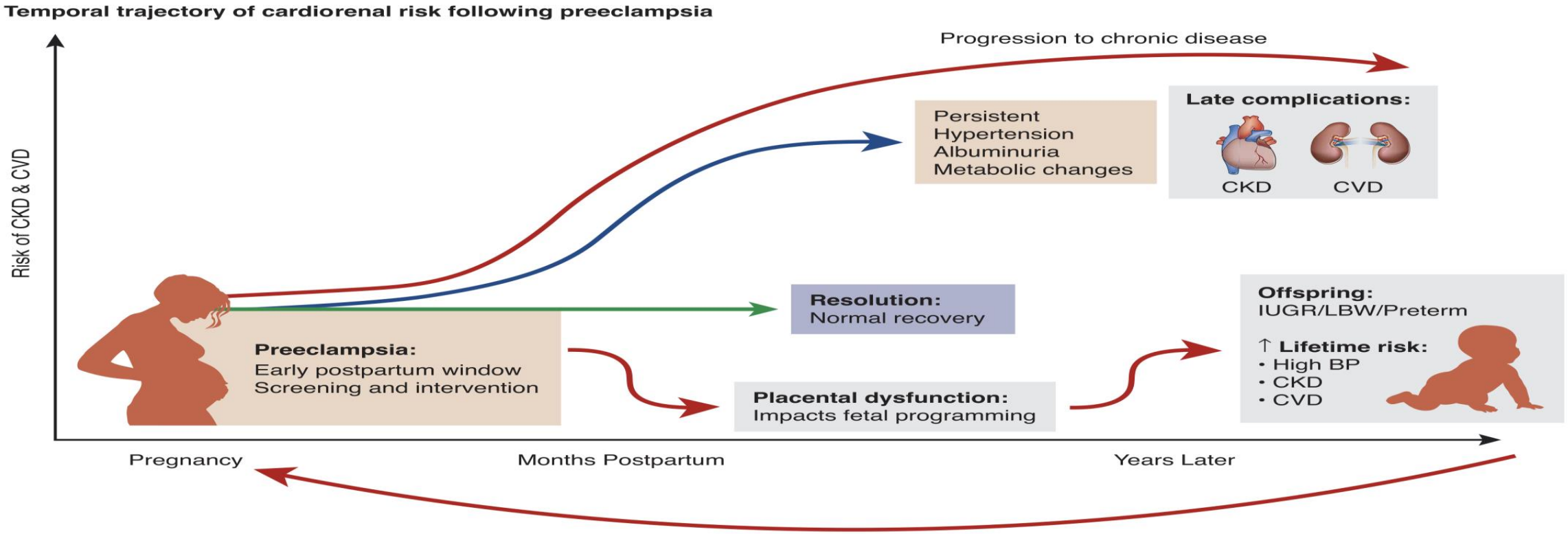
Preventing hypertension and pre-eclampsia

- Low-to-moderate-intensity exercise, especially if supervised and initiated during the first trimester of pregnancy, decreases the incidence of developing gestational hypertension.
- all pregnant women should participate in physical activity, unless contraindicated.
- Women at high or moderate risk of pre-eclampsia should be advised to take 100–150 mg of aspirin daily at bedtime from gestational weeks 12–36.
- Oral calcium supplementation of 0.5–2 g daily is recommended for preventing pre-eclampsia in women with low dietary intake of calcium(<600 mg daily)

Preeclampsia and the Early Origins of Cardiorenal Disease

Liz Lightstone

JASN 00: 1–3, 2026. doi: https://doi.org/10.1681/ASN.0000001143



Preeclampsia is associated with early postpartum emergence of subclinical abnormalities, including albuminuria, hypertension, and metabolic changes, which may resolve or persist and progress to CKD and cardiovascular disease over time. In parallel, placental dysfunction and fetal growth restriction are associated with adverse birth outcomes (e.g., IUGR, low birth weight, preterm birth) and higher lifetime cardiorenal risk in offspring, with potential propagation of risk across generations.

Lab test

- Basic laboratory tests:
- urinalysis, blood count, haematocrit, liver enzymes, serum creatinine, and serum uric acid.
- Serum uric acid is increased in pre-eclampsia and identifies women at increased risk of adverse maternal and foetal outcomes in hypertensive pregnancies.
- All pregnant women should be assessed for proteinuria in early pregnancy

(e.g. 11–14 weeks' gestation). A dipstick test of ≥1+ should prompt further investigations, including ACR, which can be quickly determined in a single spot-urine sample.

An ACR of <30 mg/mmol(<0.3 mg/mg) can rule out proteinuria. Higher values should prompt 24 h urine collection.

- Secondary hypertension during pregnancy is associated with an increased risk of adverse outcomes. The most common cause of secondary hypertension during pregnancy is CKD.
- The onset of hypertension during the first trimester, at the peak of human chorionic gonadotropin (HCG) secretion, should prompt consideration of primary aldosteronism.
- Phaeochromocytoma in pregnant women is rare (0.002% of all pregnancies) but highly morbid

Treatment initiation

Meta-analyses have found no evidence for an increased risk for delivering small-for-gestational-age babies in pregnant women with mild hypertension receiving BP-lowering medications

CHAP trial

In the CHAP trial, treating pregnant women with chronic hypertension and BP of ≥140/90 mmHg reduced the occurrence of pre-eclampsia with severe features, and reduced medically indicated pre-term birth <35 weeks, compared with only treating severe hypertension (BP ≥ 160/105 mmHg).

blood pressure targets

- In women with pre-existing and gestational hypertension with and without pre-eclampsia, we recommend lowering BP below 140 mmHg for systolic and to 80–90 mmHg for diastolic BP.
- Evidence to support a BP target as low as 120–129/70–79 mmHg is lacking in pregnancy.

Recommendations for managing hypertension In pregnancy

Recommendations	Class[a]	Level[b]
In women with gestational hypertension, starting drug treatment is recommended for those with confirmed office systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg.[661]	I	B
In pregnant women with chronic hypertension, starting drug treatment is recommended for those with confirmed office systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg.[88,660,661,678]	I	B

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Rx :

- When antihypertensive therapy is indicated in individuals planning a pregnancy or who become pregnant, labetalol and extended-release nifedipine are the preferred first-line agents.

❖B blocker

❖CCBs

❖Methyldopa

Common Oral Antihypertensive Agents in Pregnancy

Drug	Dosage	Comments
Labetalol	200-2400 mg/d orally in 2 to 3 divided doses. Commonly initiated at 100-200 mg twice daily.	Potential bronchoconstrictive effects. Avoid in women with asthma, preexisting myocardial disease, decompensated cardiac function, and heart block and bradycardia.
Nifedipine	30-120 mg/d orally of an extended-release preparation. Commonly initiated at 30-60 mg once daily (extended release).	Do not use sublingual form. Immediate-release formulation should generally be reserved for control of severe, acutely elevated blood pressures in hospitalized patients. Should be avoided in tachycardia.
Methyldopa	500-3000 mg/d orally in 2 to 4 divided doses. Commonly initiated at 250 mg 2 or 3 times daily.	Safety data up to 7 y of age in offspring. May not be as effective as other medications, especially in control of severe hypertension. Use limited by side effect profile (sedation, depression, dizziness).
Hydrochlorothiazide	12.5-50 mg daily	Second- or third-line agent.

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- no specific agent is preferred because there is a lack of data supporting the use of 1 over the other, although nifedipine is dosed once daily, which may improve adherence.
- In a meta-analysis, BB and CCB appear more effective than methyldopa for the prevention of severe hypertension.
- Methyldopa has been associated with an increased risk of post-partum depression and caution is therefore advised both intra-partum and post-partum.

- There are limited data available on the safety of amlodipine in pregnancy, but it does not appear to be associated with a heightened risk of major congenital malformations.

Beta Blockers

- Atenolol has been associated with growth restriction and lower fetal weight and should be avoided in pregnancy.
- This is likely not a class effect, as other beta-1-selective agents like metoprolol have not demonstrated similar associations with growth restriction.
- labetalol is a preferred agent with the most reassuring fetal safety data. Labetalol is a non-selective beta blocker that also acts as an alpha-blocker in higher doses.
- metoprolol and bisoprolol are also considered safe.

Attention

- ACEi, ARB, and direct renin inhibitors are fetotoxic in the second and third trimesters of pregnancy due to their effects on the developing renal system, leading to oligohydramnios and AKI.
- Based on the mechanism of action and data from animal studies, fetal exposure to spironolactone may cause feminization of a male fetus or growth restriction and is generally not recommended, even for individuals with primary aldosteronism. The feminizing effects appear to be dose-dependent.
- There are few human data on nitroprusside safety in pregnancy, but data from animal studies show that nitroprusside crosses the placenta and may lead to fetal cyanide toxicity.

Breast feeding

- All BP-lowering drugs are excreted into breast milk. Except for propranolol, atenolol, acebutolol, and nifedipine, most drugs are excreted in very low concentrations in breast milk.

Managing blood pressure post-partum

- For women with hypertension during pregnancy, BP should be measured within 6 h of delivery and, if possible, daily for at least a week after discharge from the hospital.
- Post-partum hypertension is common in the first week and associated with prolonged hospitalization.
- ACOG recommends a BP check for individuals with an HDP within 3 to 10 days of discharge.

Follow up

- HBPM for postpartum individuals with a history of HDP has been associated with lower BP and improved measures of cardiac structure and function at 6 and 9 months' postpartum compared with usual care.
- Individuals with a history of gestational hypertension and preeclampsia are at increased risk for the development of CKM risk factors, including chronic hypertension and overt cardiovascular and cerebrovascular morbidity and mortality that often occurs prematurely. Much of this increased risk is mediated through the development of chronic hypertension; thus, early detection, diagnosis, and management of hypertension in this high-risk group is essential.

- Women with gestational hypertension, especially those with pre-eclampsia, have higher risk of masked hypertension. BP measurements, ideally including out-of-office measurements, urine analyses, and CVD risk assessment, should at least be performed 6–12 weeks, 6 months, and 12 months post-partum and, after that, annually.

- Postpartum individuals with a history of pregnancy-associated hypertension in whom BP elevations resolve and antihypertensive medications are discontinued are encouraged to have their BP measured at least annually.
- HDP are also recognized as sex-specific risk enhancers that should be taken into consideration when stratifying individuals and discussing the initiation of a statin for primary prevention of CVD.
- A discussion of effective contraception in pregnancy-capable individuals with chronic hypertension being treated with potentially teratogenic medications is essential.
- Individuals with a history of HDP should be educated about the benefits of low-dose aspirin and prescribed low-dose aspirin to be taken starting at 12 weeks of gestation during subsequent pregnancies to reduce the risk of preeclampsia.

Recommendations for Individuals With Hypertension and Pregnancy* Referenced studies that support the recommendations are summarized in the Evidence Table.

COR	LOE	Recommendations
1	A	1. For individuals with hypertension who are planning a pregnancy or who become pregnant, labetalol and extended-release nifedipine are preferred agents to treat hypertension and minimize fetal risk.[1]
1	B-R	2. Individuals with hypertension who are planning a pregnancy or who become pregnant should be counseled about the benefits of low-dose (81 mg/day) aspirin to reduce the risk of preeclampsia and its sequelae.[2]
1	B-R	3. Pregnant individuals with SBP ≥160 mm Hg or DBP ≥110 mm Hg confirmed on repeat measurement within 15 minutes should receive antihypertensive medication (Table 23) to lower BP to <160/<110 mm Hg within 30 to 60 minutes to prevent adverse events.[3–7]
1	B-R	4. Pregnant individuals with chronic† hypertension (defined as prepregnancy hypertension or SBP 140 to 159 mm Hg and/or DBP 90 to 109 mm Hg prior to 20 weeks' gestation) should receive antihypertensive therapy to achieve BP <140/90 mm Hg to prevent maternal and perinatal morbidity and mortality.[1,8,9]
3: Harm	C-LD	5. Individuals with hypertension who are planning a pregnancy or who become pregnant should not be treated with atenolol, ACEi, ARB, direct renin inhibitors, nitroprusside, or MRA to avoid fetal harm.[10–14]

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In women with chronic and gestational hypertension, it is recommended to lower BP below 140/90 mmHg but not below 80 mmHg for diastolic BP.	I	C
Dihydropyridine CCBs (preferably extended-release nifedipine), labetalol, and methyldopa are recommended first-line BP-lowering medications for treating hypertension in pregnancy.	I	C
In consultation with an obstetrician, low- to moderate-intensity exercise is recommended in all pregnant women without contraindications to reduce the risk of gestational hypertension and pre-eclampsia.[654,655]	I	B
Systolic BP ≥160 mmHg or diastolic BP ≥110 mmHg in pregnancy can indicate an emergency, and immediate hospitalization should be considered.	IIa	C
HBPM and ABPM should be considered to exclude white-coat and masked hypertension, which are more common in pregnancy.[679]	IIa	C
RAS blockers are not recommended during pregnancy.[680,681]	III	B

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Thanks