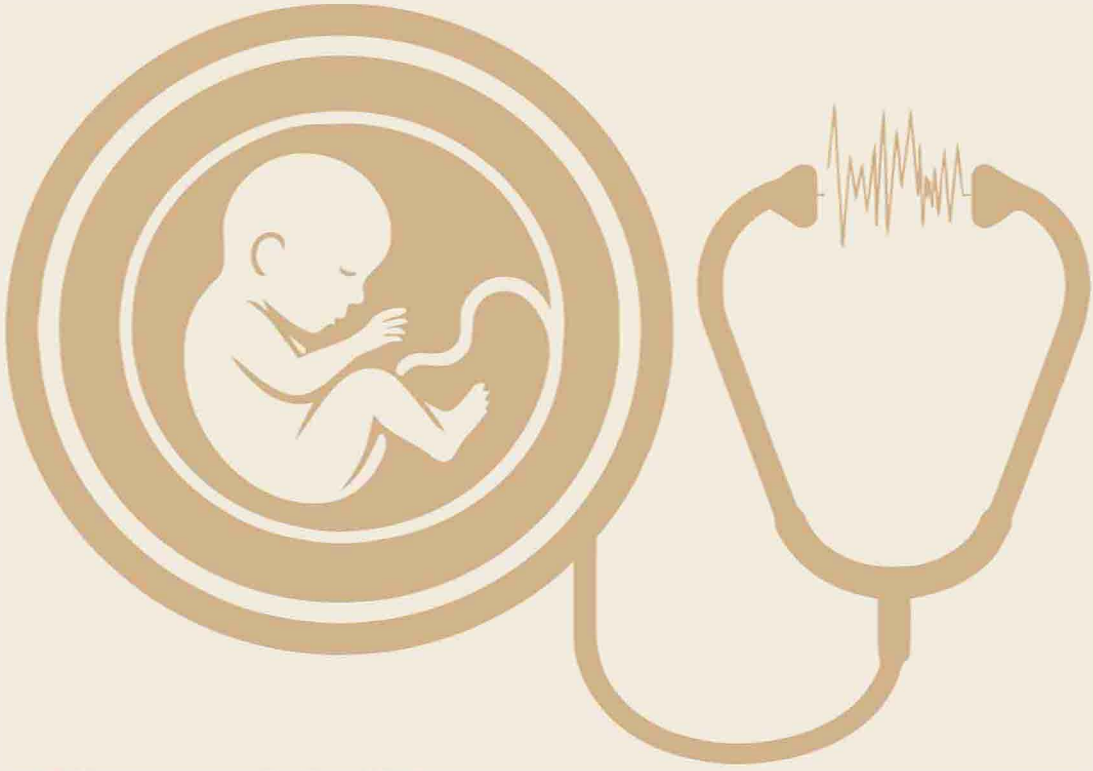
6th Edition

Bedside Clinics in Obstetrics

Clinics to Care with Illustrations

- Fully Revised and Updated as per the Latest Guidelines and Statistics
- Aligned with the new NMC curriculum
- Clinical Cases, Viva Voce, and Practical Examinations
- Richly Illustrated with Clinical Methods, Algorithms, Tables, and Surgical Procedures
- High-yield points for quick revision



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CHAPTER

Preeclampsia and Eclampsia (Hypertensive Disorders in Pregnancy)

Chapter Outline

- History Taking
- HDP-classification
- Preeclampsia: Definition, Types
- Edema, Proteinuria
- Diagnosis
- Complications, Etiopathology, Risk factors, Organ Changes
- Prediction and Prevention of Preeclampsia (PE)
- Management of PE and Severe PE
- Antihypertensive Drugs
- Other Causes of Hypertension
- Diagnosis, Management and Complications of Eclampsia

PREECLAMPSIA

History and examination findings are to be written as in the format given in Section I. The specific points related to this case are mentioned here.

I. HISTORY

- **Patient's particulars:** Teenaged mother, elderly primigravida and low socioeconomic status are the risk factors for preeclampsia.
- **Chief complaints/History of present illness:** Enquire about persistent swelling of legs (ankle joints) or other parts of the body like face, abdominal wall, vulva or whole body and tightness of the ring on the finger.
- Headache, vomiting, pain upper abdomen, visual disturbance and low urinary output are complaints of advanced disease (Ominous symptoms/signs – page 83). M/H: As usual.
- **Present obstetric history:** Time of first detection of hypertension, any history of intake of antihypertensive drugs.
- **Past obstetric history:** History of preeclampsia or eclampsia.
- **Past history:** History of preexisting hypertension, renal disease, diabetes, thyroid disorder.
- **Family history:** Hypertension/preeclampsia.
- **Functional history:** Low urinary output is a late feature.

II. PHYSICAL EXAMINATION

- **Obesity:** Present or not.
- **Weight** (serial weight measurement is more important than a single weight).
- **Edema:** Look for all possible sites—ankle, vulva, abdomen, any facial edema.
- **Pulse:** To be seen in all four limbs.
- **BP:** Measured as outlined on page 9. Sometimes it is needed to be measured in both limbs. In coarctation of aorta, hypertension in upper extremities and normal or low blood pressure (BP) are found in lower extremities.
- **Cardiovascular system:** Examined in detail as described in chapter in "Heart disease."
- **Ophthalmoscopic examination:** Note if findings are given (see page 87 for ophthalmic findings.)
- **Deep tendon reflexes** are assessed for hyperreflexia and the presence of clonus.

III. OBSTETRICAL EXAMINATION

There may be features of intrauterine growth restriction (IUGR) and oligohydramnios in some cases.

IV. INVESTIGATIONS

Along with other routine investigations, *urine for albumin* is the most important test. Any previous urine routine examination and microscopic examination (RE/ME) reports are asked for. Regarding special investigations,

24-hour urinary albumin, serum uric acid, liver function test, platelet count and renal function test reports are to be asked for. Collect ultrasonography (USG) report if available.

V. SUMMARY (SPECIMEN SUMMARY)

Mrs NB, 22 years primigravida married for _____ years coming from low S/E status admitted on _____ with swelling of legs, following amenorrhea of 39 weeks. Her last menstrual period (LMP) was _____ EDD is on _____ . She stated that she first noticed the swellings over both ankle joints at about five months of pregnancy, and that the swellings did not subside in the morning. She attended antenatal clinic regularly from the third month. From 6 month onward, her blood pressure went up and she gained more weight than expected as stated by her attending doctor. She was getting antihypertensive drugs (if she gets) along with iron, folic acid, calcium and bedtime diazepam tablet. She has taken two doses of tetanus toxoid. She has no significant past history. Her father is of good health, but mother is known to be hypertensive and is on medication (if any such history).

On physical examination, her height 5 feet, and her weight is 62 kg. Pallor +, edema ++ at ankle joints and parietals, pulse—80/min, equal on all four limbs, BP—140/92 mm Hg, Respiration—normal. Other cardiovascular system (CVS) examinations are within normal limits.

On obstetrical examination, height of the fundus—corresponds to term size, lie is longitudinal, cephalic presentation, and head not engaged. FHS (+) R—140/min, situated on left spinoumbilical line. Liquor seems to be adequate clinically.

Investigation reports:

- Hb—g%, blood group
- Venereal disease research laboratory (VDRL)—negative
- Urine for albumin +.

Other available investigation reports:
(Other investigations done in preeclampsia—see later).

VI. PROVISIONAL DIAGNOSIS

22 years primi at 39 weeks with preeclampsia.

VII. DIFFERENTIAL DIAGNOSIS

All causes of hypertension other than pregnancy induced hypertension will come in D/D. Some of the causes of hypertension not specific to pregnancy are described in short at the end of this chapter.

What is your diagnosis?
Give the provisional diagnosis: It is a case of preeclampsia at 39 weeks of pregnancy in a 22-year-old primigravida.

What is your case?
Describe the summary of the case.

Why are you saying—this is a case of preeclampsia?
This is a case of preeclampsia because:

- The woman has developed hypertension after 20 weeks of pregnancy detected first at the sixth month.
- Her urine is positive for albumin or
- Evidence of other multiorgan involvement, if any

CLASSIFICATION HYPERTENSION DISORDERS IN PREGNANCY (HDP)

How would you classify hypertension disorders in pregnancy (HDP)?
See **Table 5.1**.

What do you mean by hypertension?
Hypertension is diagnosed when systolic blood pressure exceeds 140 mm Hg and/or diastolic blood pressure exceeds 90 mm Hg.

What do you mean by pregnancy induced hypertension (PIH)?
Pregnancy induced hypertension is a term to describe any new-onset pregnancy-related hypertension. Accordingly, it includes gestational hypertension, preeclampsia and eclampsia.

Define Preeclampsia

What do you mean by preeclampsia?
Preeclampsia is the development of hypertension with proteinuria or with evidence of maternal organ dysfunction (liver, renal, hematological and neurological) after 20 weeks of gestation.

The diagnostic criteria for preeclampsia are given in Table 5.2.

Preeclampsia syndrome: Preeclampsia is best regarded as a pregnancy-specific syndrome which can affect almost all organ systems.

Clinical classification of preeclampsia

- Early onset: <34 weeks
- Late onset: ≥34 weeks
- Preterm onset: <37 weeks
- Term onset: ≥37 weeks

TABLE 5.1: Basic classification of hypertensive disorders of pregnancy (HDP).

Type of hypertensive disorder	*Definition*
Prepregnancy or at <20 weeks:	
Chronic hypertension	Hypertension detected prepregnancy or before 20 weeks' gestation
Essential	Hypertension without a known secondary cause
Secondary	Hypertension with a known secondary cause (e.g., renal disease)
White-coat hypertension	BP of ≥140 and/or DBP ≥90 mm Hg when measured in the clinic or clinic, and BP <135/85 mm Hg using HBPM or ABPM readings
Masked hypertension	BP that is <140/90 mm Hg at a clinic/office visit, but ≥135/85 mm Hg at other times outside the clinic/office
≥20 weeks:	
Gestational hypertension	Hypertension arising de novo at ≥20 weeks' gestation in the absence of proteinuria or other findings suggestive of preeclampsia
Transient gestational hypertension	Hypertension arising at ≥20 weeks' gestation in the clinic, which resolves with repeated BP readings
Preeclampsia	

Note: Gestational hypertension is nonproteinuric pregnancy induced hypertension.
(ABPM: ambulatory blood pressure monitoring; HBPM: home blood pressure monitoring)
Source: From ACOG 2020.

TABLE 5.2: Diagnostic criteria for preeclampsia (ACOG 2020).

Pregnancy associated hypertension	*Criteria*
Blood pressure	• ≥140 mm Hg systolic or more than or equal to 90 mm Hg diastolic after 20 weeks of gestation in a woman with a previously normal blood pressure (on two occasions at least 4 hours apart) • ≥160 mm Hg systolic or more than or equal to 110 mm Hg diastolic, severe hypertension can be confirmed within a short interval (minutes) to administer timely antihypertensive therapy
and	
Proteinuria	• ≥300 mg/24 h (or this amount extrapolated from a timed collection) or • *Protein:* Creatinine ratio ≥0.3 (each measured as mg/dL) or • Dipstick 1+ persistent (recommended only if sole available test)
or, in the absence of proteinuria, new onset hypertension with the new onset any of the followings	
Thrombocytopenia	• Platelet <100,000/μL (100 × 10^9/L)
Renal insufficiency	• Serum creatinine > 1.1 mg/dL or doubling of baseline in absence of prior renal disease
Impaired Liver function	• Serum transaminase levels (AST or ALT) elevated twice the normal
Pulmonary edema	• Present
Cerebral symptoms or visual symptoms	• New onset headache unresponsive to medication and not accounted for by alternative diagnosis or visual symptoms, convulsions

(ALT: alanine aminotransferase; AST: aspartate aminotransferase)

Other methods of diagnosing hypertension in pregnancy?

- An *increase of 30 mm Hg systolic or 15 mm Hg diastolic blood pressure* from midpregnancy is used as a diagnostic criterion of pregnancy induced hypertension even when absolute value is <140/90 mm Hg. However, this increase is no longer recommended as diagnostic criteria due to lack of

strong evidence of adverse pregnancy outcomes. This group of patients should be closely observed because some of these women may experience eclamptic fit even if BP remains below 140/90 mm Hg.

- **MAP:** (By measuring *mean arterial pressure* or *MAP* (as advocated by Page)).

$$\text{MAP} = \frac{\text{Systolic BP} + \text{Diastolic BP} \times 2}{3}$$

When MAP is ≥105 mm Hg or >20 mm of previous values it signifies hypertension.

Delta hypertension

- An acute increase in mean arterial pressure (MAP) later in pregnancy is called "delta hypertension".
- It may signify preeclampsia even when absolute value of BP is <140/90 mm Hg. Some of these women may develop eclampsia or HELLP syndrome.

White coat hypertension

Elevated BP near health care provider but consistently normal in the home environment i.e., <135/85 mm Hg. It is an independent risk factor of cardiovascular disease (chronic hypertension and PE).

Masked hypertension

Elevated BP outside the office but consistently normal in the office.

N.B. Elevated BP means a Blood pressure measurement of 140/90 mm Hg or more.

How do you record blood pressure in proper way?

See (page 9, **Fig. 1.4**) (**Fig. 5.1**).

Fig. 5.1: Recording blood pressure (BP) in semirecumbent posture.

Korotkoff phase V is considered as diastolic blood pressure in pregnancy.

Korrotkoff phase I remains as systolic.

Is edema included in diagnostic criteria for preeclampsia?

No, edema is not a diagnostic criterion for preeclampsia because it occurs in too many (70%) normal pregnant women.

Is overt proteinuria an essential criterion of diagnosis of preeclampsia?

In most of the cases there will be new-onset proteinuria. However, overt proteinuria may not be a feature of preeclampsia syndrome in some women. Evidence of any multiorgan involvement like maternal thrombocytopenia, hepatic, renal, cerebral, visual disturbance and pulmonary edema is sufficient to diagnose preeclampsia even in absence of proteinuria. *This is one remarkable change of diagnostic criteria of preeclampsia in the new and updated ACOG guidelines.*

What do you mean by eclampsia?

Eclampsia is the occurrence of seizures in a woman with preeclampsia, not attributed to other causes.

GESTATIONAL HYPERTENSION

What is gestational hypertension?

- Gestational hypertension is diagnosed when BP reaches 140/90 mm Hg or greater for the first time during pregnancy, after 20 weeks of pregnancy, but in whom **proteinuria has not developed** or **there is no other systemic finding** (as mentioned in diagnosis of preeclampsia).
- BP returns to normal by **12 weeks** postpartum.
- Up to one third of these women subsequently develop preeclampsia syndrome which includes signs like proteinuria and/or thrombocytopenia, or symptoms such as headache or epigastric pain, visual disturbances or deranged liver function test/renal function test.
- Gestational hypertension is reclassified as "**transient hypertension**" when preeclampsia does not develop, and BP returns to normal by 12 weeks after delivery.

Management of gestational hypertension

Routine antenatal management is done with extra vigilance for appearance of any symptom and sign

of preeclampsia. If such develops, manage like preeclampsia.

Otherwise continue the pregnancy till term and there is no indication of earlier induction.

CHRONIC HYPERTENSION

What is chronic hypertension?

Chronic hypertension is hypertension that predates pregnancy. The diagnostic criteria are:

- BP ≥140/90 mm Hg before pregnancy or diagnosed before 20 weeks' gestation not attributable to gestational trophoblastic disease.

If the woman is first seen after midpregnancy, it may be difficult to determine the type of hypertensive disorder. Causes of chronic hypertension are essential hypertension, renal disease, pheochromocytoma, connective tissue disease, and thyrotoxicosis, etc. Some of them are described at the end of this chapter.

What is superimposed preeclampsia (Preeclampsia superimposed on chronic hypertension)?

- New-onset proteinuria ≥300 mg/24 hours in hypertensive women or other findings of organ dysfunction as in **Table 5.2** is diagnosed as superimposed preeclampsia (ACOG 2019).
- Superimposed preeclampsia develops earlier in comparison to pure preeclampsia. It is likely to be more severe type, and fetal growth restriction is common.

CATEGORIZATION OF PREECLAMPSIA ACCORDING TO SEVERITY

How would you categorize preeclampsia according to severity?

There is no consensus on using terminology for grades of severity of preeclampsia syndrome. The different terms mild, moderate, less severe, non-severe and severe are used. ACOG Task Force (2013) discouraged the use of "mild preeclampsia" as it is a progressive disease. A practical classification is "nonsevere" and "severe." The "nonsevere" includes *mild* and *moderate* varieties which are not defined individually.

Severe and nonsevere varieties are summarized below, and some differentiating points are given also in **Table 5.3**.

- ***Severe preeclampsia:*** It is variety of Preeclampsia which is associated with any of the following findings:
  - Systolic BP 160 mm Hg or higher, or diastolic BP of 110 mm Hg or higher on two occasions at least 4 hours interval while the patient is on bed rest (unless antihypertensive therapy is initiated).
  - Thrombocytopenia
  - Impaired liver function test (LFT)
  - Progressive renal insufficiency
  - Pulmonary edema and
  - New onset cerebral or visual disturbances.
- **Nonsevere preeclampsia:** Systolic BP ≥140 but <160 mm Hg or diastolic [≥90 but <110 mm Hg after 20 weeks of gestation in a woman with a previously normal blood pressure (on two occasions at least 4 hours apart). To fulfill the criteria of preeclampsia proteinuria should be present.

[Although severity of preeclampsia is categorized primarily based on degree of hypertension [systolic pressure (SP) ≥160 mm Hg and ≥110 mm Hg] presence of new evidence

TABLE 5.3: Classification of gestational hypertensive disorder of pregnancy according to severity.

Features	*Nonsevere variety*	*Severe variety*
• Systolic BP • Diastolic BP • Proteinuria*	<160 mm Hg <110 mm Hg	≥160 mm Hg ≥110 mm Hg
• Convulsion	May or may not be present	May or may not be present
– New onset-cerebral or visual disturbance	Nil	Present
– Headache	Nil	Present
– Eye symptoms	Nil	Present
• Impaired liver function		
– Elevated liver enzymes (serum transaminases) elevation	Minimal	Marked
– Severe persistent right upper quadrant or epigastric pain is not responding to medication or not due to other disease	Absent	Present
• Progressive renal insufficiency	Absent	Present
– Oliguria (≤400 mL of urine/24 h)		
– Serum creatinine	Normal	Elevated
• Thrombocytopenia (<100,000/μL)	Absent	Present
• Pulmonary edema	Absent	Present

*Massive proteinuria (>5 g) has been eliminated from consideration of preeclampsia as severe as recent studies show minimal relationship between the quantity of urinary protein and pregnancy outcome.

of any of the above organ dysfunctions along with <160 mm Hg or diastolic < 110 mm Hg, should also be considered as having severe disease (ACOG)].

Classification of Gestational hypertensive disorder of pregnancy according to severity is given on Table 5.3.

What are the ominous symptoms or signs of preeclampsia?

Symptoms:

- Severe headache
- Visual disturbance such as blurring or flashing
- Epigastric or right upper quadrant pain
- Vomiting
- Sudden swelling of face hands and feet.

Signs:

- Oliguria
- Thrombocytopenia
- Renal and cardiac involvement.

Causes of swelling of legs (edema) during pregnancy (Fig. 5.2)

Physiological: Most common.

Pathological:

- Preeclampsia/eclampsia
- Hypoproteinemia/malnutrition/severe anemia
- Nephrotic syndrome
- Cardiac failure.

What are the sites to see edema?

- Leg
- Vulva **(Fig. 5.3)**
- Abdomen (parietes)
- Face

What are the characteristics of physiological edema?

- Usually mild, confined to lower limb, more common in right leg due to dextrorotation of uterus.
- Disappears after 12 hours rest.
- Not associated with other features of preeclampsia. (See also page 66)

What are the other causes of edema during pregnancy?

See page 66.

What do you mean by proteinuria? What does it signify? How would you interpret in dipstick? (Fig. 5.4)

- Protein excretion in urine is defined as proteinuria.
- ≥300 mg of protein in 24-hour urine, or persistent urinary protein of >30 mg/dL, or a protein:creatinine ratio of ≥0.3 in a random (spot) urine sample, is

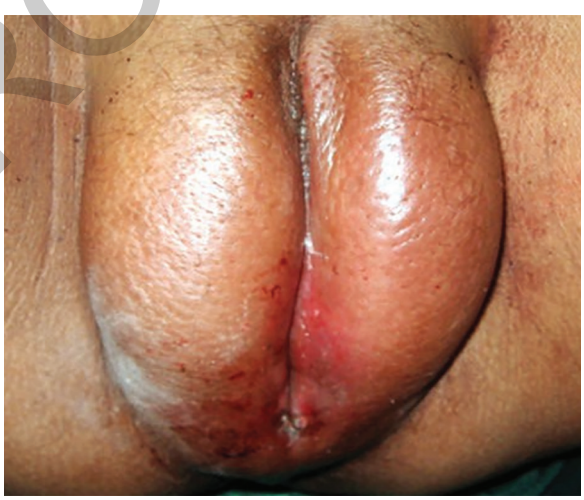


Fig. 5.3: Edema over the vulva.

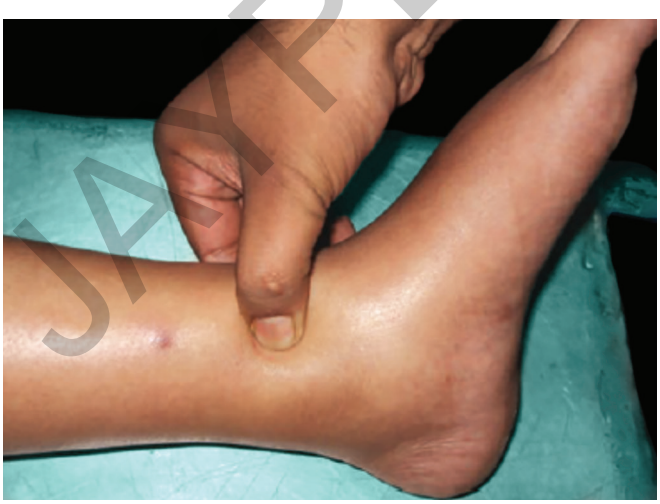


Fig. 5.2: Edema leg.

Fig. 5.4: Urine protein by DIPSTICK.

TABLE 5.4: Interpretation of dipstick (Fig. 5.4).

1	+	=	0.3	g/L	<1.0 g/L
2	+	=	1.0	g/L	<3.0 g/L
3	+	=	3.0	g/L	<10.0 g/L
4	+	=	10.0	g/L	or more

considered significant proteinuria. Where facilities of 24 hours urine examination or spot urine protein creatinine ratio is not available, a simple automated reagent dipstick device value of ≥1+ is considered significant proteinuria.

- Proteinuria signifies impaired renal function.
- Interpretation of dipstick **(Table 5.4)**

Previously, 24-hour urine collection was gold standard for measuring proteinuria. It has problems of being time consuming, inconvenient to woman, incomplete in most cases and delay in diagnosis for which, now spot sample protein creatine ratio or albumin creatinine ratio are recommended to quantity proteinuria.

Enumerate the other causes of proteinuria

- Contamination
- Urinary tract infection
- Renal disease
- Connective tissue disorder
- Orthostatic proteinuria.

What is heat and acetic acid test?

- **Qualitative:** 'Heat and acetic acid test' is a qualitative test by which the presence of protein (albumin) is detected in urine.
- A test tube is filled with urine up to three-fourth, and the upper part of urine is boiled. The boiled part may become cloudy. 5 drops of 10% acetic acid are added to it. Disappearance of turbidity indicates presence of phosphate and if the turbidity persists it indicates presence of albumin. Severity of turbidity signifies the amount of *protein* present in urine. In presence of excess protein (10–15 g/L) urine becomes solid on boiling.
- **Quantitative:** By Esbach's albuminometer, quantitative estimation of albumin is done.

What is the sequence of development of the features of preeclampsia?

Occult edema first (as evidenced by excessive weight gain) → appearance of hypertension → visible edema (pitting edema over ankles) → proteinuria at last (last feature to develop).

State the reasons for development of edema, hypertension and proteinuria in preeclampsia

- **Edema:** Due to extracellular sodium retention.
- **Hypertension:** As a result of vasospasm through a complex mechanism.
- **Proteinuria:** Due to specific changes in kidney—glomerulus endotheliosis resulting escape of protein.

What is the incidence of preeclampsia?

Varies from 5 to 10%, primi—10%, multi—5%.

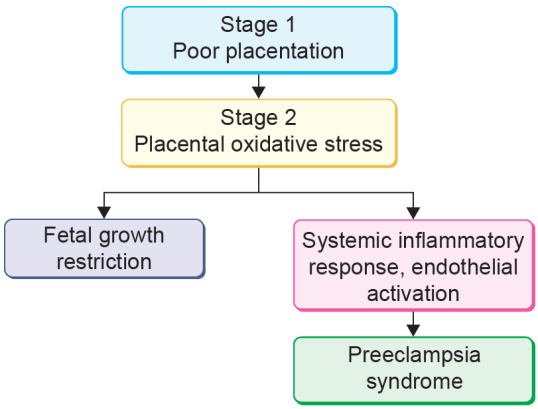
RISK FACTORS

Predisposing factors (risk factors) for development of preeclampsia

- **High risk factors:**
  - Hypertensive disease during previous pregnancy.
  - Preexisting hypertension, chronic renal disease or connective tissue disorder.
  - Autoimmune disease such as SLE and antiphospholipid syndrome.
  - Diabetes Type 1 or 2.
  - Multiple pregnancy.
- **Moderate risk factors:**
  - Nulliparity.
  - Age—35 years or older.
  - Sociodemographic characteristics (low socioeconomic status, African American race).
  - Personal history factors (prior adverse pregnancy outcome, low birth weight or small for gestational age)
  - Pregnancy interval of >10 years.
  - Obesity: BMI >30 kg/m^2.
  - Family h/o hypertension or preeclampsia, 3-4-fold increase in first degree relative of the woman suffering from eclampsia.
  - Polyhydramnios.
- **Other risk factors:**
  - Severe anemia.
  - Hydatidiform mole.
  - Severe Rh-incompatibility.
  - Assisted with reproductive technology.
  - Obstructive sleep apnea.

Predictive Test for preeclampsia, see page 87.

Flowchart 5.1: Development of preeclampsia in two stages.



ETIOPATHOLOGY

Etiological basis of preeclampsia:

It is the **deficient trophoblastic invasion** of spiral arteries which appears to be the etiology and the vascular dysfunction is said to be the prime pathophysiology. The decidual spiral arteries are invaded by trophoblasts in normal pregnancy and the endothelium and muscular wall are replaced by trophoblasts to create **low-resistance** vessels which is the unique feature.

In preeclampsia trophoblast invasion is patchy and the muscular walls are retained in spiral arteries resulting in small caliber vessel with high resistance (See Chapter 45).

Genetic factor and altered immune response may be the reason of this defective invasion.

Development of preeclampsia (Flowchart 5.1)

Preeclampsia is developed in **two stage disease**. In the **first stage** patchy invasion of trophoblasts occurs and the muscular wall of spiral arteries are retained which prevents high flow, low impendence placental circulation resulting in ischemia. In the **second stage** oxidative stress and inflammatory stress occurs due to uteroplacental ischemia. This is associated with involvement of secondary mediators which leads to endothelial dysfunction and vasospasm. Coagulation system is also activated due to this process. This disease process of the endothelial cells spread across multiple areas, thus in true sense preeclampsia involves multiple organs simultaneously making it a multisystem disorder.

State the basic pathophysiology in preeclampsia and eclampsia involving the organs.

In normal pregnancy, there is marked peripheral vasodilatation which causes fall in total peripheral resistance and a reduction in blood pressure in spite of increased cardiac output and blood volume. This vasodilatation is due to reduced vascular sensitivity to vasoconstrictors such as angiotensin and enhanced production of vasodilator substances such as nitric oxide (NO) and prostacyclin (PGI_2) by vascular endothelial cells. In preeclampsia, vessels show enhanced sensitivity to vasoconstrictors and reduced sensitivity to vasodilators that results in characteristic **vasospasm**. In preeclampsia, there is also reduction in synthesis of NO and PGI_2 and increase in production of endothelin–1 by vascular endothelium which not only causes vasospasm, but results in activation of platelets.

Thus, basic abnormalities which are found in preeclampsia are (1) **vasospasm** (2) **endothelial cell dysfunction** with subsequent (c) **platelet activation and microaggregate formation**. These basic changes account for many of the pathological features of preeclampsia and eclampsia.

Outline the pathological changes in vital organs of preeclampsia and eclampsia (Fig. 5.5)

- **Placenta:** Acute atherosis of spiral arteries with platelet microaggregates and larger thrombosis resulting in IUGR and causing uteroplacental insufficiency in labor.
- **Kidney:** Characteristic lesion is *glomeruloendotheliosis* which is comprised of endothelial and mesangial cell swelling, with basement membrane inclusions but little disruption of renal epithelial podocytes. This type of lesion is not seen with hypertension due to other causes. This lesion results in proteinuria, decreased glomerular filtration rate (GFR), decreased renal clearance of uric acid and oliguria. Patients may develop renal failure (acute tubular necrosis and cortical necrosis).
- **Liver:** Subcapsular hemorrhage and periportal necrosis are the characteristic findings which are found in severe form. There are elevated liver enzymes (EL), serum glutamic-oxaloacetic transaminase (SGOT) [aspartate aminotransferase (AST)] and lactate dehydrogenase (LDH). Along with hemolysis (H) and low platelet count (LP), a grave complication, i.e., HELLP syndrome is constituted (See page 89).
- **Central nervous system:** Hypertensive encephalopathy, necrosis, vasospasm, cerebral edema, hemorrhages, posterior reversible encephalopathy syndrome (PRES) and eclampsia.

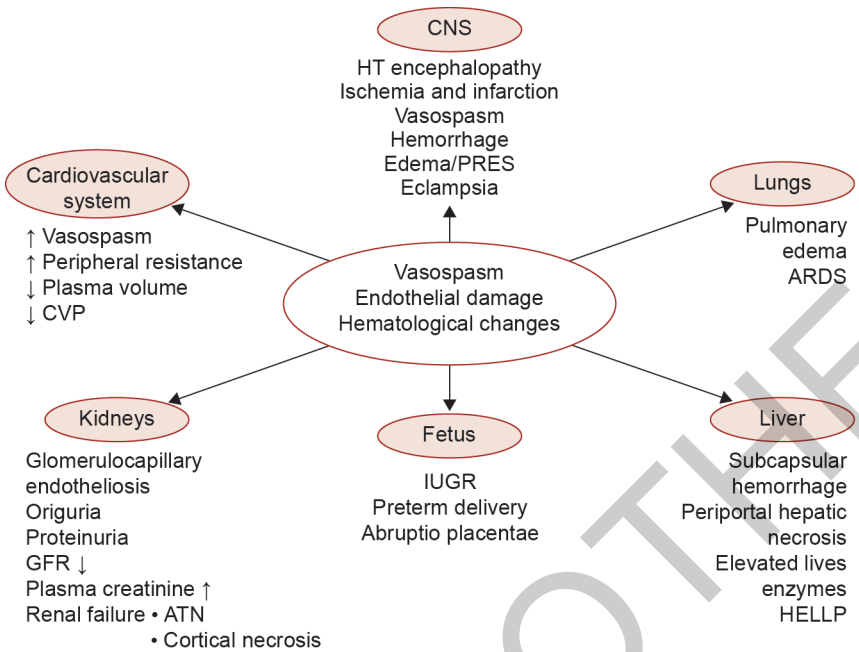


Fig. 5.5: Pathological changes in vital organs in preeclampsia/eclampsia. (ARDS: acute respiratory distress syndrome; ATN: acute tubular necrosis; GFR: glomerular filtration rate; HELLP: hemolysis, elevated liver enzymes, low platelet count; HT: hypertension; IUGR: intrauterine growth restriction; PRES: posterior reversible encephalopathy syndrome)

- **Cardiovascular:** Generalized vasospasm, increased peripheral resistance and reduced central venous pressure and pulmonary wedge pressures.
- **Hematological changes:** Platelet activation, micro-aggregation and depletion, coagulopathy, decreased plasma volume and increased blood viscosity.
- **Water and electrolytes:** There is sodium and water retention. Salt retention increases extracellular fluid volume. The ultimate result is intravascular reduction and extravascular accumulation of fluid resulting in hemoconcentration.

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME

What is PRES? (Fig. 5.6)

PRES refers to posterior reversible encephalopathy syndrome. PRES is mostly found in eclampsia but their incidence in preeclampsia is 20%.

There may be variable neurological symptoms and signs, e.g., headache, altered consciousness, visual abnormalities and seizures. The typical lesion in MRI **(Fig. 5.6)** is seen as hyper-intense lesion (white areas) involving the posterior brain—the occipital and parietal cortices but other areas (basal ganglia, brainstem and cerebellum) may be involved. These lesions represent cerebral vasogenic edema. The lesions are found in severe diseases and patients having neurological symptoms. PRES is usually reversible, but in 25% cases the lesions

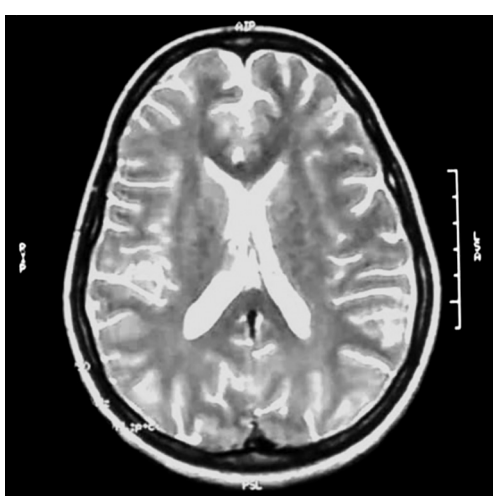


Fig. 5.6: MRI of a primigravida patient with eclampsia with recurrent convulsion showing multifocal patchy T2 FLAIR hyper-intensity areas at bilateral frontal, parietal and temporal cortex—suggestive of PRES. (MRI: magnetic resonance imaging; FLAIR: fluid-attenuated inversion recovery; PRES: posterior reversible encephalopathy syndrome)

may be due to cerebral infarctions with persistence nature.

Treatment includes antihypertensives, antiepileptics and long term neurological follow up.

Eye changes in preeclampsia

What are the eye changes (ophthalmoscopic findings) in preeclampsia?

- There may not be any eye change in mild preeclampsia.
- In severe form (1) retinal edema, (2) arteriolar constriction and changes of ratio of vein and artery from 3:2 to 3:1 and (3) nicking of veins.

  Retinal hemorrhages, exudates and papilledema are characteristics of hypertensive encephalopathy and rarely found in preeclampsia.
- *Blindness* in preeclampsia– see page 89.

PREDICTION OF PREECLAMPSIA AND ECLAMPSIA

How would you predict (screening test) who is susceptible to developing preeclampsia and eclampsia (prediction of preeclampsia)?

Currently no screening test is predictably reliable. However, some combination of tests is promising.

- By identifying pregnant women who have the **risk factors**. Taking medical history as predictor test is recommended.
- By screening test like **provocative pressor tests**, e.g., roll over test, isometric exercise test and angiotension II infusion test.
- A variety of **Biomarkers** have been proposed for the development of pregnancy in later weeks (see later).
- With the help of **USG color Doppler** as written below **(Fig. 5.7A)**.

Provocative response tests

- **Roll over test:** As described by Gant et al., 1974 this is a clinical test done at 28–32 weeks of pregnancy to predict the development of preeclampsia in later weeks. Diastolic blood pressure of the woman is recorded first after 15 minutes of rest on left lateral position. Diastolic blood pressure is again recorded after turning her in supine position for 2–5 minutes. The test is said to be positive if there is rise of BP by 20 mm Hg.
- **Isometric exercise test:** Based on some principle by squeezing a handball.

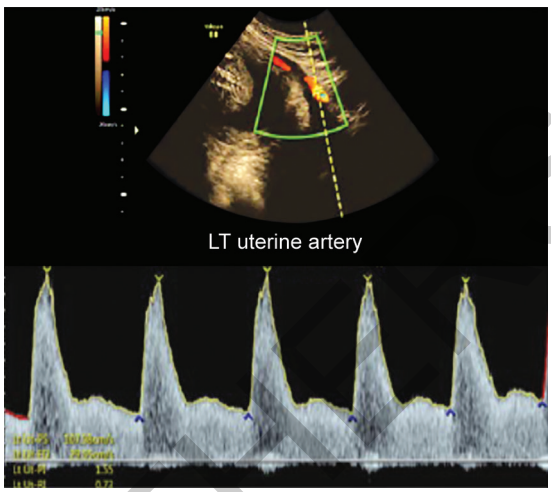


Fig. 5.7A: Uterine artery Doppler for screening for PE at 21 weeks (normal finding) (IUGR: intrauterine growth restriction; PE: preeclampsia)
Courtesy: Prof Kushagradhi Ghose, IFM and Prof Kamal Oswal, HOD, Radiodiagnosis, VIMS, Kolkata.

- **Angiotension II infusion test:** Increasing dose is infused IV and hyper tensive response is quantified.

Recent observation (2014) shows the sensitivities of these three tests is 55–70% and specificity up to 85%.

Other predictive tests (Biomarkers)

- **Fetal-placental unit endocrine dysfunction markers:** HCG, Pregnancy associated protein A (PAPPA), Inhibin A, Activin A.
- **Renal dysfunction markers:** Increases serum uric acid, microalbuminuria.
- **Endothelial dysfunction and oxidant stress markers:** Proangiogenic and antiangiogenic markers. Proangiogenic factors such as placental growth factor (PlGF) and vascular endothelial growth factor (VEGF) begin to drop before clinical development of preeclampsia. At the same time, levels of some antiangiogenic factors like sFlt-1 (FMS-like tyrosine kinase receptor-1) and soluble endoglin [sEng (endoglin)] increase and their rise becomes evident 5 weeks prior to the onset of PE.

UTERINE ARTERY DOPPLER

Prediction by USG color Doppler of uterine artery

Inadequate or incomplete trophoblast invasion of the spiral arteries is a constant feature in women with preeclampsia. It can be diagnosed by color Doppler. Uterine artery Doppler performs better in the second trimester than in

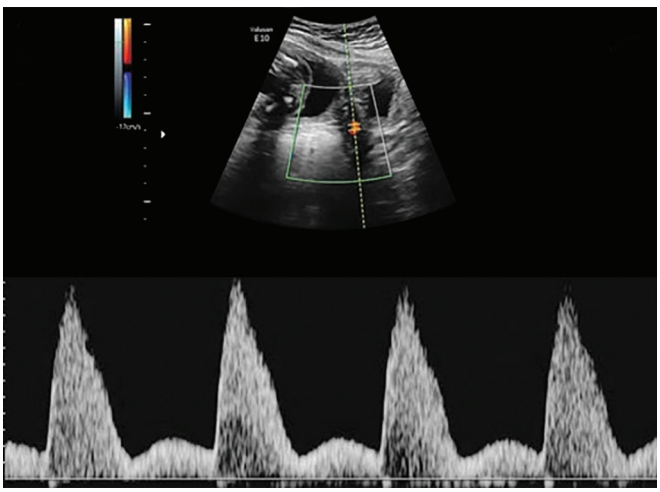


Fig. 5.7B: Uterine artery with notching (Early IUGR).
Courtesy: Prof Kushagradhi Ghose, IFM and Prof Kamal Oswal, HOD, Radiodiagnosis, VIMS, Kolkata.

the first trimester for identification of severe or early onset PE.

A high mean uterine artery Doppler PI (pulsatility index) multiple of medians (MOM) detects severe PE with a sensitivity of 78% and specificity of 95%.

Presence of notching **(Fig. 5.7B)** *on maternal uterine artery Doppler* (both right and left) at 22–24 weeks of gestation will identify more than 80% of women who will develop preeclampsia in later weeks with a false positive rate of only 5%. Notching is replaced by pulsatility index recently.

USG screening test is useful in the women who have risk factors and is less important in predicting low risk women.

First trimester low serum PIGF, high uterine artery pulsatility index, and other maternal parameters can predict development of preeclampsia in >90% cases (ACOG 2020).

DIAGNOSIS OF PREECLAMPSIA

Mention the points in favor of diagnosis of preeclampsia

- Presence of predisposing factors as mentioned previously
- Onset of the disease in the second half of pregnancy >20 weeks.
- Preeclampsia is a syndrome of more signs than symptoms. The symptoms appear in the late phase of the disease.

Symptoms may be:

- Persistent swelling of legs is not relieved by rest, gradually progressing to the face, abdominal wall, vulva and the whole body.
- Headache.
- Epigastric and right upper abdominal pain.

Signs are:

- High blood pressure. **(Table 5.2)**
- Abnormal weight gain.
- Features of fluid retention as evident from rapid abnormal rapid weight gain and non-dependent edema.
- Tenderness on right upper quadrant.
- Brisk reflexes.
- Presence of ankle clonus (more than three beats).
- Height of the fundus may be less than the period of amenorrhea due to IUGR and/or oligohydramnios (a feature of placental insufficiency).

INVESTIGATIONS

What investigations do you want to do in a case of preeclampsia?

- *Urine for protein:* By dipstick and heat acetic acid test.

If dipstick ≥1 + → 24 hours urine
→ Spot protein: Creatinine ratio

- *24-hour urine:* Total protein and protein: creatinine clearance.
- *Serum uric acid:* Level >4.5 mg% is highly suggestive of preeclampsia. Uric acid is also called *biochemical marker* of preeclampsia. Hyperuricemia precedes proteinuria. It is of value in distinguishing PIH from chronic hypertension.
- Complete blood count including platelet count, fibrinogen level (if platelets counts are low). Peripheral blood smear to diagnose hemolysis (features of hemolysis—fragmented red blood cells (RBCs), spherocytes and reticulocytes).
- Liver function test.
- Blood for urea/creatinine, electrolytes.
- **Soluble fms like tyrosine kinase (sFlt-1):** PIGF ratio (sFlt-1: PIGF ratio)

Very recently, placental growth factor (PlGF) or soluble fms like tyrosine kinase(sFlt-1): PIGF ratio testing are recommended in relation to diagnosis and timing of delivery for preeclampsia. For diagnosis and need of delivery of preeclamptic mother low PIFG value has

high sensitivity and negative predictive value within 14 days. sFlt-1: PIGF ratio of <38 excludes development of preeclampsia within 7 days. In suspected preeclampsia, estimation of PlGF or sFlt-1: PIGF ratio is useful in between 20 and 34^{+6} weeks for termination issue.

Ultrasound: Fetal size, volume of liquor amnii and fetal Doppler study.

COMPLICATIONS OF PREECLAMPSIA

What are the complications of preeclampsia?

- Maternal complications
- Fetal complications

Maternal complications:

Antenatal:

- Eclampsia
- Accidental hemorrhage (Abruptio placentae)
- Cerebral hemorrhage/PRES (see page 100)
- Cardiac failure
- Oliguria and anuria
- HELLP syndrome
- *Eye complications:* Scotoma, blurring of vision, diplopia are common. Blindness is less common.
- Coagulation failure
- Preterm labor.

Intranatal:

- Eclampsia, PPH

Postnatal:

- Eclampsia, shock

Fetal complications:

- Intrauterine growth restriction
- Intrauterine death
- Asphyxia
- Prematurity

Blindness in preeclampsia and eclampsia

- **Scotoma, blurring of vision** or **diplopia** which are common eye problems usually improve with lowering blood pressure or with $MgSO_4$ therapy.
- **Blindness** is uncommon and usually reversible. Blindness is rare with preeclampsia; the incidence may be up to 15% in eclampsia. Three areas may be involved, e.g., occipital lobe, lateral geniculate nuclei, and the retina. **Retinal blindness** is due to infarction or detachment. May be asymptomatic if there is any visual problem it usually improves. In rare cases of retinal artery occlusion there may be permanent blindness. **Occipital blindness** also known as "amaurosis" is due to either edema or infarction and resolves completely except in rare cases of extensive cerebral infarctions.
- Blindness in severe PE is due to retinal detachment and in eclampsia it is almost due to occipital lobe edema. Prognosis is good in both cases and regains vision within 1–2 weeks postpartum.

Remote complications of preeclampsia

- **Recurrent preeclampsia:** Five times more common (25%).
- **Chronic hypertension:** 20–40%. Those mothers who develop chronic hypertension have either familial diathesis or personal predisposition.

HELLP SYNDROME

What is HELLP syndrome?

- HELLP syndrome is an acronym for *hemolysis, elevated liver enzyme and low platelet* counts and a subsyndrome of severe form of preeclampsia (Weinstein 1982).
- It is rare but may occur in up to 10–15% cases of preeclampsia. HELLP syndrome mostly occurs in third trimester, but in 30% cases it is first expressed or progresses postpartum. In 15% cases of HELLP syndrome there may be insidious and atypical onset without hypertension or proteinuria (ACOG 2020).
- *Hemolysis* is due to the passage of red cells through partially obliterated vessels (microangiopathic hemolysis), as evidenced by schistocytes in blood smear, hyperbilirubinemia and absent plasma haptoglobin. *Elevated liver enzymes* are due to liver dysfunction. SGOT or AST and LDH are mostly elevated. LDH increases to 600 IU/L or more. AST and ALT are increased more than twice the upper limit of normal. *Low platelet count* (100–10^9/L) is a late feature and is probably due to platelet aggregation and platelet deposition at the sites of endothelial damage.
- In 90% cases, presenting symptoms are right upper abdominal pain and malaise and in 50% cases there is nausea and vomiting.
- **Classification:** *Partial*—one or two abnormalities, *complete*—all three abnormalities present.
- **Complications:** The chance of hepatic hematoma and rupture of liver is increased. The other complications are eclampsia, placental abruption, acute kidney injury, pulmonary edema, stroke, coagulation disorder, acute respiratory distress syndrome and sepsis. HELLP syndrome is associated with high

perinatal and maternal mortality and once diagnosed, needs aggressive treatment usually by termination of pregnancy. Stroke is commonest cause of death in HELLP syndrome (45%). The complication rates of HELLP syndrome are much higher than those of the isolated preeclampsia. For this reason, HELLP syndrome is sometimes described under "**atypical preeclampsia—eclampsia** (Sibai 2009)."

- **Management** is on the line of severe preeclampsia, i.e., antihypertensive, prophylactic anticonvulsive, close monitoring and delivery (page 91). For women with HELLP syndrome, delivery should be undertaken shortly after initial maternal stabilization. Before 34 weeks, if maternal and fetal condition remains stable a course of steroid may be completed before termination. However, the use of corticosteroids for the specific purpose of treating HELLP syndrome is not recommended.

IMPENDING ECLAMPSIA

What do you mean by "fulminant preeclampsia" or "imminent eclampsia" or "impending eclampsia?"

It is a state of preeclampsia either from the onset or appears in the case of a women already suffering from preeclampsia when there is every possibility of occurrence of eclampsia, convulsion or coma at any moment.

Features of fulminant preeclampsia/imminent (impending) eclampsia

These are **actually the features of "severe preeclampsia"**

Symptoms:

- Rapid swelling
- Headache, nausea
- Visual disturbances—blurring, dimness, diplopia, and scotoma.
- Epigastric pain
- Restlessness, trembling and twitching
- Feeling unwell
- Low urinary output.

Signs:

- *BP:* Sudden rapid rise of BP
- Facial edema
- Excitement
- Hyperreflexia
- Tenderness on upper abdomen.
- Twitching and tremor restlessness, sensorium deranged.

How will you manage a case of fulminant preeclampsia?

- Patient is brought to eclampsia room immediately. Antihypertensives are administered and prophylactic anticonvulsant therapy with $MgSO_4$ is started.
- Patient is managed on the line of eclampsia with proper monitoring.
- If the baby is mature, immediate termination is done. If the baby is premature and the patient's condition is stabilized, then the pregnancy is terminated after completing the course of corticosteroids.
- When the patient's condition has not improved, immediate termination is done irrespective of period of gestation.

PREVENTION OF PREECLAMPSIA

How will you prevent preeclampsia?

Preeclampsia is not completely a preventable disease but its severity and complications can be prevented by:

- Screening of high-risk cases
- Adequate antenatal care.
- Regular exercise during pregnancy.
  - Aspirin 75–150 mg daily from 12 weeks until delivery is given to high-risk mothers and to those with more than one moderate risk factor for the prevention of preeclampsia (in selected cases).
- **Calcium** supplementation (1.5–2.0 g elemental calcium/day) particularly in populations with dietary deficiency of calcium for the prevention of preeclampsia for all women, especially for high risk of PE.
- Antioxidants, vitamin C, vitamin E are also suggested for at risk mother, but not recommended by ACOG.
- Severity can be prevented by detecting the disease at an early stage.
- There is no evidence that bed rest or salt restriction reduces the risk of preeclampsia.

Early diagnosis of preeclampsia

During routine prenatal visit, some women without overt hypertension may show new onset increase in BP. (diastolic >80 but <90 mm Hg) or sudden weight gain (>2 pounds/week) and is advised to 7 day interval visit and is looked for development of PE.

MANAGEMENT OF PREECLAMPSIA

Principles of Management

- Early diagnosis even in symptomless condition
- To be aware of the complications
- To maintain blood pressure within the target value using antihypertensives
- To consider the use of anticonvulsant in severe variety
- To take decision whether to admit or to do home management with regular antenatal checkup and vigilance
- To terminate in optimal time to prevent maternal and fetal complications
- Postnatal follow up and advice

ADMISSION OF PATIENT SUFFERING FROM PREECLAMPSIA

When should you admit a patient suffering from preeclampsia?

Ideally, all established cases of preeclampsia should be admitted as soon as diagnosed for bedrest, for more intense monitoring and for better management. In practice, it is not always possible to accommodate large number of patients in hospital. Therefore, admission in hospital is restricted to the following group of patients.

- All cases of **severe preeclampsia** (BP 160/110 mm or more).
- In non-severe variety (BP 140/90 to 159/109 mm Hg) patient is admitted if clinical concerns for the well-being of the woman or baby or if high risk of adverse events suggested by full PIERS (at any time during pregnancy) or PREP-S (Prediction of Risk in Early-onset Pre-eclampsia-Survival) (only up to 34 weeks of pregnancy) **risk prediction models** (NICE 2019).
- In all cases of **37 completed weeks** of gestation.

ADVICES OF HOME MANAGEMENT

What advice should be given to the patient in case of home management?

- A close out patient management and a good home support is needed for pregnancy with nonsevere variety remote from term.
- Adequate bed rest is about 10 hours (8 hours night, 2 hours in the day), preferably in left lateral position.
- High protein diet with fluid and no extra salt.
- **Anti-hypertensives:** Antihypertensive is offered if BP remains above 140/90 mm Hg (NICE 2019). **Target BP** is 135/85 mm Hg or less on antihypertensive treatment. Oral labetalol is the first line of treatment, next is Nifedipine, Methyl dopa is given if labetalol or nifedipine not suitable.
- Patients should be warned regarding the **ominous features** of fulminating preeclampsia and should attend the hospital immediately if any feature develops.
- BP is measured daily or every 48 hours by the house physician.
- Dipstick proteinuria testing is done and repeated if clinically indicated, e.g., if new symptoms and signs develop or uncertainty over diagnosis.
- To attend **antenatal clinic every week or more frequently**.
- Full blood count, platelet count liver function and renal function test including uric acid twice a week.
- **Fetal assessment:** Auscultation in every visit:

Ultrasonography at diagnosis and repeat every two weeks if normal: Fetal size, amniotic fluid volume and color Doppler

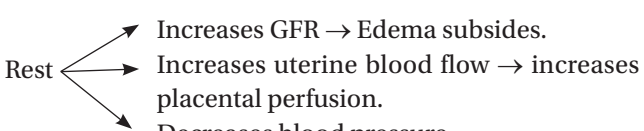
Antenatal cardiotocography (CTG) at diagnosis and then only if clinically indicated.

In majority of the cases BP is controlled and pregnancy can be continued to 37 completed weeks, when the patient is admitted.

FOLLOWING ADMISSION

Components of conservative management following admission.

- **Rest:** Reduced physical activity throughout day in any lateral position (preferably left). However, absolute bed rest is not desirable (ACOG 2013).



Reduced physical activity is beneficial but evidence is not obvious.

- **Diet:** Adequate protein and calorie diet, no extra salt and water as per requirement (no need of restriction of dietary salt intake). Vitamin D, C and E supplementation are not recommended for prevention of preeclampsia and eclampsia.

- **Medications:** Other than routine iron, folic acid and calcium, following may be needed.
  - **Antihypertensives:** Role of antihypertensive to improve maternal and fetal outcome is debatable in nonsevere variety. It is started if BP remains above 140/90 mm Hg (NICE 2019 recommendation).
    Oral labetalol is the first line of treatment, next is nifedipine, methyl dopa is given if labetalol or nifedipine are not suitable. **Target** is to keep systolic BP 135 and diastolic BP 85 mm Hg or less **(Table 5.5)**.
  - **Injection $MgSO_4$** is given in severe preeclampsia (prophylactic anti-convulsant—regime same as eclampsia, page 100). When systolic BP is <160 mm Hg and a diastolic BP is <110 mm Hg and there is no maternal symptom, $MgSO_4$ is not universally given for the prevention of eclampsia.
  - *Laxative:* Stool softener, if there is constipation.
  - Diuretics (particularly thiazide): Limited value as they impair placental insufficiency and not recommended. May be used in (1) heart failure, (2)

TABLE 5.5: Antihypertensive drugs used in pregnancy.

Drug	*Mechanism of action*	*Dose and route*	*Adverse effects*	*Comments*
Labetalol	• A combined alpha and beta blocker. By blocking α-receptors in peripheral arterioles, it reduces peripheral resistance and concurrent β-blockade protects the heart from reflex sympathetic drive normally induced by peripheral vasodilatation. Onset of action 1–2 minutes. • Maximum effect of Labetalol is reached within 5 minutes after • IV injection, peak at 30 minutes and persists for 4 hours	• Used both in oral and IV route. Oral dose—100–400 mg, twice or thrice daily orally and can be used up to 1,200 mg/day, even maximum up to 2,400 mg/day • IV route is initial choice in severe hypertension and orally in expect-ant management • Started with 20 mg IV bolus, if BP not reduced within 10 minutes, it is followed by 40 mg and then 80 mg every 10 minutes up to total dose of 220 mg. It can be given up to 300 mg per treatment cycle (ACOG 2020) • For infusion starting dose is 20 mg/h*	• May cause fetal/neonatal bradycardia • It is better avoided in asthma, heart disease and congestive heart failure • It is safe in pregnancy	• First line of treatment and most popular • Drug of choice in severe hypertension and in expect-ant management. Can cause rapid reduction of BP. Tachycardia is less common with fewer adverse effects
Methyldopa	It is a centrally acting drug with delayed onset of action (4–6 hours). Reduces sympathetic outflow	Only orally given. Dose: 250–500 mg, twice or thrice or 6 hourly daily, maximum 2 g daily	It has side effects like sedation and depression. It causes postpartum depression and should not be prescribed in postpartum period. It has no major side effect, well tested, safety proven	Commonly used drug. Useful in chronic hypertension and moderate PIH. Not suitable for severe preeclampsia
Nifedipine	• A calcium channel blocker (peripheral vasodilator) with quick onset of action (5–10 minutes) • May be used instead of labetalol and appears to be safe in late pregnancy. Onset of action is 5–10 minutes, peak at 30 minutes and action persists for 6 hours	Only oral route. 10 mg orally thrice daily. In eclampsia starting dose is 10–20 mg followed by another dose after 20 minutes if not controlled, then every 2–6 hourly with maximum daily dose 180 mg (ACOG 2020). *Sublingual route is not recommended*	• May cause nausea, tachycardia, hypotension. It may cause severe headaches that mimics worsening disease. • Nifedipine should be used cautiously as along with $MgSO_4$ it will cause maternal hypotension, profound muscle weakness and fetal distress	• Useful in severe hypertension alternate to labetalol. Nifedipine lowers • BP quickly than labetalol. • Drawback is IV preparation is not available

Contd...

Contd...

Drug	*Mechanism of action*	*Dose and route*	*Adverse effects*	*Comments*
Hydralazine (Dralazz)	Acts a s peripheral vasodilator, rapid onset of action (10–20 minutes)	• Only IV route. • Used in imminent eclampsia, eclampsia and hypertensive crisis. • Initially 5 mg. IV and can be repeated 20–40-minute intervals with 5-10 mg dose until a satisfactory result is achieved or up to total dose of 30 mg • Constant infusion 0.5–10 mg/h • Consider 500 mL crystalloids preload or with the first dose—hydralasive (NICE 2019)	Side effects are palpitation, headache, tachycardia, nausea and vomiting	• It also decreases BP quickly. Maternal and neonatal outcomes are similar with hydralazine and labetalol. • Hydralazine causes more tachycardia and palpitations. • Hypotension—labetalol results more bradycardia. • Both causes maternal hypotension

- *Others*: *Other second line antihypertensive drugs used in eclampsia*. Nitroprusside or nitroglycerine, nimodipine, verapamil and ketanserin, etc., are used in refractory cases (consultation with anesthetist or physician needed).
- **Diuretics (frusemide)** are not used in pregnancy as it compromises placental perfusion and is only used to treat pulmonary edema, heart failure.
- **ARB**s (Angiotensin receptor blockers), **ACE inhibitors** (Angiotensin-converting enzyme inhibitors) are alleged to be associated with fetal anomalies and contrandicated in pregnancy and preconception period β-blockers (atenolol and propranolol) are also not recommended in pregnancy.
- **IV infusion of labetalol:** Is started at a rate of 20 mg/h and in every 30 minutes dose is doubled till a satisfactory response is obtained or a dose 160 mg/h is reached. Commonly used IV fluids are Ringer lactate, 5% Dextrose or normal saline.

How to prepare?

- **By IV drip:** From a 100 mL bag of normal saline (0.9%) 40 mL is withdrawn. 200 mg labetalol (40 mL) (10 ampoule, each ampoule contains 4 mL, each mL contains 5 mg = total 200 mg) is added to remaining 60 mL in the bag of normal saline. Final concentration is 2 mg/mL. IV infusion is started at 10 mL/h (20 mg/h).
- **Using infusion pump:** Injection labetalol 40 mL (10 ampoule- each amp contains 4 mL, each mL contains 5 mg = total 200 mg) + 10 mL normal saline in 50 cc syringe. The syringe is fitted in infusion pump and started in 5 mL/h.
- By adjusting the rate (doubling, maintaining or halving) blood pressure is stabilized as required every 15–30 minutes to a maximum dose of 80 mL/h (160/mg h).

(BP: blood pressure)

pulmonary edema, (3) massive edema and (4) with certain antihypertensives.

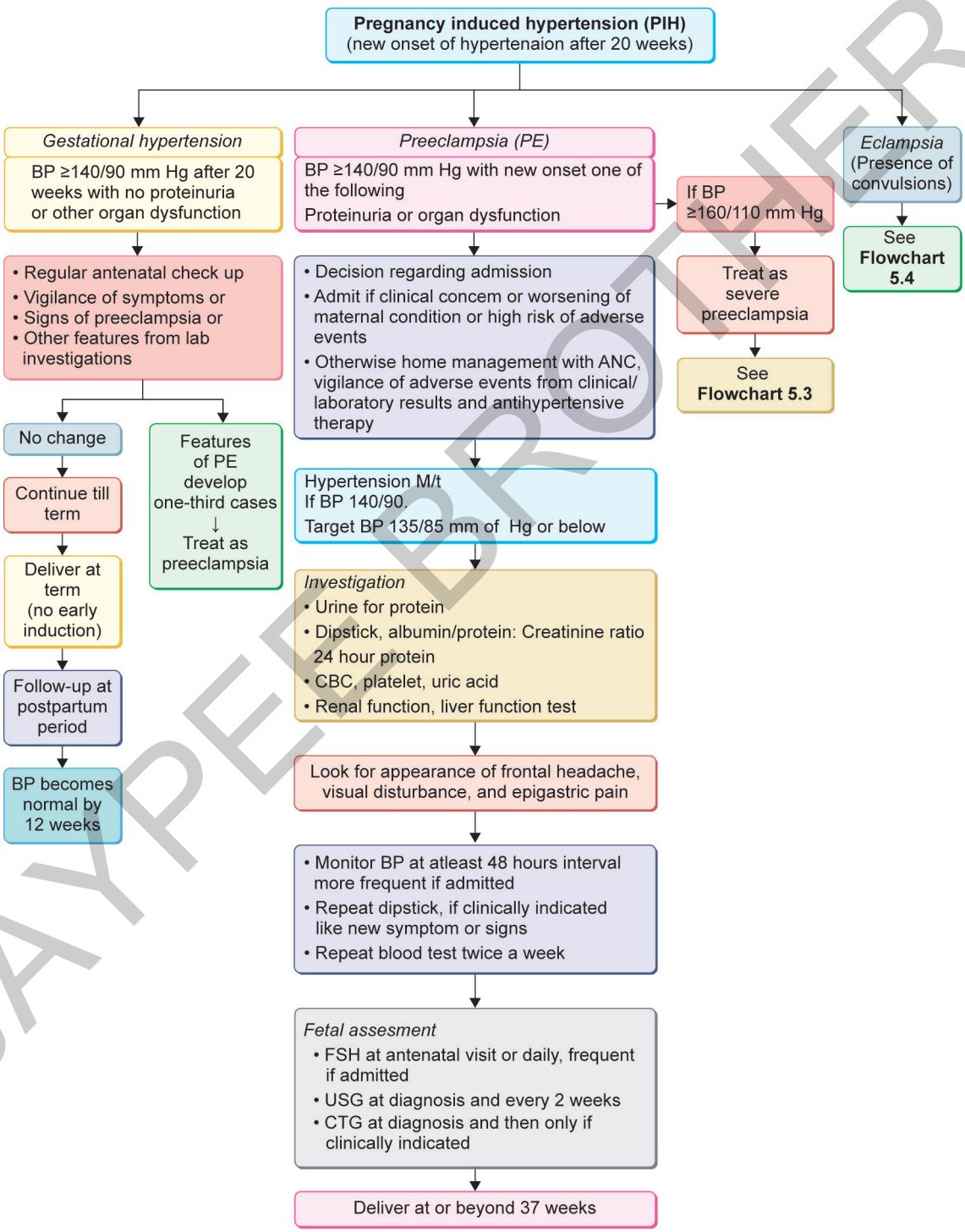
- **Maternal monitoring:**
  - Maternal wellbeing, appearance of new symptoms and signs-severe headache, visual changes, epigastric pain and shortness of breath.
  - Amount of urine every day (oliguria means <400 mL of urine in 24 hours).
  - BP measurement twice daily, at least four times a day in severe hypertension and every 15–30 minutes until BP is <160/110 mm Hg.
  - Weight measurement alternate day.
  - Dipstick proteinuria is tested. Repeated if clinically indicated, e.g., new symptoms/signs or uncertainly over diagnosis.
  - Urine for 24-hour protein or protein creatinine ratio.
  - Complete blood count, platelet count, peripheral blood smear, renal function test (urea, creatinine, and uric acid), electrolytes, liver function test–twice or thrice weekly. Serum creatinine is done daily, where creatinine is elevated beyond twice its normal value.
  - Ophthalmoscopic examination.
- **Fetal assessment:**
  - **Fetal wellbeing** is assessed by clinical examination including FHS auscultation (done twice daily) and daily fetal movement count. Other parameters such as nonstress test CTG, biophysical score

    Doppler velocimetry [in suspected fetal growth rate (FGR)] is done once weekly depending upon clinical findings (page 38).

- **Fetal growth** is assessed clinically and with the help of USG every week. Frequency is altered depending on clinical condition.

Flowchart 5.2 provides an algorithm for approaching the patient with pregnancy induced hypertension.

Flowchart 5.2: Algorithm showing approach to management of pregnancy induced hypertension.



Bedside Clinics in Obstetrics

Clinics to Care with Illustrations

Salient Features

- This book has been written keeping in mind the requirements of students preparing for theoretical, oral, and practical examinations. Although primarily intended for MBBS students, it is equally valuable for postgraduate students, with content aligned to the latest National Medical Commission (NMC) competency-based curriculum
- The book presents individual clinical cases in a systematic sequence, covering case taking, history, clinical examination, diagnosis, and management. The subject matter is presented in a reader-friendly question-and-answer format and is supported by a large number of colored illustrations, original clinical photographs, and tables. These features facilitate better understanding of the subject and help students perform confidently in examinations
- Clinical methods, examination maneuvers, modern investigations, partography, ultrasonography, cardiotocography, surgical procedures, and obstetric and gynecological instruments are comprehensively illustrated with more than 800 original photographs and 622 colored hand-drawn illustrations
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