High Risk Pregnancy

Lecture 10
Maternal Mortality: 10/100,000 pregnant women.
Leading causes: hemorrhage, hypertension, infection, preeclampsia.

High Risk Pregnancy:
A. Maternal Age < 15 & > 35.

B. Parity Factors - 5 or more - great risk.
   [PP hemorrhage] New preg. within 3 mos.

C. Medical-Surgical Hx - hx of previous uterine surgery &/or uterine rupture, DM, cardiac dis, lupus, HTN, PIH, HELLP, DIC etc.
Gestational Conditions

Hyperemesis Gravidarum

- Persistent vomiting past first trimester or excessive vomiting @ anytime.
- Severe; usually 1st pregnancy.
- Rate occurrence ^ with twins, triplets, etc.
- 7 out of 1,000. Electrolyte imbalance results.
- Possible causes: ^ levels of hCG, ^ serum amylase, decrease gastric motility.
Hyperemesis cont.

S/S:
- dry mouth, thirst, substantial wt. loss, signs of starvation, dehydration, decreased skin turgor; fruity breath - acidosis [metabolizing fat].

Management: admit to hospital for IV hydration.
- NPO then clears > full fluids> sm. solids.
- Reglan or Zofran IVPB q 6 hrs.
- Hosp. for 24-48 hrs. Add Multi Vit.& K to IVF to correct electrolyte imbalance.

Use ½ NS
Hydatidaform mole - "Molar preg."

- Degenerative disorder of trophoblast (placenta)
- Villi degenerate & cells fill with fluid
- Form clusters of vesicles; similar to “grapes”
- Overgrowth of chorionic villi; fetus does not develop.
- Partial or complete. Complete - no fetus; Partial - dev. begins then stops.
- Multiparous/older women.
- Pathophysiology: unknown; theories: chromosomonal abn., hormonal imbalances; protein/folic acid deficiencies.
Hydatidaform Mole cont.

- **S/S:** from bright red spotting to bright red hemorrhage.
- Tissue resembles grape clusters
- Uterus appears larger than expected for gest. age.
- **Management:** D&C; go to hosp. STAT; bring any passed tissue.

“Gestational trophoblastic disease” - benign hydatidaform mole & gestational trophoblastic tumors aka & chorio carcinoma.
Hemorrhagic Disorders

Placenta previa:

- Implantation near or over cervix.
- ~ 1/2 of all preg. start as previa then placenta shifts to higher position
- By 35 wks. not likely to shift

Varying Degrees:

- **Total**: internal cervical os completely covered by placenta.
- **Partial**: os partially covered by placenta.
- **Marginal**: edge @ margin of internal os.
- **Low-lying**: region of internal os near placenta.
PLACENTA PREVIA CONT…..

- **Cause:** Unknown
- **Risk factors:** multiparity, AMA, multiple gestations, previous uterine surgery
- **Manifestations:** Painless, bright red bleeding > 20th week; episodic, starts without warning, stops & starts again.
- **Prognosis:** depends on amt. bleeding & gest.age

  - **Management:** Monitor FH, maternal VS, IVF; O2; assess I&O; amt. of bleeding, CBC, & complete BR. Type & cross for poss. transfusion.
  - Ultrasound to confirm
  - No pelvic exams/no vag. del.- May lead to hemorrhage
**Abruptio placenta:**

- Separation of placenta from uterine wall > 20th wk gestation (during pregnancy)
- “Placental Abruption”- hemorrhage results; Severity depends on degree of separation.
- Common in multips, AMA.
- Fetal prognosis depends on blood lost & gest. age.
- **Grade 0:** Separation not apparent until placenta examined > delivery.
- **Grade 1:** minimal - causes vaginal bleeding & alterations in maternal VS.
- **Grade 2:** moderate - + signs of fetal distress. Uterus tense & painful when palpated.
- **Grade 3:** extreme (total) separation. Maternal shock/fetal death if immediate intervention not done.
Abruption cont.

- **Cause:** Unknown; risk factors: smoking, short umbilical cord, adv. mat. age, HTN, PIH, cocaine use, trauma to or near abdomen.
- **Manifestations:** tenderness to severe constant pain; mild to moderate bleeding depending on degree of sep..
- Total separation: tearing, knifelike sensation.
- **Management:** Assess amt. & control blood loss
  - Assess FH & mat. VS, CBC, O2, IVF [RL-volume expander], type & cross.
  - Neonate may be hypoxic, in hypovolemic shock d/t bl. loss. Prepare for emergency C/S.
Comparison: Abruption vs. Placenta Previa

**Abruptio Placenta**
- Pathology: Sudden separation of normally implanted placenta.
- Vaginal Bleeding: may not be obvious; concealed behind placenta. If visible, will be dark. Bleeding into uterine cavity.
- Pain: Sharp, stabbing pain in abd.
- Uterus: hard boardlike abd.

**Placenta Previa**
- Pathology: Abnormal implantation of placenta; lower in uterine cavity.
- Vaginal Bleeding: Abrupt onset; bright red.
- Completely painless.
- Uterus: Soft, unless uterine contraction is present.
Abnormal Amniotic Fluid Levels:
- AMF made up of fetal urine & fluid that is transported thru placenta from mat.cir.sys.
- Fetus swallows AMF & excretes urine.
- Waste excreted by maternal kidneys.
- CNS or GI abnormalities inhibits uptake of AMF.

**Polyhydramnios:** > 2,000 ml amniotic fluid.
Need ultrasound to make dx.
AFI \( \geq 24 \) = polyhydramnios.
AFI of 8-23 cm. normal.

Visual inspection may reveal rapidly enlarging uterus.
Risk Factors

- Fetal abnormalities: neonatal macrosomia, fetal or neonatal hydrops [swelling], ascites, pleural or pericardial effusions.
- Skeletal malformations: congenital hip dislocation, clubfoot, & limb reduction defect.
- Abnormal fetal movement suggestive of neurologic abnormalities (CNS)
- Obstruction of GI tract; prevents normal ingestion of amniotic fluid.
- Rh iso immunization d/t mixing of maternal/fetal blood
- Maternal hx DM.
- Assoc. w.spina bifida; anencephaly, hydrocephaly.
- Fetal death may result from severe poly.
- Not as common as oligohydramnios
Polyhydramnios cont.

Management: Monitor wt. gain. Remove excess amniotic fluid q 1-2 wks. thru amniocentesis. Replaced quickly. Most women with mild poly. deliver healthy infants.

Oligohydramnios: amniotic fluid < 1,000ml. Could be highly concentrated. Interferes w. normal fetal dev. Reduces cushioning effect around fetus.

- Causes: Failure of fetal kidney development; urine excretion blocked, IUGR, post-term preg., preterm ROM, fetal anomalies. Poor placental function.
Manifestations:
- Facial & skeletal deformities: club foot.
- Fetal demise
- Pulmonary hypoplasia - improper dev. of alveoli;
- 2\textsuperscript{nd} trimester oligo: higher rate of congenital anomalies (50.7 \% vs. 22.1 \%) and lower survival rate (10.2 \% vs. 85.3 \%) than women with oligo. in 3\textsuperscript{rd} trimester.
- Prognosis: depends on severity of disease.

Management: Careful assessment of mother/fetus
- Do frequent ante-partum testing
- determine optimal time for delivery (early)
- Antibiotics/corticosteroids with PPROM
Ectopic pregnancy:
Causes:

- Scarring of fallopian tubes (Chlamydia/Gonorrhea).
- Implantation of ovum outside uterine cavity; usu. upper 1/3rd fallopian tube; rare on ovary, cervix, abdominal cavity.
- Leading cause of death from hemorrhage in preg.
- Reduces fertility
  - ~ 1 in 100 pregnancies
  - More common > infection of fallopian tubes or surgery to reverse TL.
- Previous ectopic
- multiple induced abortions
- diethylstilbestrol (DES) exposure
Ectopic

Symptoms:
- Colicky, cramping pain in lower abdomen on affected side
- Tubal rupture: sharp/steady pain before diffusing throughout pelvic region. Fetus expels into pelvic cavity.
- Heavy bleeding causes shoulder pain, rectal pressure
- N/V 25-50% pts. think it's morning sickness.
- Dizziness/weakness - If tube ruptures, weak pulse, clammy skin, fainting. Assess for signs of shock.

Treatment:
- Immediate surgery to remove/repair tube.
- If no rupture, Methotrexate - stops cellular division in fetus; causes cell death. Conceptus expelled with bleeding. Best <6 wks. when chance of rupture less.
Rh sensitization
Rh (D) Immune Globulin aka “Rhogam”

- Rh – mom: no Rh factor.
- If Rh - mom receives cells from Rh + fetus, mother responds to (+) AG by producing AB.
- **Treatment**: Anti-Rho (d) immune globulin @ 26-28 wks. (Rhogam) & @ delivery.
- Prevents antibody formation if mom is Rh -.
- Rapid RBC lysis leads to ▲ bilirubin levels.
- **1st exposure** [Rh+ blood] IgM, antibodies develop; too large to cross placenta.
- First Rh + fetus usually not harmed.
- **2nd exposure**, smaller IgG antibodies formed; can cross placenta & destroy fetal red blood cells.
Maternal-Fetal Blood Group Incompatibility:

- Mom is type O & baby is A, B or AB.
- Blood types A, B, & AB contain antigen not present in type O blood.
- (O) blood type have anti A/anti B antibodies.
- If exchange occurs, maternal antibodies attack fetal bl.cells, causing rapid lysis of RBC's.
- Leads to byproduct bilirubin.
- Results in “jaundice”.
- ABO less severe than Rh incompatibility b/c primary antibodies of ABO system are IgM which are too large to cross placenta.
- Bilirubin levels done & phototherapy if ^.
Hypertensive States of Pregnancy:

- **Global** cause of maternal/fetal morbidity & mortality. Responsible for ~ 76,000 deaths/year.

4 categories:

1. **“Chronic HTN”**: ^BP prior to pregnancy. Not associated with proteinuria or end-organ damage; continues > delivery.

2. **“Preeclampsia”**: ^BP during preg. > 20 wks. End-organ damage may occur. 50-70% cases.

3. **“Superimposed preeclampsia on pt. w. Chronic HTN”**: 15-30% cases.

4. **“Transient HTN”**: BP >140/90 without proteinuria or end-organ damage. Normotensive pt. may become hypertensive late in preg., during labor, or 24 hours postpartum. BP normal within 10 days postpartum.
Pre-eclampsia

Defined As:
- BP ≥ 140/90
- Systolic ↑ of 30mm Hg > pre-preg. levels
- Diastolic ↑ of 15mm Hg > pre preg. levels.
- Presents with HTN, proteinuria, edema of face, hands, ankles.
- Can occur anytime > 20th wk of pregnancy.
- Usually occurs closer to due date. Will not resolve until > birth. Can progress to HELLP syndrome
General Signs of PREECLAMPSIA

- Rapid weight gain; swelling of arms/face
- Headache; vision changes (blurred vision, seeing double, seeing spots)
- Dizziness/faintness/ringing in ears/confusion; seizures
- Abdominal pain, ↓ production of urine; nausea, vomiting, blood in vomit or urine
**Mild**: mild HTN; no end-organ damage; minimal proteinuria.

**Severe**:  
- Significant HTN; severe proteinuria (>5.0 g/d); end-organ damage d/t systemic vaso-constriction.  
- Headache; visual changes; confusion; abdominal pain; impaired liver function w.hyperbilirubinemia; oliguria; proteinuria; pulmonary edema; hemolytic anemia; thrombocytopenia; fetal growth retardation.  
- Eclampsia (seizures) may follow
Eclampsia:

- Seizures or coma d/t hypertensive encephalopathy; most serious complication.
- Affects ~ 0.2% preg; 1 in 1000 preg. terminated.
- Major cause of maternal death d/t intracranial hemorrhage. Maternal mortality rate is 8-36%.
- Deliver by C/S ASAP.
Risk factors:

- < age 20 or > 40
- Twins, triplets; primagravida
- Molar pregnancy
- Preexisting: HTN, Diabetes mellitus
- Renal or vascular disease
- Prior hx of preeclampsia/eclampsia

Frequency: 5% {all pregnancies in US}

Causes: Unknown.

Theories: maternal immune reaction that leads to systemic peripheral vascular spasm >> leads to endothelial cell damage >> vasoconstriction> ^BP.

- Affects multiple organs. Reduced bl.supply to kidneys, liver, placenta, brain. Can lead to placental abruption and fetal & maternal death.
**Management:** Usually only cure is delivery. Depends on symptoms.

**Mild preeclampsia:** Bedrest. Monitor @ home or hospital. Deliver close to EDC. Frequent BP’s, 24 hr.urine, liver enzymes, FHR, & ultrasounds.

**Severe preeclampsia:** Goal: prevent convulsions & control mat. BP. 

BP = 160/110, epigastric pain, 2-4+ proteinuria, ^ liver enzymes, thrombocytopenia [↓ 100,000].

* Magnesium sulfate [drug of choice]*
Magnesium Sulfate

- Bronchodilating effects; prevent seizures; lowers BP.
- Steroids to mom for fetal lung maturity if premature delivery imminent
- Infusion pump – IVPB into mainline of RL before/during labor & 24 hrs > delivery.
- Infuse slowly.
- Baseline VS & UO **before** therapy. RR @ least 16/min
- Mag SO4 removed **solely** by kidneys.
- Don’t use in pts. w. renal disease. Other drugs: Dilantin & Valium.
- Patellar reflex, place foley, RR, fetal status q hr.
Monitor mag. levels q 2hrs. Mag level 4-6 meq./liter therapeutic.

**Magnesium Toxicity** based on clinical signs:
- sharp drop in BP
- respiratory paralysis
- disappearance of patellar reflex.

STOP infusion, give O2 & calcium gluconate ASAP.
**HELLP Syndrome:** “Hemolysis, Elevated Liver Enzymes, Low Platelets” Can progress to DIC.

- Target organ is liver.
- **Vasospasms** cause vasoconstriction & lead to reduction in blood flow to uterus/other organs.
- Leads to ↑ BP, visual disturbances, low UO, ↓ HCT
- Anemia; Epigastric/RUQ pain & tenderness d/t liver swelling. Weakness, fatigue, jaundice.
- Hemolysis > destruction blood cells; + anemia.
- ^ liver enzymes > sign of liver damage.
- Low platelets - ^ peripheral vascular destruction.
- CBC, platelet count, PT, PTT, LFT’s, uric acid.
DIC: DISSEMINATED INTRAVASCULAR COAGULATION [acute disorder]


Results in decreased blood flow & ^ tissue damage
Always a secondary dx.

Causes: ^ tissue thromboplastin d/t vascular damage
Triggers: amniotic fluid embolism, eclampsia, abruption, pre-eclampsia, HELLP, trauma, gram – sepsis.
DIC cont.  Physical Assessment
Petechiae/ecchymotic areas on skin/mucous mem.
Hemorrhaging [occult or obvious] Nose, gums, IV site, IM inj.site, etc...

S/S shock; oligouria; ^HR; diaphoresis, seizures
Diagnostic tests: CBC, prolonged prothrombin time
LAB Findings: low plts, fibrinogen, other clotting factors.

**Intervention:** Treat underlying condition.
Infection, pre-eclampsia, abruption, bleeding, shock.
- IVF (RL); O2 @ 6-10 L / min.
- Foley to monitor UO [30-50 ml/hr]
- Monitor VS; Clotting factors replaced with PRBC’s, platelets & fresh frozen plasma.
- Heparin may be given to block coagulation cascade.

Complications: organ injury; maternal mortality.
**Incompetent Cervix:**

- Passive dilation of cervix without context.
- Usually in **early** pregnancy.
- hx miscarriages.
- Causes: congenitally short cervix; composition of cervical tissue; DES exposure.
- Dx: ultrasound

**Management:** Strict BR; trendelenberg position if + bulging membranes; hydration IVF, tocolysis.

**Cerclage** **best** if placed by 10-14 wks. – binds cervical os.

No intercourse; Remove @ 37 wks. 80-90% success
Diabetes

- ^ 41% from 1990 - 1999 in US; 4.9 to 6.9 respectively. Includes gest. DM.

- Contributing factor: ^ obesity
- Since 1991, obesity ^ 57% in US

- Perinatal mortality/morbidity 6x higher w. undx. pre-existing DM.
- Most common complication of pregnancy.
- Mexican, Puerto Rican, American Indian, Asian Indian, Hawaiian: higher rates than other Hispanic, white, & black. [CDC, 1998]
Gestational Diabetes AKA “GDM”

- Glucose intolerance beginning in pregnancy.
- GDM occurs > 20th wk. with no ^ incidence of anomalies.
- ~ 2% pts. have undx type II entering preg.
- Type 1 & 2 have ^ anomalies d/t organogenesis (1st trimester).
- ~ 4% of preg. affected. May/may not require insulin.

- Maternal Risks: HTN disorders, PTL, polyhydramnios, macrosomia (^ C/S rate).

- Infant Risks: Birth trauma, shoulder dystocia, hypoglycemia, hyperbilirubinemia, RDS, thrombocytopenia, hypocalcemia, fetal death.
Pathophysiology: GDM

- Pregnancy hormones estrogen, HPL, prolactin, cortisol, progesterone, blocks insulin receptors > 20 wks. pregnancy.

- Results in $^\uparrow$ circulating glucose;
- More insulin released to attempt to maintain glucose homeostasis. Pt. feels “hungry” d/t $^\uparrow$ insulin; vicious cycle of $^\uparrow$ appetite & wt.gain results.
Screening & Diagnosis of GDM

- screen ALL women @ 24-28 wks.
- HIGHER Risk pts. screened in 1\textsuperscript{st} trimester/1\textsuperscript{st} prenatal visit & @ 24-28 wks.

**Determining High Risk Clients:**
- Family hx DM; Previous hx GDM
- Marked obesity; Glycosuria
- Maternal Age > 30
- Hx infant > 4000g
- Member of high-risk racial/ethnic group
  Hispanic, Native American, South or East Asian, African American, Pacific Islander.

*If results negative, repeat @ 24-28 wks.*
Screening & Diagnosis cont.

1st Do:

- 1 hour glucose challenge test (**GCT**) - 50g oral glucola. No fasting needed.
- Recommended GCT value <140mg/dL (detects 80%) [130 value detects 90% women w.GDM]

Follow GCT >/=140mg/dL with diagnostic 3hr.GTT [glucose tolerance test] 100 g. glucola.

- Do fasting, 1h, 2h, 3h serum. Fast @ least 8 hrs. with @ least 150 g. carb intake 3 days prior to test & normal activity level.
GTT Diagnostic Thresholds: [ADA 2003]
Fasting Blood Sugar: 95 mg/dL
Drink 100 g glucola
1 hour: 180 mg/dL plasma level
2 hour: 155 mg/dL
3 hour: 140 mg/dL
- Diagnosis of GDM made if 2 or more values ^ than above plasma levels.

Management
- *May try standard diabetic diet 1st* depends on lab values
- Initiate insulin for fasting > 95 & 2hr postprandial >120.
Interventions: Antepartum
Goal: strict glucose control.

- Provide immediate education to pt./family
- Standard diabetic diet [2000-2500 cal/day].

Distribution of calories: 40-50% carbs, 20% protein, 30-40% fat, (< 1/3\textsuperscript{rd} from saturated fat, 1/3\textsuperscript{rd} polyunsaturated, rest monounsaturated).

Recommend: 3 meals & 3 snacks evenly spaced to avoid swings in blood glucose. Snack @ bedtime. 1200 mg/day calcium, 30 mg/day iron, 400 mcg/day folate.
Antepartum Interventions Cont.

- Exercise [walking, swimming] 30 min. 3-4 x/wk
- Teach daily glucose self-monitoring & urine testing.
- Teach monitoring of fasting & postprandial levels.

  **If diet can’t control glucose, start insulin.**

- Regular & NPH insulin < breakfast & dinner.
- Does not cross placenta. Abdomen site.
- Dose based on weight & gestational age
- Obese pts. ^ dose.
GDM - Interventions cont.

**Intrapartum:** monitor glucose levels q 2hrs.
[insulin given @ 100mg/dL or <].

**Postpartum:** Most return to normal > del.
- 50% pts. with GDM develop type II later in life.
- 6 wk. PP serum glucose
- Children of GDM pts. ^ risk for obesity/diabetes in childhood/adolescence.
Diabetes Mellitus:
- Disorder of fat, CH0 [carbs], protein metabolism
- Caused by insensitivity to insulin or partial or complete lack of insulin secretion by beta cells of pancreas
- exposes organs to chronic hyperglycemia causing tissue damage.
Type I - IDDM predates pregnancy.

- Autoimmune destruction of beta cells of pancreas resulting in absolute insulin deficiency.
- Individuals < 30 yrs.; any age. Affects ~ 1-3/1000 preg.
- Need exogenous insulin to prevent ketoacidosis.

Symptoms: polyuria, polydipsia, significant weight loss. Do HgbA1c ASAP to assess recent serum glucose levels.
Type I cont.

1\textsuperscript{st} trimester:
- exogenous insulin needs may drop 10\textendash{}20\%.
- Hypoglycemia d/t fluctuations & N/V.

2\textsuperscript{nd} half of pregnancy (> 20 wks.):
- insulin needs ^ by 50\textendash{}60\% for Type I & II, respectively compared to pre-pregnancy.
- Have higher levels of depression which negatively affects glucose control.
Management:

Type I –

- Cont. insulin.
- incorporate exercise & diet into daily routine
- monitor glucose levels 5-6/day.
- Endocrinologist during pregnancy.
- Meals @ set intervals.
- Teach pt. s/s hypo & hyperglycemia.

**Goal:** Glucose control reduces risk of complications for pt. & fetus
Type II:

- More common; individuals > 30.
- Recent data [AMA 2001] 9.1% ^ in 18-29 yrs. & 69.9% ^ in 30–39 yrs. [1990 to 1998]
- Combination of insulin resistance & inadequate insulin production.
- Fewer symptoms
- Longer to dx (~ 6.5 yrs.)
- Obesity prevalent in type II.
- May require exogenous insulin; may be controlled w.diet & exercise alone.
Pre-Conception Planning: begin during reproductive years with hx of type I & II.

- Maintain normal A1C 3-6 mos. before conception & during organogenesis (6-8 wks) - minimize risk of spontaneous AB & congenital anomalies
  - A1C level > 7: ^ risk for congenital anomalies & miscarriage. Normal A1C = 4-6 %.
  - Multidisciplinary team: nutritionist, endocrinologist, high risk OB nurse.
  - Educate pt.- managing diet, activity, insulin.
    - Daily food diary to assess compliance.
    - Home visits by RN as needed.
Cardiac Disease:
Impaired cardiac function mainly from congenital/rheumatic heart disease.

Class Description:
Class I: unrestricted physical activity. No symptoms of cardiac insufficiency.
Class II: slight limitation physical activity.
Class III: mod. limitation physical activity.
Class IV: No physical activity.
RISKS FOR MATERNAL MORTALITY CAUSED BY VARIOUS HEART DISEASES

Cardiac Disorder:

**Group 1 - Mortality (0-1 %)**
- ASD; VSD; PDA; Pulmonic or tricuspid disease
- Tetralogy of Fallot (corrected), Bioprosthetic valve; Mitral stenosis

**Group 2 – (5-15%)**
- Aortic stenosis; Coarctation without valve involvement, Tetralogy of Fallot (uncorrected), previous MI, Mitral stenosis with AF, artificial valve

**Group 3 – (25-50%)**
- Marfan Syndrome; MI; pulmonary HTN; Cardiomyopathy.

**Group 4 – (> 50%)**
- CHF; advanced pulmonary edema
Goal: optimal uteroplacental perfusion.

- Thorough medical hx: rheumatic heart dis., scarlet fever, lupus, renal disease
- Birth defects involving heart &/or valves.
- Assess symptoms: SOB, chronic cough, arrhythmias, palpitations, dyspnea @ rest, headache, chest pain, etc.
- Family Hx of cardiac disease.
- Do PE.
- Lab tests: 12 lead EKG, echo, stress test, CBC, SMA12, uric acid levels, O2sat, CxR; ABG’s.
- Risks ^ with maternal age & parity.
**Arrhythmias** in Pregnancy

**Goal:** to convert to sinus rhythm or control ventricular rate by beta-blockers or digoxin.

**Fetal-Neonatal Implications:**
General Prognosis: proceed with pregnancy if disease controlled and mild to moderate. EX. severe valve damage, possible termination advised d/t ^ risk maternal mortality. Correct valve lesions before preg. *Pre-conception planning.
Peripartum Cardiomyopathy: 1 in 3000-4000 pregnancies.

Findings:
- Cardiac failure last month of preg. or within 5 months PP
- Absence of specific etiology for cardiac failure.
- Absence of cardiac disease < last month of preg.

Symptoms:

Management: minimize decreased cardiac performance. Give anticoagulants during/after delivery. High incidence of embolic events.

Prognosis: 50% pt. recover good ventricular function within 6 mos. of delivery; 50% have persistent cardiomegaly w. mortality of 80%. Must weigh risks/benefits to mom/fetus.
Infections:

A. Urinary Tract Infections

Caused by: **E coli**, Klebsiella, Proteus.

- **S&S:** Asymptomatic Bacteriuria = + bacteria in urine cx w.no symptoms.
- **Rx:** Early pregnancy: oral sulfonomides Bactrim]; Late: ampicillin, furodantin.
- if left untreated, infection lead to acute pyelonephritis.
- Can cause PTL; sexual activity > UTI.
B. Cystitis (lower UTI) - Same organisms
- S&S: Dysuria, urgency, frequency, low grade fever, clean catch leukocytes >100,000
- Same as UTI; Same as UTI

C. Acute Pyelonephritis - infection of kidney. Caused by same.
- S&S: chills, fever, flank pain, dysuria, low urine output, ^ B/P, N/V, WBCs, dx with + urine culture.
- Rx: Hospitalization, IVAB; Safe meds. during pregnancy: Bactrim, a fluoroquinolone (Cipro).
- Increased risk of premature birth & IUGR.
D. Monilial Vaginal Infection

- Caused: 80% candida albicans. Caused by change in normal vaginal Ph; pH < 5 - acidic.


- Tx: Intravaginal miconazole suppositories @ hs for 1 wk.
- Teach: yogurt in diet; no douching; cotton underwear.
- Implic: Fetus may contact thrush during delivery. Tx baby w. oral nystatin 1cc q 6h.
- Infant with thrush may give it to mom when breast fdg. Apply nystatin.
Bacterial Vaginosis & Trichomoniasis

- **BV**: Overgrowth of Gardnerella [normal vaginal flora]
  - Thin, watery vaginal discharge with fishy odor. Clue cells seen under microscope. Vaginal pH >5.
  - TX with Flagyl [Metronidazole 500mg BID x 7 days. Do not take med with alcohol. Similar to Antabuse – severe N/V.
  - Risk factor for PTL and PROM.

- **Trichomoniasis**: Different organism caused by parasite *Trichomonas vaginalis*. Vaginal discharge (thin, greenish-yellow, frothy or foamy).
  - An STI
  - Same tx as above. Safe in pregnancy.
  - Risk factor for PTL and PROM.
E. STD’s

- **Chlamydia**
  - Caused By: bacterium *Chlamydia trachomatis*
  - Most common STI in USA
  - PID > infertility by blocking tubes.
  - Often asymptomatic. Thin/purulent discharge, burning & frequency w.urination, lower abd. pain.
  - Pregnant women: Zithromax 1 g single dose; amoxicillin x 7 days.

- Newborn conjunctivitis (erythromycin ointment), neonatal pneumonia, PTL, fetal death. Perinatal transmission occurs in 50% infants where mom is infected @ time of del.
Gonorrhea

- Caused by *Neisseria Gonorrhea*. Bacterial STI.
- Can lead to PID > infertility. Green frothy dc.
- Often asymptomatic in females; males have burning with urination & penile dc.
- Dx - vaginal or urine cx. DOH notifies partners.
- Rx with Rocephin IM [ceftriaxone]. Zithromax [azithromycin] 1 g single dose or amoxicillan po.
Herpes

- Viral infection – no cure.
- HSV I – oral [cold sore] outer lesion.
- HSV 2 – genital – painful, open lesions.
- Vesicles rupture & appear right after exposure or within 20 days.
- Burning sensation with urination is 1st sign.
- Prodrome “tingling” occurs before new outbreak.
- Outbreaks several times/yr.
- Dx: vaginal cx or blood test
- Rx: Acyclovir or Valtrex 500 mg once/day during pregnancy reduces viral load enough to deliver vaginally.
**Syphilis:** Treponema Palladium [Spirochete]

- **Primary Stage:** painless sores, "chancre", approximately 2-3 wks > initial exposure. fever, malaise.
- **Secondary Stage:** 6 wks to 6 mos. Skin eruptions, arthritis, liver enlarged, sore throat,
- **Dx:** VDRL, RPR, FTA-ABS (more specific), Dark field exam: for spirochetes.

**Tx:**

- <1 yr  2.4 million u benzathine penicillin x 1 dose
- >1 yr  SAME MED 1x/wk x 3 wks.
- Sexual partners screened /tx. [allergic to PCN]: tx ceftriazone >1st trimester.
- ~ 40% chance of stillbirth or death > birth. Infant may be born w. "congenital syphilis". Ophthalmia neonatorium: can cause blindness. Appears as conjunctivitis in newborn. Give baby PCN q day x10.
GENITAL WARTS – virus [aka condyloma]

- Soft pink lesions on vulva, vagina, cervix, anus. “Cauliflower appearance”
- HPV Types 6 and 11 cause 90% of genital warts.
- ~120 strains HPV
  - Tx: Trichloroacetic acid, Aldara. Category C
  - Benefits (pregnancy) may be acceptable over potential risks.
  - Contact occurs during vaginal birth. Infant may have laryngeal warts.

Gardasil Vaccine: 3 doses.

- HPV Types 16 & 18 – [70% cervical cancer] and Types 6 & 11 – [90% genital warts] Can be given to males also.