Obstetrics & Gynecology TEAM



Hypertensive disorders in Pregnancy



very important mentioned by doctor team notes mentioned important



If uncontrolled the patient may undergo:

HELLP Syndrome: Severe form of HTN in pregnancy.

- Hemolysis
- Low platelets
- Elevated liver enzyme

1. <u>Pregnancy induced Hypertension :</u>

HTN that develops as a consequence of pregnancy. Include 3 types:

a. Gestational hypertension:

Elevation of B.P

without Pathological edema, proteinuria, hematological or biochemical changes. with or without fetal implication of HTN

b. Pre-eclampsia:

Elevation of BP <u>with</u> proteinuria and or pathological edema, biochemical and or hematogical changes.

- edema may not be present
- Edema here \rightarrow swelling of the <u>face & upper extremities</u> **not** the lower extremities!
- Pre eclampsia is further subdivided into :
 - 1. Mild { further explanation is coming!
 - 2. Severe J

→ No moderate category in pre-eclampsia.

c. Eclampsia: Convulsions

A complication of Gestational hypertension or Pre-eclampsia with clinical seizure!

2. <u>Pregnancy aggravated HTN.</u>

Pre existing hypertension that got worse during pregnancy. (already on hypertensive medication)

a. Superimposed pre-eclampsia

Patient with <u>HTN getting worse during pregnancy</u> + develop <u>Pre eclampsia symptoms</u>: (elevation of BP + proteinuria + pathological edema + biochemical changes and or hematogical changes)

Ex: Patient who has pre existing HTN getting worse by pregnancy & complicated by hematogical + biochemical changes \rightarrow super imposed pre eclampsia.

b. Superimposed eclampsia

Ex: Patient with pre existing HTN getting worse by pregnancy & complicated by hematogical + biochemical changes with seizure \rightarrow super imposed eclampsia.

3. HELLP Syndrome:

Sever form of HTN in pregnancy.

- Hemolysis
- Low platelets
- Elevated liver enzyme

Common MCQ's: components of HELLP syndrome

HELLP syndrome	
H	Hemolysis
EL	Elevated Liver EZ
LP	Low Platelets

<u>Physiological Changes in Blood pressure during pregnancy:</u>



- Slight drop in 1st trimester
- continuous through the 2nd trimester
- gradual rise to pre-pregnancy level in the 3rd trimester.
- HTN is never normal in pregnancy.
- Elevation of BP above pre-pregnancy level is always abnormal.
- The curve shows the systolic & diastolic BP changes, between 20 & 30 weeks there is a drop & continuous in the 2nd trimester & then rise again in the 3r trimester but to the pre pregnancy reading not more than that never.
- Patient with BP 120/70 in the booking appointment then she comes again in the second trimester with BP of $140/90 \rightarrow$ this is abnormal, she has HTN.

• <u>Definition of Hypertension in Pregnancy:</u>

BP > 140/90 after 20 weeks gestation in the sitting position.

Why after 20 weeks? HTN <u>before</u> $20w \rightarrow$ this is a pre-pregnancy HTN not a pregnancy induce HTN. Why sitting? BP drop in lying down → false reading.



- Old definition : if there is an increase of 30 mmHg systolic, 15 mmHg diastolic → diagnostic for pregnancy induce HTN→ this is no longer used.
- Read BP twice, readings 6 hours apart, patient is well rested.

• <u>Risk factors for HTN in pregnancy :</u>

- Nulliparity (HTN in pregnancy is called the disease of Primigravida)
- Extremes of age (teenager or old age)
- Multifetal pregnancy (twins, triplet)
- Hydrops fetalis (Rh Disease)
- Diabetes
- Renal disease
- Auto immune disorders (SLE, Thrombophilia, Antiphospholipid syndrome, Rheumatoid)

* Molar pregnancy also a risk factor for the development of fulminating pre eclampsia but the doctor said I didn't include it here cause it usually diagnosed before 20 weeks of gestation .

→ first pregnancy, or if she has a 2nd or 3rd pregnancy but from a new husband → first exposure to sperms)

• <u>Pathophysiology</u>:

Vasospasms: Vascular constriction causes resistance to blood flow and accounts for the development of arterial HTN.

- Start at level of <u>spiral vessel</u> where the invasion of the trophoblasts to the endometrium take place at the time of implantation, this process <u>start very early in pregnancy</u>.
- Vasospasm is due to ↑production of Thromboxane → (vasoconstriction)
 ↓ production of Prostocycline → (vasodilatation)

→ Management: <u>Aspirin prophylaxis</u> (thromboxane inhibitor) given early in pregnancy to protect from pre eclampsia & HTN.

• Dietary deficiency of Ca. \rightarrow not proven to be the cause but give Ca supplement.



• <u>Different scopes of the disease have maternal and fetal manifestations to different extents.</u> Depending on the <u>severity</u> of the disease & <u>duration</u> of the disease

A. Gestational HTN:

- May be asymptomatic \rightarrow (detected only by measuring BP)
- No edema
- No protein urea
- No hematological abnormalities
- No biochemical abnormalities
- -/+ Fetal involvement

e.g. IUGR, oligohydramios Δ changes in fetal heart tracing and BPP depending on the duration of the diseases. placental abruption, fetal death

- mechanism of fetal involvement :
- 1. Intra uterine growth restriction: (vasospasm & reduction in placental blood flow → reduction in O2 & nutrition → IUGR)
- 2. Oligohydramios → reduction in placental blood flow → the first organ affected is the fetal Kidney (no adequate volume from the circulation → making less urine → less amniotic fluid production (the amniotic fluid is mainly the fetal urine) → oligohydramios
- 3. placental abruption \rightarrow because of severe forms of pre-eclampsia \rightarrow fetal death
- 4. Changes in fetal heart tracing → due to reduce circulation & O2 → asphyxia = hypoxia → Changes in fetal heart rate : deceleration or reduce variability or others.



5. Changes in Biophysical profile:

- BPP = Biophysical profile → test to assess fetal well being, composed of 4 component :
 - 1- Fetal breathing
 - 2- Fetal movement
 - 3- Fetal tone
 - 4- Amniotic fluid volume

Score of 2 for each of these component, Total score out of 8. The baby get 2 or 0 for each component \rightarrow never get 1 \rightarrow this is a common mistake in exam

In exam : they will gave us finding & ask to calculate the total score of BPP \rightarrow if they mention the tone is weak \rightarrow the baby get 0 not 1 !!

Modified BPP :

Same score together with **→** Non Stress Test

we do it to improve the score if baby is missing any of the 4 items above. score out of 2 also (Reactive=2, non reactive=0)

Ex: if the baby is missing the breathing \rightarrow we do NST & the score will be out of 10

BPP used a lots in hypertensive pregnant mothers to assess the fetal wellbeing

pay attention : Fetal Heart Rate is **not** a component of BPP → common mistake in exam

B. Pre-eclampsia:

1. Mild :

- BP < 160/110.
- Edema of the face and upper extremities.
- Proteinuria > 300 mg /24 hours urine collection \rightarrow ("trace" up to "+1" in urine dipstick)

2. Severe :

- BP > 160/110
- Proteinuria 4-5 gm /24 hours (+ 2 or more urine dipstick)
- Headache
- Visual disturbance **>** spots, lightning
- Epigastric pain → (usually pregnant have heart burn but if sudden severe this is an alarm)
 → Epigastric pain is due to tension & edema in the liver capsule.
- Oliguria → because of intra vascular volume depletion → important in administration of drugs → if the drug is not excreted through urine adequately she may become toxic.
- Pulmonary edema
- ↓ Platelets count → coagulopathy + consumption of factors → patient present with DIC specially if she has abruption of placenta→ platelet is first indicator of ongoing process.
- ↑ KFTS kidney function test
- Fetal Involvement as previously mentioned. Depend on duration & severity of the illness

C. Eclampsia:

- Generalized tonic, clonic seizures
- You must Roll out epilepsy
- → The patient may have epilepsy before & the first presentation is now during pregnancy!
- \rightarrow Hx, gestational age, lab test, no proteinuria, no edema \rightarrow this may not eclampsia but epilepsy!
- Eclampsia is considered as a severe form \rightarrow indication of terminating the pregnancy!

D. Pregnancy aggravated HTN with:

 \rightarrow you have to worn the pt. about the symptoms of pre eclampsia & eclampsia

- * Super-imposed pre-eclampsia
- * Super-imposed eclampsia with fetal involvement

E. HELLP syndrome:

• Severe form with rapid deterioration.

• Active aggressive management regardless of gestation age. forget about the fetal age you have no time you must save the mother life!

Management:

- Prevention : ANC antenatal care
- Aspirin (reverse the process of thromboxane)
- Diet (Ca supplement)
- BP control in HTN patients
- Eclampsia & HELLP → need admission

Investigations:

• CBC :

- \rightarrow platelet count \rightarrow indicate severity of the disease
- \rightarrow Hg \rightarrow if drop from 12 to 10 after 2 hour \rightarrow Hemolysis
- → Hematocrit → hemoconcentrated & intravascular volume depleted
- U/A urine analysis, 24 hour urine collection -> proteinuria
- LFTS \rightarrow elevated in severe disease (alkaline phosphatase normally rise in pregnancy)
- **KFTS** \rightarrow Na, K, creatinine, uric acid (uric acid normally elevated but to certain limit)
- Coagulation profile
 - $PTT + PT \rightarrow Prolonged$
 - D dimmer (fibrinogen + fibrinogen degradation products) →
 - fibrinogen is low
 - fibrinogen degradation products is high
 - Coagulation factors → being consumed
 - micro-emboli & micro-thrombi.

Treatment depends on:

- Severity of the condition
- Fetal maturity

\rightarrow Use them in combination if :

- Disease is mild & fetal is immature \rightarrow you have time to wait
- Disease is mild & fetal mature \rightarrow deliver
- Disease is severe & fetal is immature \rightarrow terminate the pregnancy (get the baby out not abortion) regardless of the fetal age because you are exposing the mother to dangers.

- Treatment in mild cases with prematurity :
- conservative management is recommended
- close fetal and maternal monitoring
- administration of steroids.

• In severe cases:

Stabilization and delivery regardless of the fetal age is indicated

→ Anti hypertensive + fluid + Magnesium sulfate + deliver

• Mode of delivery depends on how much time you have:

 \rightarrow cervix, parity, head fetus, rapture membrane, gestational age, deceleration

 \rightarrow Induction of labour or C.S.

* depending on factors above our decisions in clinical exam scenarios *

• BASIC RULES in management:

- A. Use <u>antihypertensive drugs</u> to prevent maternal CVA cerebrovascular accident
- **B.** Use <u>Mg SO4</u> for seizure prevention
- C. Continuous fetal monitoring
- **D.** Measure Input & output

→ Use <u>Antihypertensive</u> to prevent maternal CVA cerebrovascular accident

- If Diastolic BP of $105 \rightarrow$ start antihypertensive to save the mother from CVA
- 1. commonly used is I.V. hydralazine → drug of choice
- 2. <u>Aldomet</u> is used in cases of <u>chronic HTN</u>
- 3. <u>Lasix</u> should **not** be used \rightarrow they cause intravascular depletion \rightarrow renal failure
- 4. <u>ACE inhibitors are contraindicated</u> \rightarrow fetal problem

→ Use Mg SO4 for seizure prevention → Intrapartum + 24 hours postpartum

\diamond Dosage : \rightarrow (this is the only dose you need to memorize it)

- <u>4 gm IV load over 30 minutes</u> (prophylactic)
- followed by <u>2 gm/ hour</u> Intrapartum infusion (maintenance) until the patient delivers.
- once she deliver reduce the dose to <u>1 g/hour for a period of 24 hours</u>
- intramuscular injection is painful + with different doses.

***** Monitoring of patients on Mg SO4 :

- Mg levels \rightarrow draw blood level every 4-6 hours to prevent toxicity
- U/O volume → Urine output Less than 30-40 cc/ hour → Mg level are high→ check your infusion rate
- Reflexes → CNS Depression → over-dose
- Respiratory rate → respiratory arrest
- ♦ Anti dote of Mg SO4 → <u>Calcium gluconate</u> injection.

• Summery :



Physiological changes in BP in pregnancy:

- Slight drop in 1st trimester \rightarrow continuous through the 2nd trimester
- Gradual rise to pre-pregnancy level in the 3rd trimester.

Risk factors for HTN in pregnancy

Nulliparity, Extremes of age, Multifetal pregnancy, Hydrops fetalis, DM, Renal disease

Pathophysiology :

Increase production of Thromboxane + Dietary deficiency of Ca