Gestational Hypertension, Pre-Eclampsia and HELLP: Big Update

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GH, PEC, HELLP

- Hypertensive disorders of pregnancy, including PEC complicate 10% of pregnancies worldwide
- Incidence of PEC has increased by 25% in the US during the past 2 decades

GH, PEC and HELLP

- At the end of this presentation, the participant will be able to:
 - Discuss current diagnostic criteria
 - Identify the continuum of hypertension in pregnancy
 - Describe current treatment of hypertension in pregnancy

GH, PEC and HELLP

- PEC is the leading cause of maternal and perinatal morbidity and mortality
- Worldwide-estimated 50-60 thousand deaths per year
- Probably 50-100 near misses-not death but bad outcomes

GH, PEC AND HELLP

- Hypertensive disorders of pregnancy are associated with prematurity
- PEC is now well known as future predictor of cardiovascular and metabolic disease

ACOG Task Force on HTN

- Etiology remains unclear-still "The theory of ideas"
- Still need more research-lots of ideas, money spent, but no clear path to identifying and treating-especially early onset PEC

ACOG Task Force on HTN

- Transform info into practice guidelines
- Identify and prioritize the most compelling areas of laboratory and clinical research to bridge gaps in current knowledge
- Task force met 3 times + additional hours

Preventing Maternal Death

- Two of the leading MFM researchers-Steven Clark and Gary Hankins published a paper called:
- PREVENTING MATERNAL DEATH: 10 CLINICAL DIAMONDS

Preventing Maternal Death

- Part of following The Joint Commissons (TJC) Preventing Maternal Death (2010)
- They identified 10 specific recurrent errors that account for the majority of maternal deaths, including severe preeclampsia

Preventing Maternal Death

- Use pulse oximeter
- Cerebral hemorrhage 2nd to uncontrolled HTN remains a leading cause of death in women with PEC
- Untreated BPs > 160/110 before death

Preventing Maternal Death

2 of those diamonds deal with PEC

- #1: A Patient with PEC reporting SOB should undergo a CXR immediately
- Undiagnosed pulmonary edema is a leading cause of preventable maternal death

Preventing Maternal Death

- No hospitalized woman with EITHER 160 systolic OR diastolic 110 will be harmed by a single IV bolus of 5-10 mg hydralazine or 20 mg labetolol
- "There is just no reason to withhold such therapy"

ACOG Task Force on HTN

- 17 experts: OB, MFM, HTN, Internal Medicine, nephrology, anesthesiology, physiology and patient advocacy
- Summarize current state of knowledge of PEC and HTN in pregnancy by reviewing and grading the quality of world studies

ACOG Task Force on HTN

- Eliminate dependence of dx on proteinuria
- Now PEC is dx as HTN in association with:
- Thrombocytopenia (< 100,00)
- Impaired liver function (AST, etc. 2 X nl)

ACOG Task Force on HTN

- HTN during pregnancy has only 4 categories:
- PEC-Eclampsia
- CHTN (of any cause)
- CHTN with superimposed PEC
- GH

ACOG Task Force on HTN

- New: Serum creatinine > 1.1 mg/dL or a doubling of serum creatinine w/ no renal disease
- Pulmonary edema
- New onset cerebral or visual disturbances

ACOG Task Force on HTN

- Get rid of phrase: Mild preeclampsia
- Preeclampsia in any form should never be minimized as mild
- Do not use proteinuria to classify PEC as severe or to decide to induce labor

ACOG Task Force on HTN

- Deliver women with preeclampsia without severe features at 37 0/7 weeks gestation
- Assess for preeclampsia in the pp period
- Educate women about developing preeclampsia after pp discharge

ACOG Task Force on HTN

- Amount of proteinuria has not been shown to predict either maternal or fetal outcomes
- Do not use IUGR as diagnosis of severe preeclampsia
- Use IUGR for indicated delivery if fetus is < 5th percentile

GH, PEC and HELLP

National High Blood Pressure Education Program Working Group, or the "Working Group"

GH, PEC AND HELLP

- Gestational Hypertension
- BP 140 systolic **OR** 90 diastolic or greater
- After 20 weeks gestation and had normal blood pressure
- Starts without proteinuria-25% will develop proteinuria defined as 0.3g/24 hour

GH, PEC and HELLP

- Edema is not a criteria
- Preeclampsia may be associated with many other sign/symptoms
 - Edema
 - Visual disturbances
 - Headache
 - Epigastric pain
 - Others to be discussed by system

GH, PEC AND HELLP

- Proteinuria is defined as the excretion of 300 mg or more of protein in a 24 hour period
- OR a protein/creatinine ratio of at least 0.3 mg/dL-not for diagnostic use unless you cannot get a 24 hour urine

GH

- The 30 over 15 rule
- Taken out in 1996 by ACOG as not enough evidence as prognostic indicator
- However, Working Group says these patients "warrant close observation"

GH

- I love these people!
- 30-15 rule is defined as "an elevation of more than 30 mm Hg systolic **OR** more than 15 mm Hg diastolic above the patient's baseline blood pressure"

History

- Many different treatment options
 - Bed rest has always been the hallmark
 - Diets of different kinds were tried
 - ■Tomato soup and baked potatoes
 - ■Calcium

History

- Let us look at some history
- The term was **toxemia** for years
- Toxemia led to convulsions

History

- Bed rest was at home first 1/3 of 1900's
- Clothespin for airway management
 - Supposedly prevented swallowing of tongue
 - No "squeezy" kind of clothespins
 - Pt was told to place it on her tongue if she had a convulsion

History

- BP monitored by MD at home
- A glass of Epsom salts q day
 - Cathartic-it was given to clear the toxins
 - Does oral mag sulfate prevent seizures?

History

- Delivery was the only cure
- Patients were delivered if they got worse
- Induced with various agents
 - Quinine
 - Castor oil
 - Combination of the two
 - Buccal oxytocin
 - Others I do not know about-do you??

History

- High maternal and fetal mortality rates
- Maternal death from:
 - Eclampsia
 - Cerebral hemorrhage
 - Massive cerebral edema
 - Liver rupture

Incidence

- Increased incidence of eclampsia in the intrapartum period
- 50% Intrapartum
- 25% Antepartum
- 25% Postpartum

Outcomes

- Fetal mortality if maternal mortality
- Fetal death from anoxia during eclamptic seizure
- Neonatal death from prematurity

Eclampsia

- The presence of new onset grand mal seizures in a woman with preeclampsia
- Rule/out other causes of seizures, i.e. bleeding A-V malformation, idiopathic seizure disorders

Etiology

- Theories on etiology
 - The disease of theories
 - My favorite: The parasite theory
 - Vasoactive substances, immunology, genetics and you have to have a placenta

Superimposed Preeclampsia

- New-onset proteinuria in a woman with hypertension prior to 20 weeks gestation
- Sudden increase in proteinuria if already present
- Sudden increase in hypertension

Superimposed preeclampsia

- The development of HELLP syndrome
- The development of HA, scotomata or epigastric pain may be indicators of superimposed preeclampsia

Severe Preeclampsia

- Proteinuria of 5 g or > in a 24 hour specimen or 3+ or > on 2 urine samples collected at least 4 hours apart: No more!
- Oliguria of < 500 cc in 24 hours: Not addressed
- Cerebral or visual disturbances: New

Severe Preeclampsia

- BP 160 mm Hg systolic **or** > 110 mm Hg diastolic on 2 occasions at least 6 hours apart while the patient is on bed rest
- However if the patient was just admitted with a BP of 242/140 I am not going to wait!

Severe Preeclampsia

- Pulmonary edema or cyanosis
- Epigastric or right upper quadrant pain
- Impaired liver function
- Thrombocytopenia

Severe Preeclampsia

- Fetal growth restriction: Removed as a sign of severe PEC
- BUT: may be the first sign of preeclampsia
- May be seen with unexplained increased AFP testing

HELLP syndrome

- Acronym developed by Dr Weinstein in 1982 to help his patients get diagnosed correctly in the ER
- Hemolysis, Elevated Liver Enzymes, and Low Platelet count

Severe preeclampsia

- May be very remote from term
- Oligohydramnios may develop
- Increased risk of fetal intolerance to labor
- Increased cesarean birth rate

HELLP syndrome

- Just a different presentation of preeclampsia
- Usually present in late 2nd or early 3rd trimester
- Nausea/vomiting/epigastric pain

HELLP Syndrome

- Often misdiagnosed
- Blow up (my term) several days later when symptoms of preeclampsia/eclampsia appear
- 20% of women with severe preeclampsia develop HELLP syndrome

HELLP Syndrome

- Same for women with HELLP syndrome at 34 0/7 weeks (after mom is stabilized)
- For women with HELLP from gestational age of fetal viability to 33 6/7 weeks-delay delivery if mom and fetus stable to complete course of steroids

HELLP Syndrome

- New recommendations:
- For women with HELLP syndrome and before the gestational age of fetal viability-delivery be undertaken shortly after initial maternal stabilization

Epidemiology and Risk Factors

- Usually seen in first pregnancy
- If severe preeclampsia or HELLP syndrome, higher recurrence rate
- Increased risk with multifetal gestation

Risk Factors

- Chronic hypertension
- Type 1 and 2 diabetes mellitus
- Connective tissue disease
- Vascular disease

Risk Factors

- Nephropathy
- Antiphospholipid antibody syndrome
- Obesity
- Age less than 19 years or greater than 35 years

Pathophysiology

- Who knows why?
- Decrease in circulating plasma volume
- Hemoconcentration
- Endothelial injury

Risk Factors

- African-American
- Samoan
- Family history
- New partner

Risk factors

- Hydatidiform mole
- Unexplained elevated MSAFP
- Nonimmune fetal hydrops

Pathophysiology

- This results in hemoconcentration
- Now, at the same time the body is responding to all sorts of vasoactive substances

Pathophysiology

- Let's walk through the process
- Women with GH or preeclampsia have an inadequate increase in plasma volume
- Normally increase 40-50%, these patients average 9-40% below pregnancy norms

Pathophysiology

Prostacyclin is a vasodilator

Thromboxane A₂ is a potent vasoconstrictor

Nitric oxide is a potent vasodilator

Endothelins are potent vasoconstrictors

- This results in severe vasospasm
- Which causes endothelial injury
- Which allows colloid particles and fluid to move into the extravascular space
- I Refuse to get into Vitamin D

Pathophysiology

- We also know that patients who develop preeclampsia have a higher cardiac output
- Theorized that this might cause endothelial damage over time
- Then complicated by vasospasm

Pathophysiology

- This decreases the circulating volume even more
- Decreased perfusion to maternal organs, including the uterus which currently has a placenta
- May cause IUGR more than HTN does

Pathophysiology

- So how will this affect the patient's various systems?
- We know endothelial damage is occurring and that fluid can move easily out of the intravascular space

- Hematologic System
- Hemoconcentration results in decreased tissue perfusion
- Platelets are the first line of defense for endothelium injured from vasospasm
- Platelets are rapidly used up-may result in thrombocytopenia

Pathophysiology

- Renal changes
 - Decreased glomerular filtration rate
 - Increased serum creatinine
 - Decreased creatinine clearance

Pathophysiology

- Hemolysis can occur as RBC destroyed moving through spasming vessels
- Microangiopathic hemolytic anemia (MHA) develops-Burr cells and shistocytes seen on peripheral smear
- Decreases oxygen carrying capacity even further

Pathophysiology

- Renal changes
 - BUN increases
 - Uric acid increases
 - Urine output decrease

- Renal changes
 - Edema increases
 - This can include pulmonary edema

Pathophysiology

- Hepatic changes
 - Elevated LFT's
 - Hyperbilirubinemia may be seen, especially with hemolysis
 - Subcapsular hematoma

Pathophysiology

- Renal changes
 - Persistent oliguria may lead to ATN and eventually renal failure
 - Rare, but seen in some severe cases

Pathophysiology

- Hepatic changes
 - Liver rupture may occur
 - Very high mortality rate
 - Thankfully, rare

- Neurological changes
 - Eclampsia-tonic-clonic seizure activity, woman not breathing. Average FHR decel is nine minutes. Don't go to stat C/S!!
 - Temporary blindness-in patients with DM may be permanent if already have retinopathy

Pathology

- Neurological changes
 - Scotomata
 - Hyperreflexia
 - Mental status

Pathology

- Neurological changes
 - Headache
 - Cerebral edema
 - Blurred vision

Pathology

- Fetal changes
 - Maternal side of spiral arteries spasm
 - May cause spiral artery infarction
 - Double whammy-decreased maternal circulating volume and decreased number of spiral arteries

Pathology

- Fetal changes
 - Triple whammy-same as above and maternal hemolysis decreasing amount of oxygen available
 - IUGR

Management

- Expectant vs. emergent
- Anticipatory nursing
- Never forget COP
- Preventative treatment a bust so far

Pathology

- Fetal changes
 - Oligohydramnios
 - Abruption
 - Nonreassurring fetal heart rate

Management

- Determine blood pressure accurately!
 - Appropriate size cuff: encircles 80% or > of the upper arm
 - Pt sitting position after 10 minute rest period antenatally

■ BP Measurement

- In-house: Left lateral position with arm at the level of the heart or sitting position if OK
- Automatic cuffs OK: Know in they measure lower. Sphygmomanometer remains gold standard

Management

- Assess CNS status
 - Assess deep tendon reflexes
 - Can seize even if normal
 - Check together at change of shift!

Management

■ BP Measurement

- If automatic cuff make sure arrow is over brachial artery. PALPATE THE BLOODY THING!
- If not, listen for Korotkoff phase V sound which is when the sound disappears. That is the diastolic

Management

■ CNS status

- Assess for visual changes
- Mental status-be careful because MgSO4 can mask this especially in combination with medications used for labor pain

- Assess fetal status
 - EFM for these patients
 - Low threshold for reporting nonreassurring characteristics on tracing

Management

- Accurately record I+O!!!!
- Everything on a pump
- Foley with a urimeter if not up voiding, worsening BP or decreasing output

Management

- Assess volume status
 - Need to increase circulating volume
 - But, what we put in will leak out to extravascular space-it's OK, just know that!
 - Lactated Ringer's is fine. No colloids!

Management

- Assess pulmonary status
 - Assess breath sounds before pulmonary edema develops from decreased COP, endothelial damage and IV fluids
 - Orthopnea and/or anxiety are huge signs of impending or present pulmonary edema

- Follow labs q 6 hrs to weekly
- Fetal surveillance daily/twice weekly
 - Rhythm strips vs. NST/AFI or BPP
 - Serial sonars for growth/amniotic fluid volume

Management

- Now to stop your hearts!
- For women with PEC with systolic BP < 160 AND a diastolic of < 110 AND NO MATERNAL SYMPTOMS magnesium sulfate need not be administered universally to prevent eclampsia

Management

- Anticonvulsant agents
 - MgSO4 is agent of choice: 4-6 gm loading dose then 1-3 gm/hr
 - Therapeutic levels 4-7 mg/dL
 - Watch DTR's and Mag levels

Management

- Antihypertensive agents
- Labetolol: 20 mg followed by 40 mg if not effective within 10'; then 80 mg q 10' to maximum dose of 220 mg
- Hydralazine: 5-10 mg bolus IVP q 20' prn

- Antihypertensive treatment begun when BP diastolic 105-110 mg Hg or higher
- Goal of therapy is to reduce diastolic BP to 90-100 mg Hg
- Below that blood flow to fetus decreased

Management

- Delivery with preeclampsia or eclampsia
- Make sure mama is stable
- Induction is fine
 - All depends on obstetrical indications

Management

- Seizure management
 - ABC
 - Fetal brady already mentioned
 - Do not deliver immediately!

Management

Invasive hemodynamic monitoring may be utilized in preeclamptic patients with severe cardiac disease, severe renal disease, refractory oliguria, HTN or pulmonary edema

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