HEART DISEASE IN PREGNANCY PROGRAM (HDPP)

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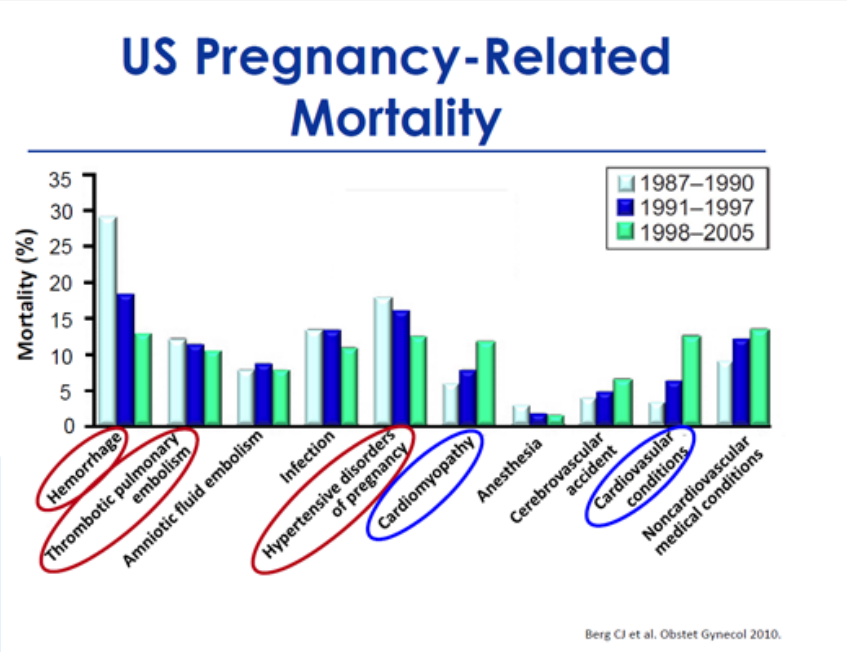
Background

- Although cardiac disease complicates a small number of pregnancies, it has become a leading cause of US maternal mortality
- Congenital
  - Reaching reproductive age and desiring fertility
- Acquired
  - Population as a whole becomes more unhealthy with a large burden of dyslipidemia, obesity and metabolic syndrome



Heart Disease in Pregnancy Program: Why is it needed?

- Complex patients
- Multiple physicians and care teams involved
  - Difficulty coordinating care
  - Input needed from specialists who rarely care for pregnant patients
- Chaos at time of delivery with divergent plans from OB, cardiology, anesthesia and ICU teams

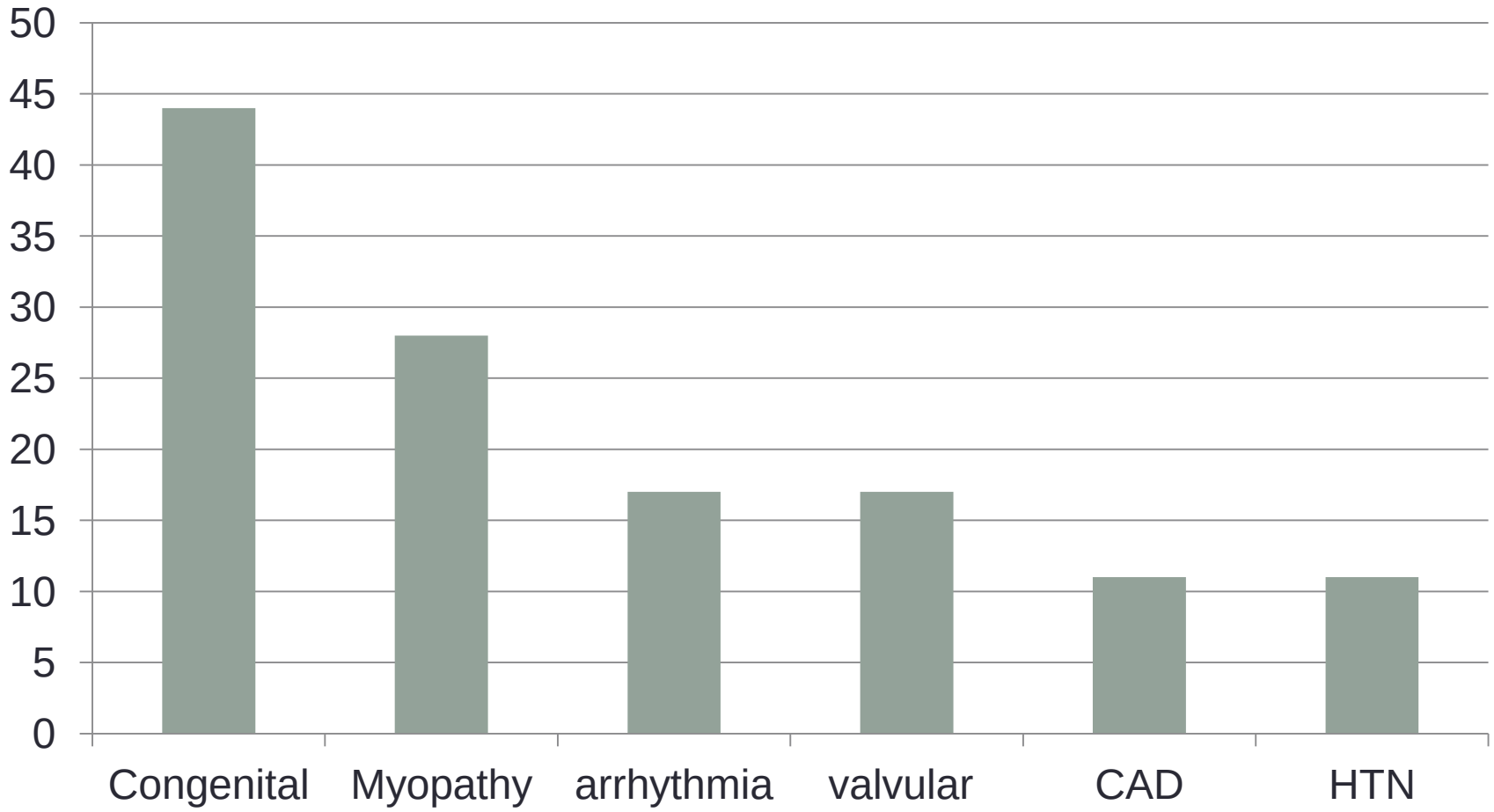
Heart Disease in Pregnancy Program

- Since starting our program:
  - 100 patients enrolled in 2015 (including consults and preconception)
  - 49 deliveries
  - 7 ICU deliveries, all others on L&D with telemetry
    - Severe AS
    - Marfan's with aortic dissection and concominant C-section and aortic root replacement
    - Hypertrophic CM
    - Turner's syndrome
  - No unplanned ICU transfers
  - Goal: improved outcomes for moms and babies

Cardiac Disease in Pregnancy Program

- Comprehensive, multidisciplinary maternal cardiac program
  - Maternal-Fetal Medicine
  - Cardiology
  - Intensivists
  - Anesthesiology
  - Genetics
- Biweekly office visits available
- Multidisciplinary setting monthly
  - Discuss current status, timing of delivery, mode of delivery and location

Represented conditions



Physiology of Pregnancy

Parameter	Percentage of change	
Cardiac output	40–50%	Increase
Stroke volume	30%	Increase
Heart rate	15–25%	Increase
Intravascular volume	45%	Increase
Systemic vascular resistance	20%	Decrease
Systolic BP		Minimal
Diastolic BP	20%	Decrease at mid-pregnancy Pre-pregnant values at term
CVP		Unchanged
O_2 consumption	30–40%	Increase

Maternal cardiac changes with pregnancy

- Increased cardiac output 30-50%
- Sodium and water retention with blood volume expansion
- Physiologic anemia
  - Increased red cell volume with increased plasma volume, peaking at 34 weeks
  - Blood volume is 50% above nonpregnant values at term
- Increased plasma renin activity
- Reduced BNP levels
- Decreased blood viscosity
  - Lower cardiac work with reduced resistance to flow
  - Enhances placental perfusion

Maternal cardiac changes with pregnancy

- Increased cardiac output
  - Blood flow largely directed to placenta, maternal kidneys and maternal skin
  - Lower maternal serum creatinine with enhanced glomerular filtration rate mediated by serum relaxin hormone, increased by hCG levels from placenta

Maternal cardiac changes with pregnancy: hypercoagulable state

- Reduced prothrombin and partial thromboplastin times by 20%
- Hypercoagulable state of pregnancy
  - Lower protein S levels
  - Increased resistance to activated protein C
  - Increased Factors I, II. V, VII, VIII, X and XII
  - Increased activity of fibrinolytic inhibitors PAI-! And PAI-2
- Increased risk of venous thromboembolism 3-4x above non-pregnant women

Cases

- Patients are typically followed frequently in clinic
- High rate of admission
- Many time-sensitive labs

Sickle-Beta Thalessemia

- 31 yo F with sickle cell trait/beta thalessemia
  - Follows with hematology at NKC
- Obtains exchange transfusion every 4 weeks while pregnant
- Potential complications:
  - Right sided heart failure, pulmonary hypertension
  - Maternal hypertension, preeclampsia
  - Stroke and thromboembolism
  - Sickle nephropathy
  - Sickle cell crisis during pregnancy
  - Sickle cell disease in offspring

Sickle cell anemia in pregnancy

- Baseline labs:
  - Hemoglobin electropheresis
  - Red cell phenotyping and screening for alloimmunization – repeated in second trimester and when admitted for delivery
  - Screening for sickle nephropathy: BMP, UA, 24-hour protein excretion
  - Hemoglobin/hematocrit, ferritin levels
  - Urine culture
  - Hepatitis B and C screening

Recent admission

- 27 weeks gestation: sickle cell crisis, abdominal and chest pain
  - Transfused
    - anti-C/anti-E/anti-K antibodies present
  - History of acute chest syndrome
    - New radiodensity on CXR with respiratory symptoms and hypoxia
    - Difficult to distinguish from pneumonia
    - Procalcitonin levels normal
- Echo:
  - Normal left ventricular systolic function, with an estimated ejection fraction of 65%.
  - No significant valvular abnormalities.
  - PA pressure 25 mmHg

Labs

- Hemoglobin electrophoresis:
  - Hgb A 63.3
  - Hbg F 1.1
  - Hgb A2 4.3
  - Hgb S 31.3
- Hemoglobin 8.8, hematocrit 26
- Creatinine 0.5, eGFR >130
- UA with trace protein, otherwise negative

Pregnancy with a mechanical valve

- 26 yo F G3P1 with history of mitral valve endocarditis
- History of embolic stroke and drug abuse
- Now s/p mechanical mitral valve in 2014 for MSSA endocarditis
- Recently released from prison, hadn’t established medical care
- Multiple admissions with labile INRs
- HIV negative, hep B and C negative
- UDS negative x 2 years

Recent admission

- 4 admissions during this pregnancy with subtherapeutic INR 1.1 requiring heparin IV
- Most recent at17 weeks gestation, presented with nose bleeds and coughing up blood
  - INR 6
- No admissions in last 2 months
- Weekly INR
- Planned admission at 36 weeks for heparin IV until delivery

Anticoagulation in pregnancy

- Warfarin
  - Pregnancy category X: associated with high risk of miscarriage and embryopathy
  - Best anticoagulation for mechanical valves
  - Fetal brain hemorrhage with vaginal delivery
- Heparin
  - Low risk of fetal complications
  - Higher risk of valve thrombosis
- Lovenox
  - Low risk of fetal complications
  - Higher risk of valve thrombosis

Warfarin embryopathy

Maternal lupus

- 21 yo F with lupus
- Pulmonary hypertension
- Marantic (sterile) endocarditis 2015
- Presented for planned mitral valve replacement
  - UPT negative on admission for valve surgery
- Missed her period following surgery, thought to be secondary to stress…

Echo

Pre-MVR

- normal ejection fraction of 65%
- Diffusely thickened mitral leaflets consistent with marantic endocarditis. Compared to the previous study dated 3/12/2015, severe mitral regurgitation persists.
- PA pressure 43 mmHg

3 month Post-MVR

- Normal ejection fraction of 65%.
- Bioprosthetic mitral valve replacement functioning normally, mean gradient = 8 mmHg, without regurgitation.
- PA pressure 24 mmHg

Maternal lupus

- Potential pregnancy complications:
  - Worsening renal failure
  - Fetal complete heart block due to maternal Smith antibodies
- Recommended labs:
  - aPLs: lupus anticoagulant (LA), immunoglobulin G (IgG) and IgM anticardiolipin (aCL) antibodies, and IgG and IgM anti-beta2-glycoprotein (GP) I antibodies
  - Anti-Ro/SSA and anti-La/SSB antibodies
  - Renal function (creatinine, urinalysis with urine sediment, spot urine protein/creatinine ratio)
  - Complete blood count (CBC)
  - Liver function tests
  - Anti-double-stranded deoxyribonucleic acid (dsDNA) antibodies
  - Complement (CH50, or C3 and C4)
  - Uric acid

Maternal Lupus

- DNA antibody >300
- RO/SSA antibody negative
- LA/SSB antibody negative
- Complement levels normal
- UA normal
- CBC, CMP normal

Maternal lupus

- Now 3rd trimester
- Required IV steroids and prednisone taper for nephritis and lupus flare, resolved
- Persistent sternal wound with possible infection
- Presented with acute dyspnea and heart rate 130s at rest in clinic

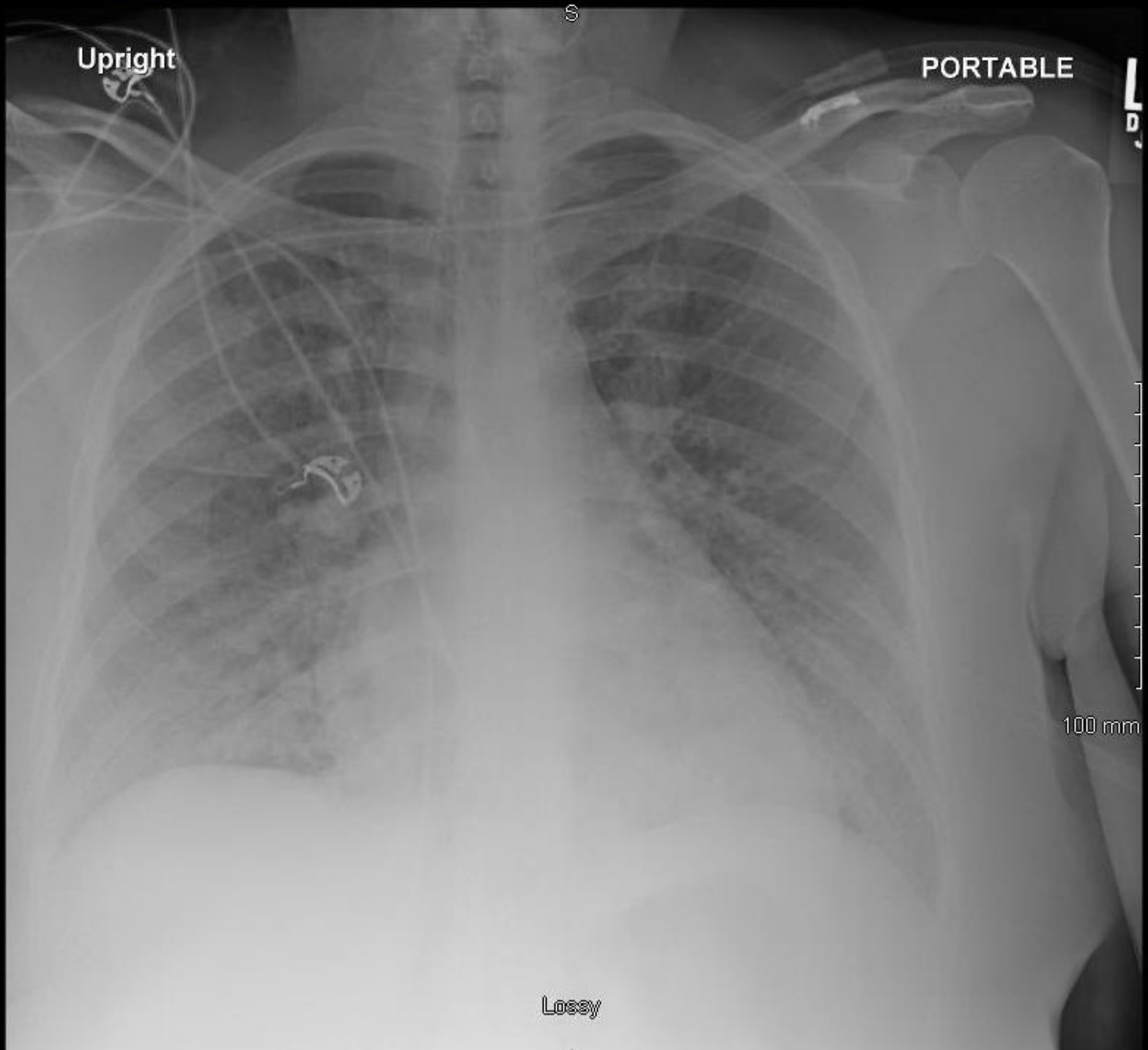
Echo

Peripartum CM

- 34 yo F, G5
- Presented 8 weeks post-partum with worsening dyspnea
- Pregnancy uncomplicated
- Delivered at home
  - EMS called but delivered before their arrival
- Post partum hemorrhage requiring IV iron infusion
- Readmitted with worsening dyspnea

BP - labs

- Troponin 0.05
- NT pro BNP 1570
- WBC 7.4, Hbg 11.2, Hct 37, plt 362, MCV 80
  - Previous CBC at delivery: WBC 11, Hbg 5.7, Hct 19, plt 302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Upright
PORTABLE
L
D
100 mm
Lossy

PPCM

- Echo
  - Severely reduced left ventricular systolic function, with a calculated ejection fraction of 26%.
  - Mild left ventricular dilatation. Global hypokinesis.
  - Mild left atrial dilatation. Other chamber dimensions normal.
  - Thickened mitral valve with mild to moderate regurgitation.
  - Estimated PA pressure = 26 mmHg.

Peripartum cardiomyopathy

- Definition:
  - Heart failure occurring in last month of pregnancy or first 5 months post-partum in absence of other causes of heart failure
  - LVEF <45%
- Incidence in US 1:2000-4000 live births
- Endemic in Haiti and parts of Africa
- Potential risk of mortality up to 15%

Risk factors

- Maternal age >30 years
- Multiple gestation
- African American race
- Premature labor
- Hypertension, Pre-existing HTN, Gestational HTN, Preeclampsia/eclampsia
- Anemia
- Tobacco abuse
- Thyroid dysfunction

- Risk factors for poor recovery of cardiac ejection fraction
  - Elevated troponin at presentation
  - Elevated BNP at presentation
  - LVEF <35% with dilated left ventricle

PPCM: etiology

- Multiple disease processes presenting in peripartum period
  - Related to abnormal prolactin cleavage?
  - Autoimmune
    - Autoantibodies against adenosine nucleotide translocator, branched-chain alpha-keto acid dehydrogenase, myosin heavy chain correlate with LV dimension and EF
  - Myocarditis
  - Genetic/familial cardiomyopathy
  - Viral/post viral
    - Parvovirus B 19, HSV 6, EBV, CMV, chlamydia found on biopsy series

PPCM

- Outcomes
  - 50% recover entirely
  - 35% partial recovery
  - 15% progressive heart failure requiring transplant or LVAD

Cardiac Disease in Pregnancy Program: Research Goals

- Build a comprehensive, prospective database
  - Congenital and acquired disease
  - Obstetrical, maternal and neonatal outcomes
    1. Validity of CAR-PREG in a racial diverse population
    2. Evaluate for additional risk factors during gestation
    3. Stratify by congenital and acquired disease
    4. Assess fetal growth and Doppler parameters that may be predictive of adverse outcome
    5. Assess placental pathology in women with cardiac disease and a matched population
- **Help guide clinicians across the country for the care of women with heart disease in pregnancy**

Thank you for your consideration