HYPERTENSIVE DISEASE AND PREGNANCY

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Purpose

Define the diseases and risks
Review evidence-based medicine and consensus on managing women

• With hypertension who become pregnant
• Who develop hypertensive disorders during gestation
Historical Perspective

- Mauriceau 1673 - primip > multip; eclampsia distinct from epilepsy
- De Sauvages 1739 - “eclampsia”
- Lever 1843 - proteinuria; not renal disease
- Ballantyne 1885 - ↑ BP
- Lazar 1925 - MgSO₄
- Herrick 1926 - preeclampsia superimposed on HTN, renal disease
- De Lee 1930-40’s
Classification

Based on JNC VI definition for HTN:

\[ \text{SBP} \geq 140 \text{ mmHg} \]

or

\[ \text{DBP} \geq 90 \text{ mmHg} \]

K5 determines DBP
Definitions

- **Preeclampsia-Eclampsia:** $\uparrow BP$ and protein
  - $> 20$ weeks
  - $\uparrow SBP \geq 140$ mm Hg, $\uparrow DBP \geq 90$ mm Hg
  - Proteinuria $\geq 0.3$ g/24 hrs or 1+ on dip

- **CHTN:**
  - Present prior to pregnancy
  - or Diagnosed $< 20$th week
  - or Diagnosed $> 20$ wks, persists $> 12$ wks pp
Definitions

• **Preeclampsia Superimposed upon CHTN:**
  – Established diagnosis of CHTN
  – Sudden \( \uparrow \) BP over well-controlled baseline
  – New or \( \uparrow \) proteinuria
  – Platelets < 100,000 c/mm\(^3\) \( \uparrow \) or AST or ALT

• **Gestational HTN:** only until a more specific diagnosis assigned postpartum
  – \( \uparrow \) BP in pregnancy > 20 wks
  – No proteinuria
  – BP normal by 12 wks pp: diagnosis is Transient HTN
  – BP remains high at 12 wks pp: diagnosis is CHTN
Maternal Mortality and Hypertension

**Developing Nations**
- Hemorrhage: 20%
- Sepsis: 40%
- HTN: 15%
- Other: 25%

100-800 / 100,000 (deaths/birth)

**Developed Nations**
- Embolism: 20%
- Abortion: 17%
- Sepsis: 8%
- Hemorrhage: 13%
- HTN: 17%
- Other: 25%

12 / 100,000 (deaths/birth)
Risk Factors for Preeclampsia-Eclampsia

- Primigravid 6-8X
- Twins 5X
- Diabetes
- Molar pregnancy 10X
- Hydrops 10X
- Family history 2-3X
  - Mother with severe in 1st pregnancy 3-4X
  - Sister with severe in 1st pregnancy 3-6X
- Extremes of age
Pathophysiology of Preeclampsia

Cause: Unknown

Characterized by:

- Vasospasm
- Activation of coagulation system
- Perturbations in humoral and autacoid systems related to volume and BP control
- Oxidative stress and inflammatory-like responses
- Pathologic changes ischemic in nature
Pathophysiology: Placenta

- Normal pregnancy:
  - Spiral arterioles invaded by endovascular trophoblast
  - Flaccid diameter, flaccid,
  - Sac-like vessels

- Preeclampsia:
  - Invasion incomplete
  - Failure to re-model: thick walled, muscular arterioles
  - Acute atherosis in basal arteries: necrosis, foam cells
  - Decreased perfusion, early placental hypoxia, infarction
Pathophysiology of Preeclampsia

↑ Blood Pressure

• Vasoconstriction:
  – Marked ↑ in PVR
  – May have slight ↑ “normal” BP by 20th wk
  – Hyper-responsive to vasoactive peptides and amines

• Mechanisms:
  – Prostanoids ↑
  – ↓ activity NO-synthase and ↓ EDRF
  – Inflammatory cytokines
  – Oxidative stress
Pathophysiology: Renal

- Glomerular capillary endotheliosis
- GFR and renal blood flow
- Proteinuria: nonselective, late in clinical course
- Hyperuricemia (marker for preeclampsia)
- Hypocalciuria, altered Ca\(^{+2}\) regulatory hormones
- Impaired Na\(^{+}\) excretion, suppression of renin-angiotensin system: fluid retention, edema
- Plasma volume, hemoconcentration
Pathophysiology: Coagulation System

- Activation coagulation system
  - Procoagulants
    - Thrombocytopenia
      - Most common hematologic abnormality
      - Platelets < 100,000 cells/mm³: serious disease
      - Fetal platelet count unaffected
    - Fibrinogen
    - Antithrombin III
  - FDP
  - Microthrombi
**Pathophysiology: Cardiac**

- **Normal:** CO↑, HR, AC↓, TPR, BP, nl contractility

- **Preeclampsia:**
  - CO↓, TPR↑, nl load-independent contractility (Wallenburg et al, Lang et al)
  - CO↑, TPR↓; “cross over” later (Easterling et al, Boslo et al)

- **HELLP:** subendocardial hemorrhages

- **Cardiac decompensation:** preexisting heart disease
Preeclampsia: Hemodynamic Changes

- **extracellular fluid edema**
  - Endothelial damage, capillary leakage
  - Plasma colloid oncotic pressure
  - Interstitial colloid osmotic pressure

- **Pulmonary edema**
  - *left-sided filling pressures*
  - *CO*
  - Capillary permeability
  - Associated with excessive crystalloid, colloid, b-methasone
  - More common: older, CHTN, obesity
Pathophysiology: Hepatic

- Hemorrhagic lesions
- Infarction
- ↑ ALT, AST, LDH
- HELLP syndrome: hemolysis, ↑ LFT’s, ↓ platelets
  - Markedly ↑ ALT, AST, LDH
  - Subcapsular bleeding
  - Hepatic rupture
**Pathophysiology: CNS**

- **Headache**
- **Visual disturbances**
  - Blurred vision
  - Scotomata
  - Cortical blindness
- **Convulsions**: cerebral vasospasm
- **CT or MRI**: normal vs transient abnormalities: cerebral edema, hemorrhage, global ischemia induced by vasospasm
- **Pathology**: Hemorrhages, petechiae, vasculopathy with vessel wall damage, fibrinoid necrosis, ischemic damage and microinfarcts
Distinguish preeclampsia from chronic or gestational hypertension:

- **Systemic syndrome**: common pathogenetic factor to all organs: *poor tissue perfusion* 2° to *profound vasospasm*

- "Nonhypertensive" complications can be life-threatening when BP elevations are mild!
New Players in Preeclampsia

• sFlt1 – receptor soluble fms-like tyrosine kinase 1
  – Produced by placenta
  – Binds VEGF and PlGF
  – Results in dysfunctional vasculature, endothelium
• VEGF- vascular endothelial growth factor
• PlGF – placental growth factor

• What up-regulates sFlt1 expression?
a) Optical density units vs. VEGF (pg/ml)

b) Optical density units vs. PIGF (pg/ml)

c) Free VEGF (pg/ml)

Free VEGF (pg/ml) for Normal (11), Mild PE (11), Severe PE (10), Preterm (6)

- Normal: ~15 pg/ml
- Mild PE: ~5 pg/ml
- Severe PE: ~1 pg/ml
- Preterm: ~10 pg/ml

- * denotes significant difference
- ** denotes highly significant difference

d) Free PIGF (pg/ml)

Free PIGF (pg/ml) for Normal (11), Mild PE (11), Severe PE (10), Preterm (6)

- Normal: ~600 pg/ml
- Mild PE: ~200 pg/ml
- Severe PE: ~50 pg/ml
- Preterm: ~600 pg/ml

- * denotes significant difference
- ** denotes highly significant difference
# Blood pressure and proteinuria in rats

<table>
<thead>
<tr>
<th></th>
<th>$n$</th>
<th>Mean arterial pressure (mmHg)</th>
<th>Urine albumin/creatinine ratio ($\mu g/mg$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fc (pregnant)</td>
<td>5</td>
<td>75 ± 11</td>
<td>62 ± 21</td>
</tr>
<tr>
<td>sFlt1 (pregnant)</td>
<td>4</td>
<td>109 ± 19&lt;sup&gt;A&lt;/sup&gt;</td>
<td>6923 ± 658&lt;sup&gt;B&lt;/sup&gt;</td>
</tr>
<tr>
<td>sFlk1-Fc (pregnant)</td>
<td>4</td>
<td>73 ± 15</td>
<td>50 ± 32</td>
</tr>
<tr>
<td>Fc (nonpregnant)</td>
<td>5</td>
<td>89 ± 6</td>
<td>138 ± 78</td>
</tr>
<tr>
<td>sFlt1 (nonpregnant)</td>
<td>6</td>
<td>118 ± 13&lt;sup&gt;A&lt;/sup&gt;</td>
<td>12947 ± 2776&lt;sup&gt;B&lt;/sup&gt;</td>
</tr>
<tr>
<td>sFlk1-Fc (nonpregnant)</td>
<td>4</td>
<td>137 ± 2&lt;sup&gt;A&lt;/sup&gt;</td>
<td>2269 ± 669&lt;sup&gt;B&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Pregnant and nonpregnant rats were administered adenovirus expressing Fc (control), sFlt1, or sFlk1-Fc protein. Mean arterial blood pressure (diastolic plus one third of the pulse pressure in mmHg) ± SEM and mean urine albumin/creatinine ratio (micrograms of albumin per milligram of creatinine) ± SEM were measured 8 days after adenoviral administration corresponding to the early third trimester in the pregnant rats. $^A P < 0.05$ and $^B P < 0.01$ as compared with the control group (Fc). Mean plasma sFlt1 levels were 388 ng/ml (pregnant) and 101 ng/ml (nonpregnant) in the sFlt1-treated rats. Mean plasma sFlk1 levels were 775 ng/ml (pregnant) and 1000 ng/ml (nonpregnant) in the sFlk1-Fc-treated rats.
a) Normal pregnancy

- Vessel remodeling
- Blood flow increases
- Normoxia
- sFlt-1

Maintenance levels of free VEGF and PIGF

- Normal endothelial function
- Maternal vessel

Normal organ function
- No hypertension
- Normal glomerular function
- No proteinuria
- No brain edema
- No liver edema
- No coagulation abnormalities

b) Preeclampsia

- No vessel remodeling
- Blood flow decreases
- Hypoxia
- sFlt1

- Free VEGF and PIGF
- Endothelial dysfunction

Multi-organ disease
- Hypertension
- Glomerular dysfunction
- Proteinuria
- Brain edema
- Liver edema
- Coagulation abnormalities
Prevention of Preeclampsia

Unproven Benefit:
- Low-dose aspirin
- Calcium supplements
- Magnesium supplementation
- Fish oil
- Antihypertensive agents
- Diuretics
- Low sodium diet

Possible Benefit:
- Vitamins C and E: encouraging results in HR
Zuspan and Ward, 1964
on the treatment of the eclamptic gravida:

“she has been blistered, bled, purged, packed, lavaged, irrigated, punctured, starved, sedated, anesthetized, paralyzed, tranquilized, rendered hypotensive, drowned, been given diuretics, had mammectomy, been dehydrated, forcibly delivered, and neglected.”
Clinical Implications of Preeclampsia

- Range: mild to severe
- Progression: slow or rapid
  - hours ➔ days ➔ weeks

Clinical management of preeclampsia: over-diagnose to prevent maternal and perinatal morbidity and mortality!

Key:
Differential Diagnosis for Preeclampsia

- Chronic HTN:
  - Essential
  - Secondary
    - Renal disease
    - Reno-vascular HTN
    - 1º aldosteronism
    - Cushing syndrome
    - Pheochromocytoma
- Gestational HTN
- Intrinsic renal disease
- Systemic disease:
  - IDDM
  - Collagen vascular disease
  - Hyperthyroidism
- Cocaine abuse
Early Recognition of Preeclampsia

**Early signs:**
- ↑ BP in late 2nd or early 3rd trimester
- BP changes in absence of proteinuria
- Proteinuria late sign: progression of disease

**Baseline lab data**
- Hct: ↑ 2° hemo-concentration, ↓ 2° hemolysis
- Blood smear: schistocytes
- Platelets: ↓ severe preeclampsia
- Urinalysis: > 1+ protein, do 24 hr urine
Early Recognition of Preeclampsia

- Baseline lab data (cont)
  - Creatinine: ↑ or rising: 24 hr clearance
  - Uric acid: ↑ indicates disease severity
  - SGOT(AST): ↑ suggests severe preeclampsia
  - LDH: ↑ with hemolysis, hepatic involvement
  - Albumin: ↓ \(2^\circ\) capillary leak, hepatic involvement
Early Recognition of Preeclampsia

- **Gauge rate of progression**
  - Ambulatory Management - Mild
    - Office exam in 24-48 hrs
    - Home bedrest, BP and urine protein
    - Laboratory data
  - Subsequent observations:
    - data and progression
  - Hospitalize for worsening of disease
Early Recognition of Preeclampsia

- Gauge rate of progression
  - Hospital Management - Severe
    - Bedrest
    - Intensive BP monitoring
    - Daily weights, Strict I and O’s
    - Laboratory tests
    - Monitor signs and symptoms
    - Fetal assessment
  - Delivery
Preeclampsia

- Fetal assessment:
  - US for growth
  - Kick counts
  - NST
  - BPP and AFI’s
  - Doppler blood flow
Ominous Signs of Preeclampsia

- SBP persistently $\geq 160$ or DBP $\geq 110$ mm Hg
- Proteinuria $\geq 2.0$ g/24 hrs (2+ or 3+ on dip)
- Creatinine $\geq 1.2$ mg/dl
- Platelets < 100,000 per ul
- Microangiopathic hemolytic anemia/↑LDH
- ↑hepatic enzymes
- Persistent HA, visual, cerebral disturbances
- Persistent epigastric pain, N/V
Preeclampsia: HELLP Syndrome

- Hemolysis
- Elevated Liver enzymes
- Low platelets

Maternal Complications:
- DIC 21%
- Abruption 16%
- Liver hematoma
- PP hemorrhage
- Renal failure 8%
- Pulmonary edema 6%
- Cerebral edema
- ARDS
- Cerebral hemorrhage (45% of all mortalities)
Differential Diagnosis for Severe Preeclampsia/HELLP

- HELLP Syndrome
- Thrombotic Thrombocytopenic Purpura (TTP)
- Hemolytic Uremic Syndrome (HUS)
- Acute Fatty Liver of Pregnancy (AFLP)

<table>
<thead>
<tr>
<th>Clinical/Laboratory Finding</th>
<th>HELLP</th>
<th>TTP</th>
<th>HUS</th>
<th>AFLP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>77%</td>
<td>rare</td>
<td>present</td>
<td>25–50%</td>
</tr>
<tr>
<td>Proteinuria</td>
<td>90%</td>
<td>variable</td>
<td>present</td>
<td>variable</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>100%</td>
<td>100%</td>
<td>50% (onset)</td>
<td>variable</td>
</tr>
<tr>
<td>LDH</td>
<td>↑</td>
<td>↑</td>
<td>↑↑</td>
<td>↑</td>
</tr>
<tr>
<td>PT and PTT</td>
<td>↔</td>
<td>↔</td>
<td>↔</td>
<td>↑</td>
</tr>
<tr>
<td>Fibrinogen</td>
<td>↔</td>
<td>↔</td>
<td>↓</td>
<td>↓</td>
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<tr>
<td>Fibrin degradation products</td>
<td>↔</td>
<td>↔</td>
<td>↑</td>
<td>↑</td>
</tr>
<tr>
<td>Antithrombin III</td>
<td>↓</td>
<td>↔</td>
<td>↔</td>
<td>↓</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>↑</td>
<td>↑</td>
<td>↑</td>
<td>↑↑</td>
</tr>
<tr>
<td>Ammonia</td>
<td>↔</td>
<td>↔</td>
<td>↔</td>
<td>↑↑</td>
</tr>
<tr>
<td>Hyperglycemia</td>
<td>↔</td>
<td>↔</td>
<td>↑↑</td>
<td>↓↓</td>
</tr>
<tr>
<td>Renal abnormalities</td>
<td>↑</td>
<td>↔</td>
<td>↑↑</td>
<td>↑</td>
</tr>
<tr>
<td>Neurologic abnormalities</td>
<td>↑</td>
<td>↑↑</td>
<td>↔</td>
<td>↑</td>
</tr>
<tr>
<td>Fever</td>
<td>↔</td>
<td>↑</td>
<td>↑</td>
<td>↔</td>
</tr>
</tbody>
</table>
Indications for Delivery in Preeclampsia

**Maternal criteria**
- Severe $\geq 34$ wks
- Persistent HA, visual disturbance, epigastric pain
- ![Platelets < 100,000]
- Deteriorating liver or renal function
- HELLP syndrome
- Eclampsia

**Fetal criteria**
- Fetal distress
- Non-reassuring testing
- Oligohydramnios
- Severe IUGR
Preeclampsia

• Route of delivery
  – Vaginal preferred
  – Aggressive labor induction; deliver within 24 hrs

• Anesthesia
  – Epidural, spinal
    • Safely used in most gravidas delivering vaginally
    • Cesarean: risk of extensive sympatholysis, hypotension, CO and placental perfusion
  – General: ↑BP with laryngoscopy, intubation, emergence, extubation
**Preeclampsia: Seizure Prophylaxis**

**MgSO₄**: Drug of choice
- Lucas et al 1995: >2000 preeclamptics randomized, MgSO₄ vs phenytoin; MgSO₄ superior in preventing seizures
- Coetzee et al 1998: 685 preeclamptics, RCT MgSO₄ vs placebo; 10X ↓ in eclampsia with MgSO₄
- Collaborative Eclampsia Trial, 1995: 1680 eclamptics randomized to MgSO₄ vs phenytoin or diazepam; MgSO₄ superior to both
- Mild preeclampsia: unclear benefits of MgSO₄
Preeclampsia: Seizure Prophylaxis

Parenteral MgSO₄ for 24 hours:
- 4 g IV loading, 2 g/hr IV maintenance
- 4 g IV loading, 10 g IM, then 5 g Q 4 hrs

Avoid Toxicity:
- +DTR
- Adequate urine output
- No respiratory depression
- Mg levels: 4-7 mEq/L (4.8-9.6 mg/dl)
Preeclampsia: Acute Severe HTN Intrapartum Treatment

SBP ≥ 160 mmHg and/or DBP ≥ 105 mmHg

- Parenteral hydralazine first-line
- Parenteral labetalol second-line (avoid in asthma and CHF)
- Oral nifedipine: caution with MgSO₄, not FDA approved
- Sodium nitroprusside used rarely if no response; risk of fetal cyanide poisoning
Is there a role for conservative management of preeclampsia?

**Goal**

- Prevent eclampsia
- Reduce perinatal morbidity and mortality
- Avoid severe maternal complications

• Delivery
  - Always appropriate for Maternal well-being
  - May not be so for Fetal well-being
Conservative Management of Severe Preeclampsia

- **32-34 wks**: consider delivery; marginal benefit from conservative management
- **24-28 wks**: no randomized trials, conservative management superior, but can be hazardous (Sibai et al, 1990)
- **HELLP and eclampsia**: delivery indicated
Selection of Appropriate Candidates for Conservative Management

- IV MgSO$_4$, 4 g loading, then 2 g/hr
- B-methasone for fetal lung maturity
- Antihypertensive medications
  - IV hydralazine or labetalol
  - Oral aldomet, hydralazine or labetalol
- Strict I and O’s, weight
- Ultrasound for EFW, AFI, BPP, Doppler
- Continuous FHR monitoring
Postpartum Counseling and Follow-up

Risk of recurrent preeclampsia with:

- Multiparas preeclamptic
- African Americans
- Preeclampsia before 30 weeks: 40%
  - May be risk vascular thrombosis
  - Homocystiene, LA, ACA, b-2-glycoprotein, Factor V Leiden, Factors II and VIII, Protein C, S
- Previous HELLP: 25-50%
- Previous superimposed preeclampsia upon CHTN: up to 70%
Remote Prognosis
Preeclampsia-Eclampsia:

* not a cause of essential HTN *

risk essential HTN later in life if

• Severe early onset disease
• HTN occurs in subsequent pregnancy
• Multiparas develop preeclampsia
• Gestational/Transient HTN in any pregnancy
Pre-pregnancy counseling:

- Evaluate using JNC VI criteria
- Determine if
  - **Essential**: 90%
  - **Secondary**: 10% renal disease, renovascular hypertension, primary aldosteronism, Cushing syndrome, pheochromocytoma, DM, SLE, ect
- Evaluate for target organ damage
- Discontinue ACE inhibitors and ARBs
- Discontinue tobacco, alcohol
- Discuss lifestyle changes
# JNC VI Classification of Blood Pressure for Adults

<table>
<thead>
<tr>
<th>Category</th>
<th>SBP (mm Hg)</th>
<th>DBP (mm Hg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Optimal</td>
<td>&lt; 120</td>
<td>&lt; 80</td>
</tr>
<tr>
<td>Normal</td>
<td>&lt; 130</td>
<td>&lt; 85</td>
</tr>
<tr>
<td>High-normal</td>
<td>130–139</td>
<td>85–89</td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stage 1</td>
<td>140–159</td>
<td>90–99</td>
</tr>
<tr>
<td>Stage 2</td>
<td>160–179</td>
<td>100–109</td>
</tr>
<tr>
<td>Stage 3</td>
<td>≥ 180</td>
<td>&gt; 110</td>
</tr>
</tbody>
</table>

When SBP and DBP fall into different categories, use the higher category.
Mild - Moderate CHTN: Management

- Consider d/c anti-hypertensives first visit
  - No evidence Rx improves fetal outcomes
  - Concern for ↓ utero-placental perfusion
- Home BP monitoring
- More frequent prenatal visits
  - Serial lab evaluation
  - Serial sonography for growth
- Pharmacologic Rx if significant ↑ BP
- Fetal testing: NST; no evidence better outcomes
Mild - Moderate CHTN: Management

- No weight reduction
- Moderate exercise; not vigorous
- Rest periods
- Known salt sensitive HTN: low sodium diet
- No ETOH
- No cigarettes
Mild - Moderate CHTN: Management

- Favorable outcome in most cases
- ↑ risk superimposed preeclampsia
- Complications more likely with
  - Pre-existing renal disease
  - Diabetes mellitus
  - Collagen vascular disease
- History of HTN in previous pregnancy

Important predictor!
Severe CHTN: Goals of Therapy

Decrease risk of:
- Cerebral hemorrhage
- Cardiac failure
- Myocardial infarction
- Perinatal morbidity and mortality

No decreased risk for:
- Superimposed preeclampsia
Severe CHTN: Outpatient Management

- Home BP monitoring
- Bedrest
- Hospitalize as needed
- Serial lab evaluation
- Serial ultrasound: fetal growth
- Antepartum testing: Start early
  - NST, BPP, AFI
  - Doppler
Severe CHTN: Inpatient Management

- Hospitalize for:
  - Worsening BP
  - Fetal compromise
- Bedrest
- Control HTN with pharmacologic agents
- Rule out superimposed preeclampsia
- Evaluate renal and cardiac function
- Intensify fetal surveillance, Betamethasone
- Severe, uncontrolled HTN: delivery
Pregnancy, HTN and Renal Disease

Risk: pre-pregnancy creatinine and HTN

- Mild disease: creatinine < 1.4 mg/dl
  - Fetal survival $\geq$ 95%, LBW 25%
  - Generally no worsening of disease
  - HTN develops in 25%
- Moderate: creatinine 1.4-2.0, or Severe: > 2.0
  - HTN develops in > 50%
  - Renal disease may accelerate
  - creatinine, BW
  - Fetal survival lower
  - Caution using MgSO$_4$
Pregnancy, HTN and Renal Disease

- **Dialysis:**
  - Significant maternal morbidity
  - Prior to conception: Fetal survival 40-50%
  - Begun post-conception: Fetal survival 74-80%
  - LBW and PTD the rule

- **Renal Transplant:**
  - Wait 1.5-2 yrs after successful transplant
  - Only if creatinine stable, < 2.0 mg/dl
  - HTN absent or mild; manage aggressively
  - Medications to maintenance levels
Severe CHTN: Prognosis

- 50% develop superimposed preeclampsia
- 25% perinatal mortality
- High neonatal morbidity
- Previous poor outcome: 60% loss in current pregnancy
- Compromised renal function: may deteriorate more
- 10% abruptio placenta
Chicago Lying-in Hospital
<table>
<thead>
<tr>
<th></th>
<th>Normal (n = 11)</th>
<th>Mild preeclampsia (n = 11)</th>
<th>Severe preeclampsia (n = 10)</th>
<th>Preterm (n = 6)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maternal age (yrs)</td>
<td>34.5 ± 0.9</td>
<td>32.4 ± 1.6</td>
<td>30.2 ± 1.3</td>
<td>33.3 ± 1.5</td>
</tr>
<tr>
<td>Gestational age (wks)</td>
<td>38.8 ± 0.2</td>
<td>34.0 ± 1.0</td>
<td>31.2 ± 0.9</td>
<td>29.9 ± 1.7</td>
</tr>
<tr>
<td>Primiparous (%)</td>
<td>17%</td>
<td>73%</td>
<td>70%</td>
<td>80%</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>&lt;140</td>
<td>151 ± 4.3</td>
<td>170 ± 5.6</td>
<td>&lt;140</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>&lt;90</td>
<td>102 ± 2.6</td>
<td>103 ± 3.8</td>
<td>&lt;90</td>
</tr>
<tr>
<td>Proteinuria (g protein/g creatinine)</td>
<td>&lt;0.3</td>
<td>1.1 ± 0.2</td>
<td>7.0 ± 1.8</td>
<td>&lt;0.3</td>
</tr>
<tr>
<td>Uric acid (mg/dl)</td>
<td>NA</td>
<td>6.4 ± 0.3</td>
<td>7.0 ± 0.2</td>
<td>NA</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>35.4 ± 0.6</td>
<td>34.6 ± 0.9</td>
<td>34.9 ± 1.5</td>
<td>34.4 ± 1.4</td>
</tr>
<tr>
<td>Platelet count</td>
<td>215 ± 18</td>
<td>214 ± 34</td>
<td>204 ± 27</td>
<td>220 ± 16</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>0.6 ± 0.04</td>
<td>0.6 ± 0.03</td>
<td>0.5 ± 0.03</td>
<td>0.5</td>
</tr>
</tbody>
</table>

Values shown are means ± SEM. Of the six patients in the preterm group, four had preterm labor, one had intrauterine growth retardation, and one had placenta previa. NA, not available.