Hypertensive Disorders of Pregnancy (ch 14)

- **Introduction**:

  - HTN during pregnancy can present in the following types:

1. Chronic hypertension,
2. Preeclampsia/eclampsia
3. Chronic hypertension Superimposed preeclampsia,
4. Gestational hypertension

  - The worldwide incidence of these disorders is reported to be about 10%,
  - Blood pressure readings vary, depending on maternal position and the gestational age of the pregnancy:

    - Arterial blood pressure **normally declines** during the first and second trimesters of pregnancy and **rises** to prepregnancy levels in the third trimester.
    - **The diagnosis of hypertension should be reserved for pregnant women with a systolic blood pressure ≥140 mm Hg and/or a diastolic blood pressure ≥90 mm Hg,**
    - Blood pressure measurements should be taken with the woman in the sitting position after she has rested ≥10 minutes if she is ambulatory

1 CHRONIC HYPERTENSION:

The diagnosis of chronic hypertension requires at least one of the following: (book definition)

- **known** hypertension **before** pregnancy or - the **development** of hypertension **before 20 weeks' gestation.**
- Or where hypertension is first noted during pregnancy and persists for **longer than 12 weeks postpartum** (video case answer)
  - Most pregnant women with chronic hypertension will have **essential hypertension**
  - Small percentage will have **secondary hypertension** that has renal, vascular, endocrinologic, or behavioral causes.
    - Depending on the associated symptoms, signs, and response to medication, a workup to determine the etiology of the hypertension may be indicated.

➢ MANAGEMENT:

The goal is control hypertension and detect the development of superimposed preeclampsia in the mother and IUGR in the fetus.

- It is important determine the type of the HTN whether it is essential hypertension or a secondary cause of high blood pressure
- Early laboratory test is **to establish a baseline** for the patient, **review the antihypertensive medications** being taken and to discontinue any that are potentially teratogenic.
- Start Rx: **if the systolic blood pressure is ≥160 mm Hg or the diastolic blood pressure is ≥105 mm Hg.**[1]
  - The **Safest** antihypertensive medication in pregnancy is **Methyldopa**, other safe medication includes: **calcium channel blockers** and **labetalol**,
  - **Angiotensin-converting enzyme inhibitors**, **angiotensin II receptor blockers**, **renin inhibitors**, and **mineralocorticoid blockers** are contraindicated at all stages of pregnancy …?
    Bc they have **potential fetal toxicity**.
  - **Beta blockers** should be used with caution because they may cause fetal growth restriction and may affect the interpretation of the NST.
- **Follow up:**

- **both early and serial ultrasonic examinations are indicated:**
  1. The early ultrasound (before 12 weeks) is primarily for dating,
  2. 18- to 22-week ultrasound is for the assessment of fetal anomalies.
  3. Serial ultrasonic examinations (every 3 to 4 weeks after 26 to 28 weeks) are of great assistance in detecting IUGR[2]
- Remember the incidence of superimposed preeclampsia varies from 15-25% (you have to monitor for new-onset proteinuria or significant increase in the BP).

BOX 14-3

INITIAL LABORATORY EVALUATION FOR A PATIENT WITH PREECLAMPSIA

- CBC, platelet count, LDH: if abnormal, order D-dimers, coagulation panel, and smear
- Renal studies: serum BUN creatinine and uric acid, urinalysis, 24-hr urine for protein and creatinine, or protein/creatinine ratio
- Liver function tests: AST, ALT, and bilirubin

[1] If less than it, consider that lowering the blood pressure too much may result in **decreased uterine perfusion pressure and** iatrogenic fetal growth restriction + in many women, blood pressures will decrease to normal in the second trimester, and no antihypertensive medication will be needed

[2] NSTs, amniotic fluid assessment, and umbilical artery Doppler studies should be commenced by 32 to 34 weeks in all patients with hypertension.

➢ Delivery:

Depends on the clinical circumstances.

- **For patients without evidence of fetal growth restriction or superimposed preeclampsia, in whom blood pressure is well controlled and who have no other indications for delivery, pregnancy may be allowed to progress until at least 38 weeks' gestation, provided that fetal well-being is normal.**
- The presence of IUGR, blood pressure deterioration, or the advent of proteinuria may dictate earlier delivery.
  - **The route of delivery should be vaginal in the absence of other obstetric reasons for cesarean delivery. (preeclampsia is NOT reason for C.S).**

2 GESTATIONAL HYPERTENSION: (MIX BAG)

A hypertension that is:

- **without proteinuria** or other **signs of organ dysfunction** **first** appears **after 20 weeks' gestation**

OR

- **within 48 to 72 hours of delivery and resolves by 12 weeks postpartum**

➢ SYMPTOMS and PHYSICAL EX: nothing related to preeclampsia

➢ LABORATORY FINDINGS: no proteinuria.

How to diagnose it?

- **Can only be made in RETROSPECT:**

Completion of pregnancy without the development of proteinuria or other evidence of pre-eclampsia

+

blood pressure returns to normal before the 12th week postpartum

- **A significant percentage go on to develop proteinuria and the full preeclamptic syndrome at a later stage in pregnancy, while others will have previously unrecognized chronic hypertension.**

3 PREECLAMPSIA/ECLAMPSIA:

A- PREECLAMPSIA is a multisystem disorder unique to pregnancy and has varying clinical presentations.

How to diagnose it?

new-onset hypertension[3] after 20 weeks of pregnancy

+

new-onset proteinuria[4] (and/or) other evidence **(one or more)** of organ dysfunction*.

* Evidence of Organ Dysfunction:
- Thrombocytopenia,
- (DIC),
- ↑ Transaminases or other signs of Hepatic Injury,
- CNS Symptoms
- ↑ Serum Creatinine Level,
- Pulmonary Edema

- **Sever preeclampsia:**

- Since it's a **progressive** disease, it may have severe features,
- **If any feature of the listed criteria is present** it is very likely that the pt. has **severe** disease.
  - **HELLP syndrome:**
- A variant of severe preeclampsia with particularly high morbidity,
- **Preeclampsia + evidence of *h*emolysis, *e*levated /iver enzymes, and /ow *p*late- lets (thrombocytopenia).**
- More in: - **multiparous**, - **>25 age**, - **<36 w gestation**.
- Can progress into DIC.

BOX 14-1

CRITERIA FOR SEVERE PREECLAMPSIA

- Severe hypertension (systolic BP ≥160 mm Hg or diastolic BP ≥110 mm Hg) at rest on two occasions at least 4 hr apart*
- Renal insufficiency (serum Cr >1.1 mg/dL or doubling of baseline values)
- Cerebral or visual disturbances
- Pulmonary edema
- Epigastric or right upper quadrant pain
- Elevated liver enzymes (AST or ALT at least two times normal level)
- Thrombocytopenia (platelet count <100,000/μL)

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[3] **defined as the development of hypertension** (systolic blood pressure ≥140 mm Hg or diastolic blood pressure ≥90 mm Hg on two occasions 4 hours apart) **in a woman whose blood pressure readings were previously normal after the 20th week of pregnancy.**

[4] **defined as ≥0.3 g of protein in a timed 24-hour urine collection or a protein/creatinine ratio ≥0.3 after the 20th week of gestation.**

B- ECLAMPSIA is the presence of new-onset grand mal seizures (before, during or after delivery) **in a woman with preeclampsia that cannot be attributed to other cause.**

- **Pt. with sever Preeclamsia + they have CNS symptoms are at greater risk** (this doesn't role out it incidence in less sever form).
- **38-53% of eclamptic seizures occurred before labor, 18-36% occurred during labor, and 11-44% occurred after delivery** (usually within the first 24 to 48 hours).
- it is important to consider other causes of seizures, such as underlying seizure disorder; hypertensive encephalopathy; metabolic abnormalities…etc.

C- CHRONIC HYPERTENSION WITH SUPERIMPOSED PREECLAMPSIA

- Superimposed preeclampsia can be very difficult to distinguish from poorly controlled chronic hypertension, especially if the woman is not seen until after the 20th week of gestation,
- In general, superimposed preeclampsia carries a worse prognosis than either condition alone.
  - **How to diagnose it?**

- **women with chronic hypertension ® develop new-onset proteinuria (≥0.3 g in a 24-hour collection) after the 20th week of gestation.**

OR

- **women with preexisting hypertension and proteinuria and she experiences sudden significant increases in blood pressure "OR" proteinuria "OR" the new onset of any of the other signs and symptoms of severe preeclampsia** listed in Box 14-1

➢ PREECLAMPSIA/ECLAMPSIA:

Pathogenesis: "disease of theories"	Risk factors:	Pathology:
There is **no single, definitive "cause"** has been identified and the origins of the disease are considered to be multifactorial. **Inadequate uteroplacental perfusion leading to placental ischemia, or hypoxia, appears to be central to the development of the disease**, this ischemia will result in endothelial dysfunction (↑vasoconstrictors ↓vasodilator), vasospasm, and activation of the coagulation system and eventually vascular damage. Placental hypoxia could be: ○ due to failure of the cytotrophoblasts to adequately invade the uterine spiral arteries and establish the low-resistance uteroplacental circulation characteristic of normal pregnancy. Or ○ due to **underlying maternal vascular disease.**	➢ Maternal Risk Factors: - Advanced Maternal Age, - Methamphetamine Use, - Family Hx of preeclampsia in 1st degree relative, - Hx of: • preeclampsia in previous pregnancies, • Chronic HTN, • Renal disease •Hypercoagulability • DM • Obesity • SLE ➢ Immunologically mediated placental vascular damage in cases like: - Primigravidas and - Autoimmune Disorders such (Antiphospholipid Syndrome) ➢ Ischemia induced by increased metabolic demands: - Multiple gestation, - A large singleton fetus, - Gestational trophoblastic disease (GTD).	- **lack of decidualization of the myometrial segments of the spiral arteries,** - **ischemia, hemorrhage, and necrosis in many organs, presumably secondary to arteriolar constriction.** - **Glomerular Capillary Endotheliosis:** Seen by electron microscopy **CLINICAL AND LABORATORY MANIFESTATIONS (MATERNAL)** ➢ **Weight Gain and Edema:** - **Early with preeclampsia and reflect an expansion of the extravascular fluid compartment** (endothelial injury>↑ permeability). - ↑ hematocrit (reflect **hypovolemia**), - ↑ the risk of **pulmonary edema** (if u treat aggressively with fluids), - **don't use diuretic** (unless ur dealing with pulmonary edema). ➢ **Hypertension:** Cardiac output in untreated women is not different from normal women in her 3rd tri ➢ **Renal function:** - ↓renal flow and GFR, - afferent vasoconstriction > damage to the glomerular membranes > increasing the permeability of these membranes to proteins and > PROTEINURIA - Renal involvement may progress to significant oliguria and frank renal failure. ➢ **Coagulation system:** - Thrombocytopenia is the most common abnormalitym, - DIC if there was placental abruption (severe preeclampsia) ➢ **Liver function:** - RUQ or epigastric pain and **elevated serum enzyme levels.** ➢ **Placental function:** **- IUGR, -** oligohydramnios, or - fetal heart rate abnormalities (assessing placental perfusion by umbilical artery Doppler study), - retroplacental hemorrhage, or abruption in severe cases. ➢ **CNS:** Visual disturbances, new-onset headache, hyperreflexia.

Fetal complication: • **Growth restriction** • **Prematurity** • **Perinatal death**

➢ Evaluation and Management of Preeclampsia:

1- Does the disease process have severe features?
2- Is there evidence of fetal compromise?
3- Is the fetus mature enough for a reasonably uncomplicated course after delivery?

Delivery is the only definitive cure for preeclampsia.

1. In those with **no evidence of fetal compromise**, and whose **disease does not appear to be severe or progressing** will generally **not be delivered unless** the **gestational age is 37 weeks or older**.

2. woman with **severe preeclampsia or eclampsia** whose disease presents **at or beyond 34 weeks' gestation** should usually be **delivered after a brief period of stabilization**.

3. **Severe preeclampsia** presenting **at less than 34 weeks' gestation** may in certain situations be **stabilized**, and with **careful monitoring** of the mother and fetus, and **delivery may be delayed until the pregnancy reaches 34 weeks.**

- **initial maternal assessment:**

- Hx (past, presenting symptoms)
- PE (vital sign, weight gain, edema, fundal height, reflexes, qualitative assessment of urinary protein excretion with a dipstick)
- role out sever preeclampsia,
- laboratory studies (see up Box 14-3)

- **fetal evaluation:**

- evaluating the GA, fetal growth, amniotic fluid index by US,
- nonstress test,

- **Initial stabilization: for patient with severe preterm preeclampsia with magnesium sulfate for seizure prophylaxis, along with medical control of severe hypertension and corticosteroids for fetal lung maturity.**
- **Labor should be induced and vaginal delivery** anticipated in the absence of any obstetric indications for cesarean delivery,
- Chronic antihypertensive therapy or diuretic therapy does not prevent the progression to severe disease and is not recommended.
- **The most common errors that occur in the management of these patients are fluid volume overload, resulting in pulmonary edema, and excessive volume restriction.**

SEIZURE PROPHYLAXIS

In patients with preeclampsia, **severe headaches, visual changes, sustained clonus, or a positive Chvostek sign** can be prodromal symptoms or signs of **eclampsia.**

- **Magnesium sulfate**[5] (IV, IM) given by controlled infusion pump should be instituted in patients with severe preeclampsia during 1- **the initial period of stabilization** and again during the 2- **intrapartum** period and 3- continued for **24 hours postpartum** or until there is evidence of **resolution of the disease**.
  - **Signs of toxicity**: loss of patellar reflex, warmth and flushing, somnolence and slurred speech, and, most significantly, paralysis and cardiac arrest.
  - Therapeutic **levels to avoid toxicity** should not be allowed to rise above 7 to 8 mg/dL
  - Monitor the urine output[6], deep tendon reflexes, and respirations + with serial measurements of serum magnesium levels every 6 hours > to catch toxicity
  - toxicity is treated by stopping the infusion and, **when severe**, administering IV calcium gluconate, 10 mL of a 10% solution, along with resuscitative measures if necessary.

ANTIHYPERTENSIVE THERAPY

Arterial blood pressure ≥160 mm Hg systolic or ≥110 mm Hg diastolic is a hypertensive emergency must be treated promptly.

- The goal is to **stabilize** the mother by lowering blood pressure carefully to **prevent CNS hemorrhage**.
- Blood pressure **should not be** lowered to **normal levels or to <130/80 mm Hg** …? **decreases uteroplacental blood flow and result in fetal distress and C.S might be necessary.**
- **The safest, most efficacious drugs for the acute control of severe hypertension complicating preeclampsia are labetalol and hydralazine.**

Drug	Action	Side Effects	Comments
Hydralazine	**Direct vasodilator**	**Headache, tachycardia, flushing, vomiting**	↑ CO and uterine renal blood flow; has historically been drug of choice for short-term control.
Labetalol	**Nonselective α1-blocker β1-blocker**	**Nausea, vomiting, heart block, bronchoconstriction, dizziness**	Current drug of choice in many centers. **Avoid if evidence of asthma or acute heart failure.**

➢ Management of Eclampsia:

- This emergency should be treated in an isolated labor room, with minimal noise and not too much light.
- Prevent injury, clear the airway, give oxygen by face mask to relieve hypoxia, administer 2 IV lines and urine Foley catheter. (remember Box 14-3)
- **Magnesium sulfate is the most efficacious drug for preventing recurrent eclamptic seizures and has the best safety profile for the mother and fetus.**
- Stabilize before induction of labor of C.S (correct hypoxia, control the convulsion, reduce the BP "diastolic 90-100")

[5] **for seizure control and associated with low neonatal morbidity.**
[6] **Magnesium toxicity can occur even in a patient with apparently normal renal function**