

Hyportoneiya	Disordore	of Pregnancy

□ •5-10% of pregnancies •↑ maternal-fetal morbidity & mortality worldwide • Risk of maternal/fetal injury related to CNS irritability*seizures* placental abruption*-

Preeclampsia

- □ •after 20 weeks •BP > 140/90 x 2 •with or without proteinuria (PCR > 0.3)
- □ severe features: •thrombocytopenia •liver failure (LFTs 2x normal) •new renal insufficiency (serum creatinine > 1.1 mg/dL) •pulmonary edema •new onset cerebral or visual disturbance

Pathophysiology of Preeclampsia

- □ inadequate vascular remodeling → II placental perfusion & hypoxia → endothelial cell dysfunction → vasospasm & ↓ tissue perfusion
- □ •HTN •IUGR •h/a •hyperreflexia •seizures •scotoma •epigastric pain

Preeclampsia RISK Factors

- □ •Primipara < 19 yrs or > 40 yrs •Previous hx of PEC •Family hx of PEC •Multiples •Obesity •African descent
- □ •Pregestational Diabetes •Chronic Hypertension •Renal disease •First pregnancy with new partner •Thrombophilia

Assessment Preeclampsia

34yo G4P3 @ 34 weeks with BP 142/88, 145/90

- □ Labs? → CBC platelets ALT/AST creatinine uric acid u/s NST 24hr urine PCR
- □ Prenatal follow-up? →•Weekly visits with AFI •BP 2x week •NST 2x week •platelets and LFTS weekly •FKC •Consider IOL @ 37 weeks
- □ Counseling? →•Risks of IUGR •abruption •oligohydramnios : •Warning signs: •h/a •visual changes •epigastric pain → risk of seizure

Preeclampsia with Severe Features

- □ •BP >160/>110 •Severe features
- ☐ Hospitalized until birth →•Bedrest •Code cart nearby •Quiet calm low light •Padded side rails?
- □ Frequent assessment Vitals →•q 10 Assess edema, clonus, DTRs •HA, visual changes •Epigastric pain (liver is getting involved) •Foley strict I&O •Fetal well-being •Platelets, liver enzymes
- ☐ If < 34-37 weeks, steroids for lung maturity

Magnesium Sulfate: Seizure Prophylaxis

- □ •Decreases neuromuscular irritability •Decreases CNS irritability •Promotes maternal vasodilation
- □ Watch for magnesium toxicity •Loss of knee-jerk reflexes •Respirations <12 p/min •Urine output <30ml/hr •Cardiac or respiratory arrest
 •Toxic serum levels >9 mg/dL •Therapeutic range 5-9 mg/dL •Sign of fetal distress •Calcium Gluconate is the antidote •10% Calcium gluconate 10cc, IV



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Management of Preeclampsia

□ •MAG: 4g loading dose, then 2g/hr to depress (not eliminate) reflexes •Strict I&O (consider Foley) q hour •BP check q 15-30 mins •Pulse Ox, Lung Sounds •DTRs, Clonus, and hand grasps •FLUID RESTRICTION •Control hypertension •BP meds via IV meds if severe •Continue observations 24-48hrs PP •Symptoms usually resolve within 48 hours PP

Practice Question

☐ You are caring for a 34yo G2P1 who was admitted for IOL at 36 weeks for PEC with severe features. After you administer the Mag Sulfate bolus, the patient reports that she feels "sleepy and a little nauseated." You also notice that the variability of the FHR tracing is now minimal.administer the Mag Sulfate bolus, the patient reports that she feels "sleepy and a little nauseated." You also notice that the variability of the FHR tracing is now minimal.

•What is your first action? •What would you assess? •What would you anticipate? •What monitoring is necessary for this patient?

ECLAMPSIA

- •Onset of seizure activity or coma in pregnancy without CNS lesion •Treat with Magnesium Sulfate + PEC measures
- -Assessment → ↑HTN precedes seizure followed by hypotension and collapse •Coma may occur •Labor may begin, putting fetus in great jeopardy
- □ •Expect postictal non-reassuring FHR tracing. Allow in utero resuscitation for 20-30 mins. □ •C/S risk of maternal cerebrovascular hemorrhage!

Eclampsia

- •Patent airway & patient safety •ABCs •Side rails up •Call for help!Do not leave! •Suction •Prevent aspiration •Fetal Monitoring •Maternal VS
- •Meds^(O2)

Chronic Hypertension in Pregnancy

- ☐ Diagnosis→•Before pregnancy or diagnosed before 20 weeks. •Use of anti-hypertensives before pregnancy
- □ •Monitor →Labs, u/s, NST AFI, IOL (37-38 weeks) •Persists > 12 weeks postpartum
- □ •Risk: IUGR, PTL, placental abruption, renal failure, CHF, CVA, and superimposed PEC •Low dose ASA (12-36wks)
- □ •Mild-moderate: no evidence of improved outcomes with meds •CHTN with superimposed PEC

Chronic HTN with superimposed Preeclampsia

□•HTN before 20 weeks with new onset proteinuria •Worsening HTN plus one

□ 1•New onset of sx □ 2•Thrombocytopenia □ 3•↑liver enzymes □ 4•Pulmonary Edema □ 5•New onset renal insufficiency •↑morbidity for mom & fetus



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Gestational	Ηv	pertension	in	Pregnancy
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□ •Elevated BP > 20 weeks •>140/>90 •No proteinuria •25% will develop PEC •If persists > 12 weeks PP → CHTN

HELLP

- □ •Hemolysis •Elevated •Liver Enzymes •Low •Platelets (<100K)
- □ •Laboratory diagnosis with PEC •Non-specific clinical presentation •Prompt delivery on dx vs. wait 48hrs for steroids if < 37wks
- □ •Life threatening •Pulmonary edema •Acute renal failure •DIC •Abruption •Liver failure, hemorrhage •ARDS •Sepsis •Stroke

PEC, Chronic HTN, Gestational HTN

- □ Preeclampsia •After 20 weeks •BP >140/>90 x2 •Proteinuria and/or severe features
- ☐ Chronic Hypertension •Before 20 weeks •>140/>90
- ☐ **Gestational Hypertension •**After 20 weeks •>140/>90

PEC with severe features, HELLP

- ☐ Preeclampsia with severe features •BP >160/>110 x2 or severe features •Magnesium Sulfate* •Seizure precautions
- ☐ HELLP •May not have s/s of PEC •High maternal and fetal mortality •Progresses rapidly

Medications you need to know (table 27-5)

□ •Labetalol •Nifedipine •Methyldopa •Hydralazine •Magnesium Sulfate •Calcium Gluconate •No ACE inhibitors •Avoid Methergine for PPH

HTN Disorders in Pregnancy

- □ Intrapartum Care → •Maternal-Fetal VS •Continuous EFM •Epidural? •Fluid restriction? •Quiet, dark, environment
- •Emergency drugs, 02 @ 10L, suction ready •Magnesium Sulfate •Calcium gluconate
- □ Adverse Outcomes →•Restricted fetal growth •Placental abruption •Preterm birth •Early degeneration of placenta

Case Study

Your client, Julie, is a G3 P2002 at 39 weeks of gestation. She presented to the high risk labor and delivery triage are an hour ago. Her blood pressure has been steadily increasing for the past 3 weeks. Today her blood pressure was 160/110, and she presents to the triage area with complaints of a severe headache and "spots in my vision." Her cervical exam is 2 cm/80%/-2 firm midposition.

•What type of pregnancy hypertensive disorder do you suspect Julie may have? •What other priority information is it important for the nurse to assess and gather?

Case Study cont.

Julie is admitted to the labor and delivery unit for induction for preeclampsia. The provider orders magnesium sulfate: 4 gram IV loading dose and then 2 grams/hour maintenance dose. Julie asks, "What is this medication for? Will it affect my baby?"

What is the nurse's best reply?



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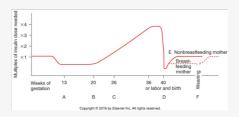
Endocrine & Metabolic Disorders of Pregnancy

□ •Pre-existing DM type 1 & 2 •GDMa-1 •GDMA-2 •Hyperemsis gravidarum •Hyper & hypo thyroidism •PKU

Maternal Insulin ResistancePathophysiology

- □ •Metabolic changes in pregnancy → □ •Normal pregnancy alters maternal glucose metabolism, insulin production, and metabolic homeostacis
- □ •Glucose is the primary fuel for the fetus □ •Glucose crosses the placenta, insulin does not
- \square •Insulin needs \downarrow during the first trimester \square •Risk of hypoglycemia for IDM patients
- □ •Diabetogenic effect in second and third trimesters □ •↑insulin resistance □ •Placental hormones act as insulin antagonists
- □ •Expulsion of the placenta drops insulin requirements

Changing insulin needs during pregnancy.



Gestational Diabetes (GDM)

- □ •Gestational Diabetes ⇒•Common-Hispanic, Native American, Asian, African American •Diagnosed 2nd trimester with 1 and 3 hr. GTT •Screening algorithm •High risk should screen early
- □ •GDMA1- well controlled with diet •GDMA2- need meds

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Gestational DM





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Fetal Implications	
□ •Glucose crosses the placenta •↑ fetal insulin production in response to high glucose from matern □ •Fetal macrosomia •Labor risks •Maternal Risks •Fetal Risks •Newborn Risks	nal circulation
GDM Nursing Plans and Interventions	
□ •Patient Counseling •Pathology of disease •Low-glycemic diet •Exercise •Teach/Demonstrate •Glucose r	monitoring •Insulin Administration
□ •Signs of hypo- and hyperglycemia & immediate actions to be taken if signs noted •Fetal surveilla	nce
NCLEX HINT	
□•Glucose Screen Gold standard is 3hr GTT. □•GDMA1 – Diet controlled GDMA2 – on medication	n (metformin, glyburide, insulin)
Pregestational Diabetes	
riegestational biabetes	
 Monitoring and TX→•Blood Sugar Testing •Dietary Counseling •Exercise •Insulin •Oral hypoglyc Risks and Consequences→•IUFD •Congenital malformations •Macrosomia •RDS •Infections •Po •DKA 	_
Pregestational Diabetes	
□ Intrapartum→ •Testing q hour •Fluids and insulin*(70-100 mg/dl) •Risks? •Polyhydramnios •Macroson	
□ Postpartum→ •First 24hrs: ↓insulin demands •½ dosage of insulin •PPH •Infections •Breastfeeding •	Family planning
Hyperemesis Gravidarum	
□ •Severe and persistent NVP •Weight loss, electrolyte imbalance, nutritional deficiencies and ketor	nuria.
□ •Idiopathic/Multifactorial •Can be a debilitating complex metabolic disorder •Linked to Hydatidiform	n mole
Hyperemesis Gravidarum-Assessment	
□ •Persistent vomiting before 9 weeks •Ketonuria •Dehydration •> 5% weight loss •Altered nutritional	al status •Electrolyte imbalance (hypok-
alemia)	
Hyperemesis Gravidarum Dietary Modification	
□ •Small frequent meals •Don't over eat •Eat what sounds good •Avoid triggers (odors) •Avoid spicy	•Bland, low fat
□ •Cold may be more tolerable than warm •Drink from a cup with a lid and straw •Carbonated bever	ages- real ginger ale
NCLEX HINT	
Research has found that infection by H. Pylori is a possible causative factor in hyperemesis.	
Other pregnancy and non-pregnancy risk factors for hyperemesis include:	
□ •first pregnancy•prior hx HG•hyperthyroid disorders•multiple gestation•trisomy 21•triploidy•obesity	y•female fetus
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Hyper or Hypo thyroid?					
□ HYPER•Rare in pregnancy □ HYPER•Labs: elevated T4 □ HYPO•Risks: PEC, miscarriage, GHTN, placental abruption, preterm birth, stillbirth □ HYPO•Tx: Levothyroxine □ HYPER•Miscarriage, preterm birth, stillborn, infants with goiter, hypo/hyper thyroidism □ HYPO•Med interaction: Fe	☐ HYPER•Thyroid storm ☐ HYPO•Labs: elevated TSH ☐ HYPO•SX: weight gain, lethargy, cold intolerance ☐ HYPER•SX: weight loss goiter, tachycardia ☐ HYPER•Tx: PTU/methimazole ☐ HYPO•Risk: fetal neuropsych damage ☐ HYPER•Breastfeeding issue ☐ HYPER•Med reaction: pruritus skin rash				
PKU					
□ •Inborn error of metabolism caused by an autosomal recessive trait that creates a deficiency in the enzyme phenylalanine hydrolase, which impairs the body's ability to metabolize foods with protein □ •If unrecognized, can cause cognitive impairment □ •Prompt diagnosis and therapy with a phenylalanine-restricted diet significantly decreases the incidence of cognitive impairment. □ •Women with PKU may be advised against breastfeeding because their milk contains a high concentration of phenylalanine.					
Question					
You are counseling a woman with PKU who is planning to go off her birth control. Which statement indicates the need for further teaching? A. "I should eat like a vegan to avoid problems with my baby's brain."					
B. "I'll have to be monitored throughout the pregnancy."					

- C. "I may not be able to breastfeed my baby."
- D. "The placenta will help protect my baby from phenylalanine."



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