Learning objectives

- To understand the classification of hypertension in pregnancy
- To be able to differentiate the different risks associated with various types of hypertensive disorders in pregnancy
- To understand the pathophysiology of pre-eclampsia
- To be aware of the clinical presentation of PE & understand the principles of management
- To understand the long term risks to both mother & baby from PE

Introduction

- HTN is common in pregnancy.
- Approx. 1/10 women will have raised BP prior to delivery
- Gestational HTN In majority & is benign
- = 1/3 women will develop pre-eclampsia- leading cause of maternal death

WHO estimates: 50,000 -75,000 women globally die of this condition

Women with chronic HTN assoc. with increased risks to mother & baby

Classification

- Non-proteinuric pregnancy induced HTN-
 - Elevated BP during pregnancy
- · Pre-eclampsia-
 - Hypertension in pregnancy
 - Proteinuria
 - End organ damage
- · Chronic hypertension-
 - Elevated BP prior to pregnancy

Gestational hypertension

- Elevated BP after 20 weeks
- No proteinuria or evidence of preeclampsia
- Not associated with adverse pregnancy outcome
- Mild to moderate increase in BP do not require T/M
- 1/3 will progress to pre-eclampsia



Chronic hypertension

- Women who have confirmed hypertension in the first half of pregnancy.
- · HTN before 20 weeks
- Majority have Essential HTN
- Predisposes to later development of preeclampsia



Preeclampsia

- Multi-system disorder of pregnancy
- Complicates 2-3% of pregnancies
- Leading cause of death in low resource settings
- Hypertension
- · Proteinuria
- End-organ dysfunction



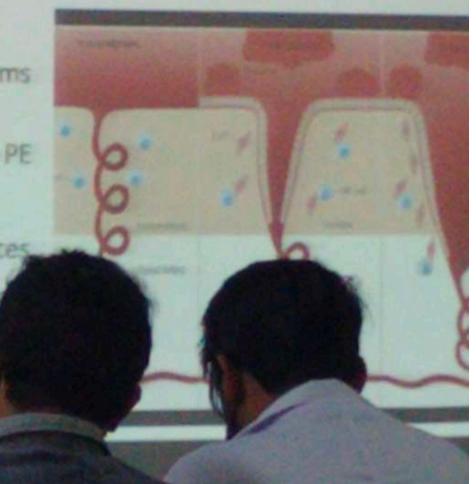
Definition

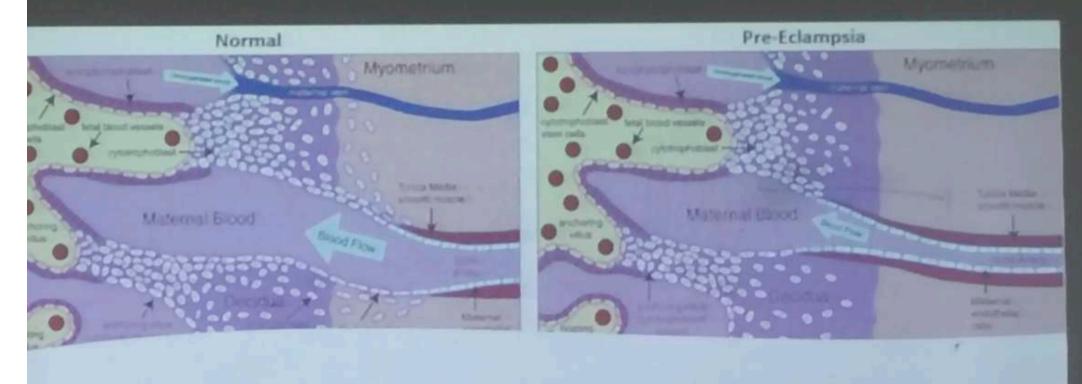
- PE is defined as hypertension of atleast 140/90 mmHg recorded on atleast two separate occasions, at least 4 hours apart
- The presence of 300 mg protein in a 24 hour collection of urine
- Arising after 20th week of pregnancy in a previously normotensive woman
- Resolving completely by the sixth postpartum week



Pathogenesis

- Disorder of the placenta
- Normally trophoblast invades/transforms spiral arteries
- Abnormal invasion/transformation --> PE
- * Placental under-perfusion
- Leads to release of circulating substances
- Diffuse maternal endothelial dysfunction
- * Vasospasm & coagulation
- * Resolves with delivery (placental rem





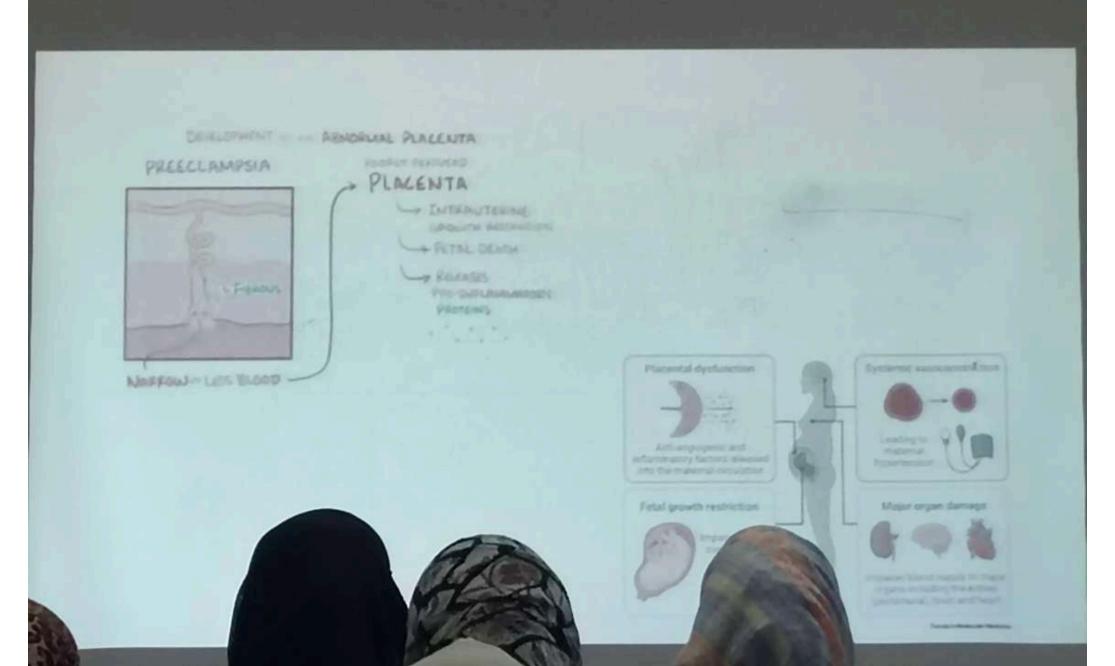
Pathogenesiscontd.

- Extravillous trophoblast fails to penetrate myometrium
- Spiral arteries do not develop normally
- Remain narrow---> placental hypoperfusion
- Placental biopsy: Fibrinoid necrosis of vessels

Pathophysiology of PE

- CVS: Marked peripheral vasoconstriction

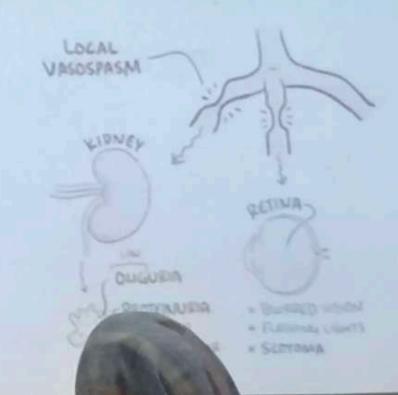
 HTN
- Renal system: Glomerular endotheliosis loss of albumin, edema
- · Haematological system: Increase fibrin & reduction in platelet count
- Liver: Subendothelial fibrin deposition
 - Haemolysis
 - Elevation of liver enzymes
 - Low plate ts d/t platelet consumption (HELLP)
- = Neurologi (asospasm & cerebral edg

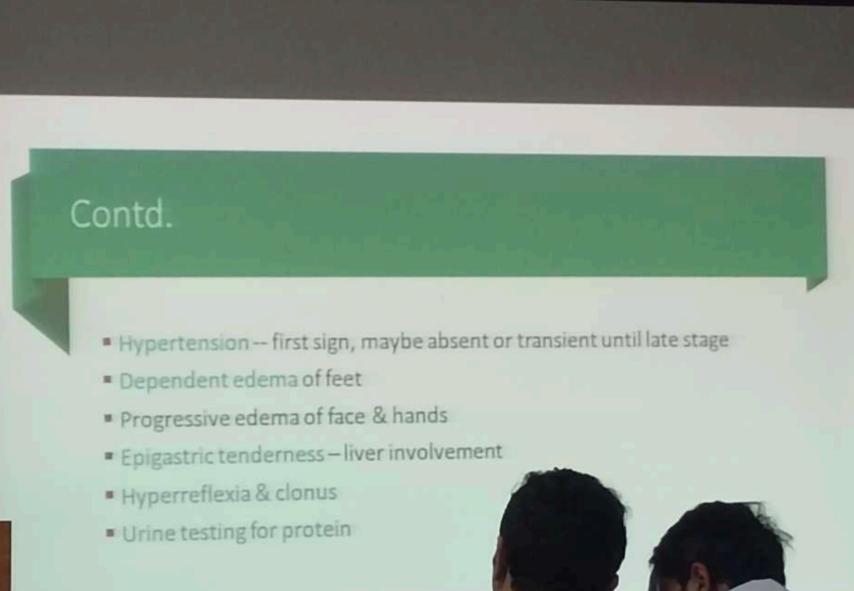


Clinical presentation

Classical symptoms:

- * Frontal headache
- Visual disturbance
- Epigastric pain
- Asymptomatic, flu-like symptoms



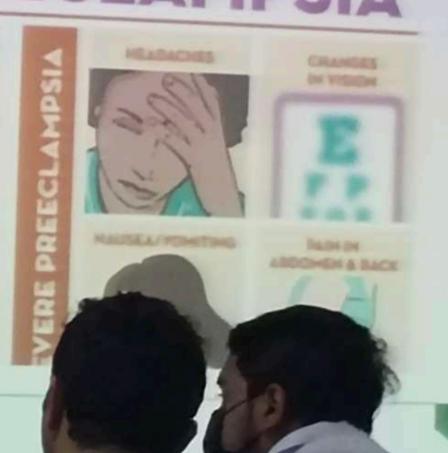


SYMPTOMS OF PREECLAMPSIA

MILD PREECLAPSIA

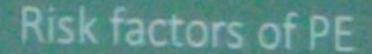






Principles of management

- Early recognition of symptomless syndrome
- " Awareness of the seriousness of the condition in severe form
- Adherence to guidelines for admission to hospital
- Investigations
- Antihypertensives
- Anticonvulsants
- Well timed delivery to pre-empt serious
- Postnatal follow-up
- Counselling for future pregnancy



- First pregnancy
- * Multiparous previous H/O PE
- * PE in previous pregnancy
- * 10 yrs. or more since last baby
- Age 40yrs. or more
- . BMI of 35 or more
- * Family history
- Booking diastolic BP > 80 mmHg
- * Booking proteinuria
- Multiple pregnancy

Risk factors

Maternal conditions (prior to pregnancy)

- * Diabetes
- * HTN
- · Obesity
- · Chronic kidney disease
- * Lupus/antiphospholipid syndrome



Investigations

To monitor maternal complications:

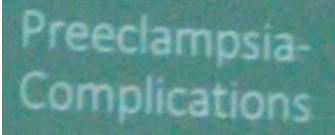
- Full blood count -
 - Haemoglobin
 - Platelet count (falling)
 - Haematocrit (rising)
- " Clotting studies --
- · Serum renal profile urea, creatinine, uriq
- Serum liver profile Bilirubin, SGPT, SGC
- Urine -- Frequent proteinuria quantificat



Treatment of hypertension

Aim:

- To lower the BP & reduce the risk of maternal CVA without reducing uterine blood flow & compromising the fetus
- Methyldopa Centrally acting, oral
- * Labetalol Alpha & beta blocking-first
- Nifedipine Calcium channel blocker, c
- Hydralazine In fulminating disease, IV

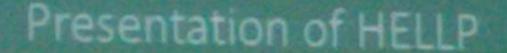


- Pulmonary edema.
- · Heart failure
- * Liver haematoma with/without rupture
- * Liver failure
- * Disseminated intravascular coagulation
- * Stroke
- Dialysis (advanced renal failure)

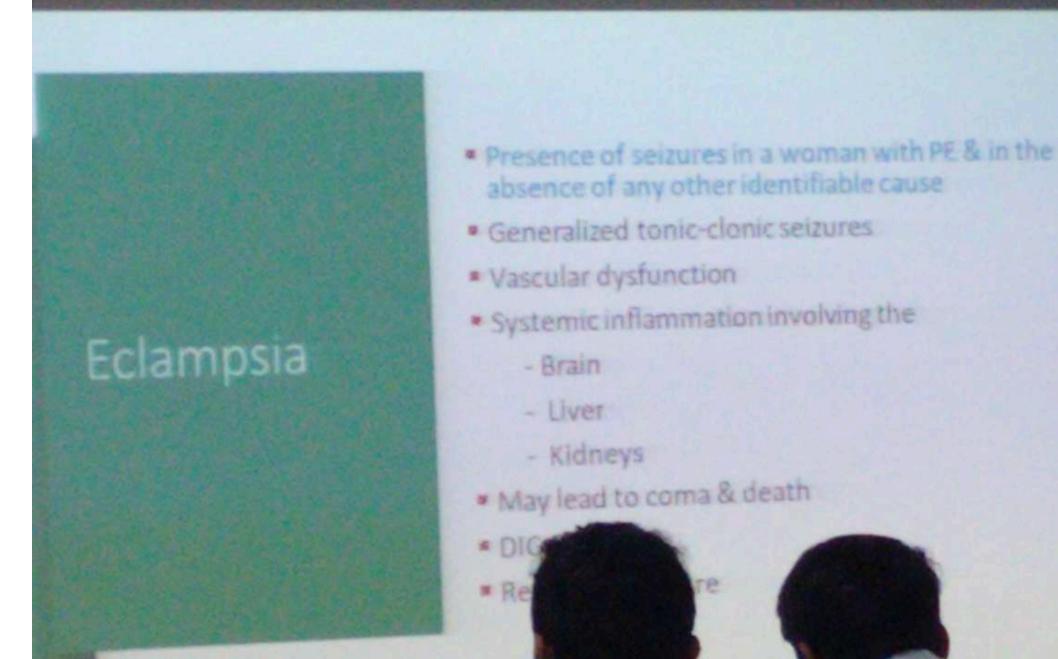
HELLP syndrome

- * Variant of preeclampsia
- * Hemolysis
- * Elevated liver enzymes
- * Low platelet count
- Complication of preeclampsia (severe form)
- * Coagulation activation & liver infarction





- * Typically epigastric pain, nausea & vomiting
- HTN may be mild or absent
- Associated with complications of:
- > ARF
- Placental abruption
- > Stillbirth



Risk factors

- Uncontrolled HTN
- · Primigravida
- Obesity
- Black ethnicity
- = H/O DM
- Age < 20 years



Warning signs

- Epigastric pain & right upper quadrant tenderness
- Headache
- Uncontrolled hypertension
- Agitation
- Hyper-reflexia & clonus
- * Fascial (periorbital) edema
- Poor urine output
- Papilloedema



Management of eclampsia



- * Call senior help & emergency alert team
- * Airway, breathing, circulation
- * Anticonvulsant of choice: Magnesium sulfate
- Loading dose of 4 g is given followed by maintenance of 1 gm/hr. for 24 hrs. after delivery
- * Overdose: Respiratory depression, cardiac arrest
- * Antidote: 10y
- * Definitive to

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