Pregnancy Induced Hypertension

A multisystem disease which occurs in 6-8% of all pregnancies and is characterized by generalized endothelial damage and dysfunction, vasospasm and platelet aggregation; disturbed endothelial control of vascular tone causes hypertension, increased vascular permeability resulting in edema and proteinuria and abnormal endothelial expression of procoagulants leading to coagulopathy; these changes ultimately can cause ischemia of end organs, including the brain, liver, kidney, and placenta

ANESTHETIC CONSIDERATIONS:

- Considerations of pregnancy
- Multisystem disease
  - Potentially difficult airway d/t edema and aspiration risk
  - CNS → cerebral edema, seizures and intracranial hemorrhage
  - CVS → increased SVR, hypertension with potential for hypertensive crisis, relative volume depletion, decreased colloid osmotic pressure, increased interstitial volume leading to edema including pulmonary edema, LV dysfunction, CHF, hemodynamic instability and shock
  - Renal dysfunction, proteinuria, oliguria, ATN
  - Thrombocytopenia, coagulopathy, infrequently DIC, HELLP (Hemolysis, Elevated Liver enzymes, Low Platelets)
  - Drugs – exaggerated pressor response, sensitivity to non-depolarizing NMBAs
  - Decreased uteroplacental perfusion, IUGR, abruptio placentae, premature labor and delivery
- Considerations of antihypertensive / anticonvulsant therapy
- Increased risk of premature delivery and may require resuscitation of premature infant

ANESTHETIC GOALS:

- Control maternal hemodynamics, prevent a hypertensive crisis and its possible sequelae
- Optimize end organ perfusion and oxygen delivery, including that of the uteroplacental unit and fetus

HISTORY

- Always consider the broader differential:
  - Hypoxemia / hypercarbia
  - Preeclampsia / eclampsia
  - Chronic HTN
  - Pain
  - Drugs
    - Cocaine intoxication
    - Opioid / alcohol withdrawal
  - Endocrine
    - Thyrotoxicosis
    - Pheochromocytoma
    - Carcinoid
    - Serotonergic syndrome
    - NMS
- Gestational HTN
  - Onset of HTN without proteinuria or edema developing in latter part of pregnancy/immediate post-partum
  - Usually mild
- Preeclampsia = new onset of hypertension (SBP ≥ 140 or DBP ≥ 90) **and** proteinuria (0.3 g or more in 24 hr specimen) +/- generalized edema after 20 weeks gestation in a previously normotensive woman
- Severe Preeclampsia = new onset of proteinuria and HTN with at least one of following:
  - Severe BP elevation: SBP ≥ 160 mmHg or DBP ≥ 110 mmHg on two occasions at least 6 hrs apart
  - Proteinuria: > 5 g in 24 hrs or 3+ or more on two random samples 4 hrs apart
  - Symptoms of CNS dysfunction: blurred vision, scotoma, altered mental status, severe headache
  - CVA
  - Pulmonary edema or cyanosis
  - Symptoms of liver capsule distension: RUQ or epigastric pain, N/V
  - Hepatocellular injury: serum transaminase levels at least 2x normal
  - Thrombocytopenia: platelets < 100,000
  - Oliguria < 500 mL in 24 hrs
  - IUGR
- Eclampsia = preeclampsia with seizures
- **HELLP syndrome =** severe form of pre-eclampsia
  - Diagnosis criteria (some controversy):
    - Microangiopathic hemolytic anemia on blood smear (schistocytes or helmet cells)
      - May also see elevated indirect bilirubin, low serum haptoglobin
    - Platelet count < 100,000 cells/mcL
    - Serum LDH > 600 IU/L or total bilirubin > 1.2 mg/dL
    - Serum AST > 70 IU/L
    - Partial HELLP = women who do not meet all of the above
- **CNS** – headache (may signal impending seizure), blurred vision, seizures, anxiety
- **RESP** – dyspnea, chest discomfort, "hoarse" voice (airway edema)
- **CVS** – edema, dyspnea
- **GI** – epigastric pain, nausea / vomiting
- **RENAL** – weight gain
- **HEME** – easy bruising

PHYSICAL

- **GENERAL** – Vitals, including oxygen saturation
- **CNS** – retinal edema, CNS exam, hyperreflexia (seizures, ICH, cerebral edema)
- **HEENT** – careful airway exam (edema)
- **RESP** – cyanosis, rales / rhonchi (evidence pulmonary edema)
- **CVS** – volume status, edema, JVD, right / left sided heart failure (systemic vasoconstriction, ↓ intravascular volume, CHF/LV dysfunction)
- **HEME** – petechiae, oozing around IV puncture sites (hypercoagulability, thrombocytopenia, PLT dysfunction, DIC, HELLP)
- **GI** – enlarged liver edge (hepatic subcapsular edema)
- **RENAL** – generalized edema (↑ capillary permeability)
- **OB** – FHR – lack of variability or bradycardia (↓ placental perfusion)

INVESTIGATIONS

- **Labs**
  - CBC, peripheral blood smear, G/S
  - Lytes, BUN, creatinine
  - INR, PTT, fibrinogen (esp. if ALT or AST > 2 x normal or platelets < 100,000)
  - Liver enzymes, LDH, albumin
  - Uric acid
  - U/A protein, 24 h urine collection for protein and creatinine clearance
- **Imaging**
  - CXR, ECG, Echo, CT head as dictated by history / physical
- **Special**
  - Assessment of fetal wellbeing, including a non-stress test, amniotic fluid volume determination, estimation of fetal growth, and umbilical artery Doppler velocimetry, if possible

OPTIMIZATION

- (Delivery = treatment)
- Antihypertensive treatment to reduce risk of CVA/ICH in severe PIH & anti-seizure prophylaxis:
  - Magnesium 6 g IV bolus over 15 minutes, then 1-2 g/h by infusion
    - Goal 2.0 – 3.5 mEq/L
    - Do not use magnesium in patients with myasthenia gravis as this can precipitate myasthenic crisis
    - Toxicity – N/V, somnolence, hyporeflexia then weakness and apnea; > 10mEq/L (arrest)
  - Labetalol 5-10 mg IV prn
  - Hydralazine 5-10 mg IV q10min
  - NTG 50-100 mcg IVP or 10mcg/min and titrated to response
  - Sodium nitroprusside 0.25mcg/kg/min titrated to response
  - Nifedipine
- Preoperatively:
  - Aspiration prophylaxis: Sodium citrate 30 mL + Ranitidine + Metoclopramide
- Optimize maternal perfusion, replete intravascular volume
  - Perioperative judicious fluid challenge with plain balanced electrolyte solutions (+/- albumin)
  - If no improvement, CVP / PAC may identify patients with low CO
- Steroids for fetal lung maturation as per neonatologist
- Obstetricians balance timing of delivery – delayed delivery results in better fetal outcomes but runs the risk of increased maternal complications
  - If there is time to optimize, ensure that the delivery will happen in a hospital equipped to care for both an ill mother and (likely) premature neonate

ANESTHETIC OPTIONS

- Vaginal Delivery:
  - Non-pharmacologic methods
  - IV opioids
  - Pudendal nerve block
  - Epidural (preferred technique)
    - Advantage over other techniques in PIH = BP attenuation
    - May want to avoid using epi in infusate
- Cesarean section
  - Epidural
  - CSE
  - Spinal – controversial but becoming more widely accepted
    - Quoted disadvantage: rapid onset of sympathetic blockade may result in profound hypotension
      - Little evidence for this
    - Advantage over CSE / Epidural: possibly less risk of hematoma with single-shot technique vs. placement of a catheter?
  - GA – indicated if regional anesthesia contraindicated, poor maternal condition with cardiopulmonary deterioration, or in situation of severe fetal distress and anticipated easy airway
- Regional Anesthesia
  - Ensure no contraindications: coagulopathy, thrombocytopenia, evidence of ↑ ICP on neurologic examination
  - Ensure volume resuscitation and be prepared to treat post block hypotension
- General anesthesia
  - Single biggest risk is maternal hypertension → ICH

ANESTHETIC SETUP

- **Drugs**
  - Drugs to rapidly decrease or increase BP
- **Equipment**
  - Standard CAS monitors plus 5-lead ECG
  - Be prepared for difficult airway – have variety of sizes of styletted ETTs available, difficult airway cart and a backup plan for airway management
  - Consider arterial line monitoring
  - Consider CVP / PA catheter if pulmonary edema, renal failure, or severe cardiac disease present
  - Foley catheter to urometer
  - Nerve stimulator
  - Fetal heart monitoring

MANAGEMENT OF ANESTHESIA

- **Induction**
  - Consider AFOI if suspected difficult airway on exam
    - Edema may obscure normal structures, making RSI difficult or impossible
    - Mask ventilation may be difficult if face, lips, tongue also swollen
  - RSI indicated
  - If GA, need to prevent hypertensive response to intubation
    - Fentanyl: tell pediatrician
    - Lidocaine
    - +/- Labetalol up to 1 mg/kg IV
    - Consider NTG or nitroprusside for induction
    - STP 4-5 mg/kg or propofol 2 mg/kg
    - Succinylcholine 1.5 mg/kg
- **Maintenance**
  - Pregnancy lowers MAC by 30% for all inhalational agents
  - 0.5 – 1 MAC volatile +/- Nitrous
  - All inhalational anesthetics cause uterine relaxation
    - May require greater than normal oxytocin infusion to contract uterus after delivery
  - Beware of hemorrhage at delivery which may lead to dramatic hypotension
- **Emergence**
  - Extubation - ensure adequate reversal of NMBAs – remember $MgSO_4$ potentiates relaxants
    - Half of maternal aspirations occur during emergence

DISPOSITION & MONITORING

- Risk for developing pulmonary edema due to previous intravascular hydration
  - Diuresis after delivery
- Still at risk for severe complications and may need intensive care for several days and ongoing antihypertensive therapy and $MgSO_4$

COMPLICATIONS

- Preeclampsia with fetal distress
  - Approach would depend on airway
    - Difficult Airway
      - Attempt regional anesthesia
      - Simultaneous topicalization of airway by assistant so can rapidly switch to awake intubation if failed regional
    - Easy Airway
      - GA

PATHOPHYSIOLOGY

- 5-6% nulliparous develop preeclampsia, 2-3% develop severe preeclampsia
- Preeclampsia and eclampsia account for 20% of maternal and perinatal deaths
- **Etiology**
  - Exact etiology of PIH not well understood – thought to be 3 main factors
    - Vasospasm caused by sensitivity of vascular smooth muscle to catecholeamines
    - Ag-Ab reactions b/t fetal and maternal tissue causing placental vasculitis
    - Imbalance of vasoactive PGs (TXA and prostacyclin) causing vasoconstriction and aggregation of platelets
- **Treatment**
  - Cure = delivery
  - Supportive treatment = antihypertensives, seizure prophylaxis and support of maternal perfusion, intravascular rehydration
  - Analgesia (esp. epidural analgesia) for labor reduces catecholamine response to pain and increases placental perfusion
- **Definitions**
  - **PIH** = BP > 140/90 after 20 weeks gestation in previously normotensive patient; BP must show this elevation at least twice > 6h apart and not associated with uterine contraction
  - **Preeclampsia** = above BP rise, plus evidence of other organ system involvement, e.g. proteinuria, nondependent edema, increased LFTs, decreased PLTs, CNS dysfunction
  - **Severe preeclampsia** = either BP > 160/110 or proteinuria > 5 g/24h, or evidence of consumptive coagulopathy (DIC), or liver swelling / failure (epigastric or RUQ pain), or pulmonary edema (desaturation), or evidence of CNS edema (severe headache)
  - **Eclampsia** = one or more generalized convulsions and / or coma in the setting of preeclampsia (and not due to other neurological conditions)
    - Eclampsia used to be thought of as the end result of preeclampsia, but now seizures are considered to be only one of several manifestation of severe preeclampsia

        - Can occur up to one week postpartum
    - **Chronic hypertension** = SBP 140 mmHg, DBP 90 mmHg, or both, that precedes pregnancy, is present before the 20th week of pregnancy, or persists longer than 12 weeks postpartum
    - **Preeclampsia superimposed on chronic hypertension** = woman with chronic hypertension who develops new onset proteinuria after 20 weeks of gestation
        - Women with chronic hypertension and preexisting proteinuria (before 20 weeks) are considered preeclamptic if there is an exacerbation of blood pressure to the severe range (systolic 160 mmHg or diastolic 110 mmHg) in the last half of pregnancy, especially if accompanied by symptoms or a sudden increase in proteinuria
    - **Gestational hypertension** = hypertension (usually mild) without proteinuria (or other signs of preeclampsia) developing in the latter part of pregnancy
        - If it resolves by 12 weeks postpartum, then in retrospect, it is classified as **transient hypertension of pregnancy**
            - If the hypertension persists beyond 12 weeks postpartum, then the diagnosis is chronic hypertension that was masked in early pregnancy by the physiologic decrease in blood pressure
    - **HELLP** develops in 1/1000 pregnancies and in 10-20% of cases of severe preeclampsia / eclampsia
        - Most are diagnosed between 28-36 weeks gestation, but can occur prior to this and also postpartum (30% of the time – usually within 48 hrs, but can be up to 7 days postpartum)
        - Not necessarily associated with hypertension and proteinuria

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