Gestational Hypertension, Pre-Eclampsia and HELLP: Big Update

MARY ELLEN BURKE SOSA, RNC-OB, MS

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
Part of following The Joint Commission’s (TJC) Preventing Maternal Death (2010)

They identified 10 specific recurrent errors that account for the majority of maternal deaths, including severe preeclampsia

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

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

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”
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”

Many different treatment options

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

Let us look at some history

The term was toxemia for years

Toxemia led to convulsions

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

- 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

- 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

- 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

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

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

Same for women with HELLP syndrome at 34 0/7 weeks (after mom is stabilized)
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
Pathophysiology

- 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
Pathophysiology

- 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
Pathophysiology

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
Pathophysiology
- 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
  - Nonreassuring 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
Management

BP Measurement

- In-house: Left lateral position with arm at the level of the heart or sitting position if OK
- Automatic cuffs OK: Know 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
Management
- Assess fetal status
  - EFM for these patients
  - Low threshold for reporting nonreassuring 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
Management

- 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’ pm
Management
- 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
References


