DIFFERENTIAL DIAGNOSIS OF HYPERTENSION IN PREGNANCY

PREECLAMPSIA—new onset or worsening hypertension, ± proteinuria (≥300 mg/day or ≥30 mg/mmol spot urine protein to creatinine ratio), ± adverse clinical signs or symptoms or abnormal labs. A disease >20 weeks gestation

ECLAMPSIA—preeclampsia with generalized tonic clonic seizures

HELLP SYNDROME—a variant of preeclampsia with Hemolysis (i.e. microangiopathic hemolytic anemia), Elevated Liver enzymes (i.e. RUQ or epigastric pain), and Low Platelets

PREEXISTING HYPERTENSION—BP >140/90 mmHg prior to 20th week of gestation, complicates 3–5% of pregnancies, 20% risk of developing preeclampsia

PREECLAMPSIA SUPERIMPOSED UPON PREEXISTING HYPERTENSION—new-onset proteinuria in women with preexisting hypertension or worsening of blood pressure despite 3 antihypertensive medications

GESTATIONAL HYPERTENSION—hypertension ≥20 weeks without proteinuria without adverse effects

CLINICAL FEATURES (CONT’D)

failure, oligohydramnios, IUGR, abnormal uterine or cord dopplers, and fetal demise

PHYSICAL—check vitals (BP in both arms) and look for retinal vasospasm, heart failure, edema (facial, limbs), RUQ tenderness, hyperreflexia, and clonus

CAUSES OF DEATH—maternal cause of death is cerebral hemorrhage in developing countries and fluid overload in developed countries

INVESTIGATIONS

BASIC
- LABS—CBC, Cr, spot urine for protein to creatinine ratio, AST, ALT, albumin, uric acid

SPECIAL
- BLOOD TESTS—peripheral smear, lytes, urea, bilirubin, INR, LDH if indicated
- FETAL EFFECTS—biophysical profile and fetal U/S

MANAGEMENT

ACUTE—ABC, O2 to keep sat >95%, IV with judicious fluid volume

ACUTE LOWERING OF SEVERE HYPERTENSION (SBP ≥160 mmHg or DBP ≥110 mmHg)—labetalol (start with 20 mg IV, repeat 20–80 mg IV q10–30min, or infusion 1–2 mg/min, max 300 mg), nifedipine short-acting capsule 5–10 mg PO q30min, or nifedipine PA tablet 10 mg PO q45min, max 80 mg/day, avoid SL tab) or hydralazine (start with 5 mg IV, repeat 5–10 mg IV q20–30min, max 20 mg). Severe cases may require continuous infusion. Consider urgent delivery if not controlled

CHRONIC MANAGEMENT OF NON-SEVERE HYPERTENSION (SBP 140–159 mmHg or DBP 90–109 mmHg)—target BP at 130–140/80–90 mmHg if renal disease, diabetes, cardiovascular disease, or cerebrovascular disease. Otherwise target BP 130–155/80–105 mmHg. Methyldopa 250–1000 mg PO BID–TID, max 3 g/day, labetalol 100–800 mg PO BID–TID, max 2400 mg/day, nifedipine PA tablet 10–20 mg PO TID, max 180 mg/day, or nifedipine XL 20–60 mg PO daily, max 120 mg/day are good choices. Avoid ACE inhibitors, ARBs, and atenolol. Other β-blockers, clonidine, hydralazine are alternatives
MANAGEMENT (CONT’D)

SEIZURE PREVENTION AND TREATMENT—MgSO₄
4 g IV bolus, then 2 g/h (contraindicated in myasthenia gravis)

DELIVERY—the cure for preeclampsia, eclampsia, and HELLP. Administer steroids to promote fetal lung maturation prior to 34 weeks if early delivery

RECURRANCE—recurrence rate of preeclampsia is 18–66% in subsequent pregnancies. Rule out antiphospholipid syndrome if preeclampsia or placental insufficiency <34 weeks. ASA 81 mg/day before and during next pregnancy is recommended

Pulmonary Diseases in Pregnancy

ASTHMA—treatments similar to non-pregnant patients. β-Agonists, anticholinergics, and glucocorticoids (inhaled, systemic) are all safe. Leukotriene antagonists only if refractory to above. Keep O₂ sat >95% at all times. Stress dose steroids during delivery if patient required moderate systemic steroids for >3 weeks in the preceding year

VENOUS THROMBOEMBOLISM

PATHOPHYSIOLOGY—increased risk of DVT/PE due to ↑ factors II, VII, X, and fibrinogen, as well as ↓ protein S and fibrinolytic activity, especially during T3. Also stasis due to ↓ venous tone and flow. Similar risk of DVT/PE in each trimester but highest post-partum; 90% of DVT in pregnancies are left sided

DIAGNOSIS—if suspect venous thromboembolism, consider initiation of LMWH while waiting for investigations. For DVT workup, perform compression U/S; if pelvic vein DVT suspected, consider MRV pelvis (without gadolinium in pregnancy), doppler study, or (post-partum) CT of pelvic veins. Otherwise, repeat compression U/S in 5–7 days if still symptomatic. For PE workup, rule out other etiologies by performing a CXR. If PE still suspected, consider initial low-dose perfusion (Q) scan and proceed with CT chest if abnormal. CT chest is associated with lower fetal radiation exposure than V/Q scan in T1–2, but higher risk of maternal breast cancer

RADIATION RISKS—fetal exposure of <5 cGy [5 rad] accumulatively in each pregnancy is acceptable, but oncologic effects controversial (e.g. childhood leukemia). Consider proximity of fetus to radiations site (i.e. radiation from CT chest > V/Q scan in T3)

Related Topics
Asthma (p. 1)
Pulmonary Embolism (p. 8)

TREATMENTS—LMWH (monitor anti-Xa level). LMWH is contraindicated for 12–24 h prior to neuraxial analgesia, so switch to unfractionated heparin for ≥24 h prior to labor or cesarean delivery. Switch to warfarin post-delivery and continue for a minimum of 6 months after an acute clot. Rule out thrombophilia. IVC filters may be considered if prolonged interruption of anticoagulation is unsafe. In regard to DVT prophylaxis for future pregnancies, give low-dose SC LMWH during pregnancy and 6 weeks post-delivery. Warfarin is teratogenic (deformities, fetal hemorrhage). Thrombolysis is generally contraindicated as Risk of fetal demise

AMNIOTIC FLUID EMBOLISM

PATHOPHYSIOLOGY—can occur during labor and delivery or with uterine manipulation. Risk factors include older age and multiparity

DIAGNOSIS—clinical diagnosis. Differential diagnosis includes septic shock, pulmonary embolism, aspiration pneumonia, uterine rupture, abruptio placentae, and venous air embolism

TREATMENTS—supportive. ICU admission. Rapid delivery of the fetus

NEJM 2008 359:19
AMNIOTIC FLUID EMBOLISM (CONT’D)

COMPLICATIONS—10% of maternal mortality, 25–50% of which die within the first hours of onset of the disease. Patients who survive are at high risk for DIC and ARDS

Cardiac Diseases in Pregnancy

PATHOPHYSIOLOGY

PHYSIOLOGIC CHANGES DURING PREGNANCY—↑ cardiac output and ↓ peripheral vascular resistance. Risk of cardiac decompensation highest in 28–32 weeks (maximum increase in maternal blood volume), labor (hemodynamic changes), and post-partum (fluid shifts)

HIGH-RISK CARDIOPULMONARY CONDITIONS—generally advise against pregnancy in following conditions: tetralogy of Fallot with severe cyanosis, Eisenmenger’s syndrome, severe pulmonary hypertension, functional limitation NYHA 3 or 4, recent cardiac transplantation with high-dose immunosuppression, Marfan’s syndrome with aortic root >40 mm [1.6 in.], interstitial pulmonary fibrosis, lymphangioleiomyomatosis, and active lung cancer

Related Topics
Endocarditis (p. 52)
Heart Failure (p. 33)
Valvular Disorders (p. 51)

VALVULAR DISORDERS

REGURGITANT VALVULAR HEART DISEASE—may improve during pregnancy due to ↓ systemic vascular resistance. Avoid Valsalva maneuver. Assist second stage with forceps

STENOTIC VALVULAR HEART DISEASE—may worsen during pregnancy. Consider β-blockers to decrease HR in mitral stenosis. Supportive measures with aggressive pain control during labor. Avoid fluid overload

PROSTHETIC HEART VALVE—for metal valves, continue oral anticoagulation until conception, can switch to LMWH before 6th week and continue throughout first trimester and possibly throughout pregnancy (aim for higher anti-Xa level). Warfarin, which crosses the placenta and may cause fetal bleeds, may be considered during 2nd and 3rd trimesters for more thrombogenic valves until 36th week, and then switch back to unfractionated heparin in preparation for delivery. Preconception counseling should be emphasized

ENDOCARDITIS PROPHYLAXIS—normally not required for vaginal delivery and cesarean sections; optional for high-risk lesions (complex congenital heart disease, prosthetic heart valve, cardiac transplant recipients with valvuloplasty, previous endocarditis)

MYOCARDIAL DISORDERS

PERIPARTUM CARDIOMYOPATHY—T3 to 5 months post-partum. One-third recovers spontaneously. May treat with diuretics, β-blockers (except atenolol), nitrates, hydralazine, and digoxin. Avoid ACE inhibitors and ARBs. Anticoagulate as risk of thromboembolism. Patients with residual left ventricular dysfunction are at high risk of progression or death with future pregnancies and should be counseled to avoid future pregnancies

ISCHEMIC HEART DISEASE—may become more common in pregnancy. Stress echocardiogram (preferred), exercise stress test, MIBI, and angiograms (radiation) can be done

RHYTHM DISORDERS

PALPITATIONS—sinus tachycardia and ectopic beats are common. Increased SVT in patients previously diagnosed with SVT. May treat with adenosine, β-blockers (except atenolol), calcium channel blockers, or digoxin. DC cardioversion if unstable, but fetal monitoring devices should be removed first. CPR can be performed on pregnant woman, but pull uterus to left side to decrease IVC compression and improve venous return

Hepatic Diseases in Pregnancy

DIFFERENTIAL DIAGNOSIS

NOTE: GESTATIONAL AGE HELPS WITH DIAGNOSIS

HYPEREMESIS GRAVIDARUM (T1–2, incidence 0.3–1%)—nausea, vomiting, mild jaundice, weight loss, ↑ ALT > AST, N bili

INTRAHEPATIC CHOLESTASIS OF PREGNANCY (T2–3, incidence 0.1–0.2%)—functional disorder of bile formation with severe pruritus. Jaundice in 20–60% 1–4 weeks after pruritus starts. ↑ ALT, ↑ AST, ↑ bilirubin (less common), ↑ bile acids. Resolves following delivery without hepatic sequelae. Fetus at risk for sudden death especially with bile acids >40 μmol/L [16 μg/mL]
DIFFERENTIAL DIAGNOSIS (CONT’D)

ACUTE FATTY LIVER OF PREGNANCY (T3, incidence 0.008%)—may be associated with preeclampsia. Characterized by severe liver dysfunction (encephalopathy, hypoglycemia, coagulopathy) and commonly jaundice. ↑ ALT, ↑ AST, ↑ bilirubin, ↑ WBC, ↑ PT, ↑ uric acid. U/S is often normal (microvesicular fat on biopsy) and CT shows a low-density liver. May progress to acute hepatic failure and DIC in >75%. Increased maternal and fetal mortality

PREECLAMPSIA/ECLAMPSIA (T2–3, incidence 5–10%)—see section under preeclampsia. May progress to HELLP (4–12%), DIC (7%), jaundice (5–14%) later

HELLP SYNDROME (T3, incidence 0.1%)—preeclampsia symptoms. ↑ ALT, ↑ AST, ↑ bilirubin, ↓ platelets, ↓ LDH. May progress to DIC (30%)

OTHER CONDITIONS—drug-induced hepatitis, ascending cholangitis, acute cholecystitis, malignancy, HBV, and HCV

CLINICAL FEATURES

HISTORY—jaundice, pruritus, abdominal pain, ascites, swelling, encephalopathy, nausea and vomiting, headache, visual disturbances, fever, obstetrical history (current pregnancy course, previous births, previous preeclampsia), past medical history (hypertension, hepatitis, alcohol, IDU), and medications

PHYSICAL—check vitals (hypertension), edema (facial, limbs), heart (elevated JVP, S3, S4), hepatic tenderness, ascites, jaundice, and hyperreflexia

INVESTIGATIONS (CONT’D)

• MICROBIOLOGY—HBV and HCV serology
• IMAGING—U/S abd
• LIVER BIOPSY—if not coagulopathic

MANAGEMENT

HYPEREMESIS GRavidarium—rule out molar pregnancy and hyperthyroidism. Supportive fluids. Metoclopramide, dimenhydrinate, and diclectin acceptable. Consider ondansetron if refractory. Continuous metoclopramide infusion if severe

INTRAHEPATIC CHOLESTASIS OF PREGNANCY—ursodeoxycholic acid or cholestyramine, increase fetal monitoring, consider early delivery as risk of fetal demise if high bile acids

ACUTE FATTY LIVER OF PREGNANCY—vitamin K if coagulopathic, early delivery

HELLP—anti-hypertensive, MgSO4, early delivery

HEPATITIS B OR C—no proven treatment during pregnancy (but risk of vertical transmission especially if co-infection with HIV)

SPECIFIC ENTITIES

OTHER GI DISORDERS
• GERD—very common during pregnancy. Treatments include lifestyle changes, antacids, ranitidine, PPIs, and metoclopramide
• CHOLECYSTITIS—pregnant women are at increased risk due to hormonal changes. Medical management with IV fluids, NG, and opioids. Broad-spectrum antibiotics may be added for severe disease. Cholecystectomy safest during 2nd trimester

Related Topics
Acute Liver Failure (p. 128)
Dyspepsia (p. 113)

Infectious Diseases in Pregnancy

URINARY TRACT INFECTIONS

ASYMPTOMATIC BACTERIURIA—occurring in 2–7% of pregnancies, associated with pre-term birth, low birth weight, and perinatal mortality. 30–40% will develop symptomatic UTI if untreated, and therefore should be treated (depending on culture and local antibiotic resistance pattern, consider amoxicillin–clavulanate 500 mg PO BID ×7 days, nitrofurantoin 100 mg PO BID ×7 days [risk of hemolytic anemia]). Avoid trimethoprim if alternatives available. Follow-up culture 1 week following treatment completion and then monthly until pregnancy complete

ACUTE CYSTITIS—occurring in 1% of pregnancies, with treatment and follow-up as asymptomatic bacteriuria

PYEONEPHRITIS—occurring in <1% of pregnancies, complicated by septic shock and ARDS in 20%. In-patient treatment with IV antibiotics (cefazolin, ceftriaxone, or ampicillin plus gentamicin) until symptomatic improvement and afebrile for 24–48 h then PO based on drug sensitivities. Low-dose suppressive antibiotic (nitrofurantoin 50–100 mg PO qhs [risk of hemolytic anemia] or cephalaxin 250–500 mg PO qhs) for remainder of pregnancy as recurrent pyelonephritis occurs in 6–8% of women without prophylaxis
**Human Immunodeficiency Virus (HIV)**

**Antepartum Care**—determine HIV symptoms, infections, immunization status, and perform ophthalmologic examination if CD4 < 50/mm³. Baseline testing include CBCD, lytes, urea, Cr, AST, ALT, ALP, bilirubin, CD4 count, viral load, TB skin test, toxoplasma, VDRL, pap smear, cervical swabs for gonorrhea and chlamydia, CMV, HBV, and HCV serologies. Counsel regarding perinatal transmission (30% without treatment, <1% with optimal and effective combination therapy), contraceptive use during pregnancy (condoms), and mode of delivery. If on HAART already, continue as combination therapy which should contain AZT. For pregnant women not already on HAART, consider zidovudine from 2nd trimester onwards. Prophylaxis for opportunistic infections same as in non-pregnancy patients. Amniocentesis or other invasive procedures may increase vertical transmission risk.

**Intrapartum Care**—upon onset of labour or rupture of membranes, give zidovudine 2 mg/kg IV over 1 h, then 1 mg/kg until delivery (even if on HAART already). For cesarean section, start infusion at least 3 h before procedure. Consider use of cesarean delivery if viral load > 1000/mL. Avoid invasive monitoring, use of instruments to assist delivery, and prolonged interval between rupture of membranes and delivery.

**Postpartum Care**—treat newborn with zidovudine for 6 weeks, to be followed by PJP prophylaxis. Determine HIV status at 1–2 days, 2 weeks, 1–2 months, and 3–6 months. Avoid breast feeding. Ensure good support system for mother. Counsel regarding contraceptive use.

**TORCHES Infections**

**Infections Associated with Birth Defects**

★ TORCHES ★—TORxoplasma, Rubella, CMV, HErpes, and Syphilis infections during pregnancy are associated with birth defects.

**Tuberculosis**

**Management**—treat patient as risk of infection to fetus is greater than risk of medications. Use isoniazid, rifampin, and ethambutol for 9 months minimum. Breastfeeding is safe. Pyridoxine 25 mg PO daily is recommended for all pregnant or breastfeeding women taking isoniazid.

**Antibiotics**

**Acceptable**—penicillins, cephalosporins, azithromycin, vancomycin, metronidazole, clindamycin, erythromycin (except erythromycin estolate), nitrofurantoin (caution as risk of hemolytic anemia), and acyclovir. Consider trimethoprim–sulfamethoxazole (avoid in first trimester but use with folate if no other alternatives) and aminoglycosides (except streptomycin) in some circumstances.

**Avoid**—tetracyclines, streptomycin, fluoroquinolones.

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**Endocrine Disorders in Pregnancy**

**Diabetes in Pregnancy**

**Risk Factors for Gestational Diabetes**—previous history of gestational diabetes, prior delivery of macrosomic infant, ethnic group (Aboriginal, Hispanic, Asian, African), maternal age ≥ 35, obesity, PCOS, polyhydramnios, multiple gestation, fetal macrosomia (>4000 g or >90th percentile) or unexplained stillbirth, family history of diabetes, corticosteroid use.

**Diagnosis of Gestational Diabetes**

- **Step 1**: Gestational Diabetes Screen (GDS) 50 g oral glucose and draw blood after 1 h
  - If blood glucose ≥ 10.3 mmol/L [≥185 mg/dL], diagnosis of GDM can be made
  - If blood glucose ≥ 7.8 mmol/L [≥140 mg/dL], perform 2 h OGTT
  - If blood glucose is < 7.8 mmol/L [<140 mg/dL], then no GDM but re-test if continued at high risk or high suspicion (e.g. macrosomia, polyhydramnios)

- **Step 2**: 2 h Oral Glucose Tolerance Test (OGTT) 75 g glucose after overnight fast
  - Abnormal if fasting blood glucose ≥ 5.3 mmol/L [≥95 mg/dL]
  - Or 1 h blood glucose ≥ 10.6 mmol/L [≥190 mg/dL]
  - Or 2 hour blood glucose ≥ 8.6 mmol/L [≥155 mg/dL]

- **Step 3**: Diagnosis based on OGTT
  - If 1 value abnormal, then impaired glucose tolerance of pregnancy (IGTP)
  - If ≥ 2 values abnormal, then GDM

**Monitoring**—monitor blood glucose ac all meals for type 1 diabetics (goal < 5.3 mmol/L [<95 mg/dL]), 1 h post all meals (goal < 7.8 mmol/L [<140 mg/dL]), and qhs (goal < 6 mmol/L [<108 mg/dL]), Hyperglycemia during the 1st trimester is a teratogen. Check urine ketones every morning.

**Related Topics**

HIV (p. 259)

Tuberculosis (p. 250)

Urinary Tract Infections (p. 244)
**DIABETES IN PREGNANCY**

**TREATMENTS**—diabetic diet and exercise. Ensure excellent glycemic control with normal HbA1C prior to and throughout pregnancy. Increase bedtime snack portion if ketonuria in morning

- **TYPE 1 DIABETICS**—insulin injections and insulin pump equally effective
- **TYPE 2 DIABETICS**—switch oral hypoglycemics to insulin, preferably preconception
- **GESTATIONAL DIABETES**—insulin (Insulin Lispro or Aspart TID ac meals and Humulin N or NPH qhs) required if hyperglycemia persists. Glyburide acceptable if mild

**COMPLICATIONS**—maternal complications include preeclampsia, polyhydramnios, preterm labor, progression of existing diabetic retinopathy and nephropathy. Fetal complications include macrosomia, shoulder dystocia, malformations, intrauterine death, cardiomyopathy, polycythemia, hypoglycemia, hypocalcemia and hyperbilirubinemia

**http://www.diabetes.ca/files/cpg2008/cpg-2008**

**HYPOTHYROIDISM IN PREGNANCY**

**PATHOPHYSIOLOGY**—may be due to ↑ thyroid-binding globulin, ↑ volume of distribution of T4, and ↑ destruction of T4 by placental deiodinases. There is also increased metabolic demand during pregnancy

**TREATMENTS**—levothyroxine can be safely given during pregnancy. Dose may need to be increased in pregnancy. Take levothyroxine separate from vitamins, which decrease its absorption

**COMPLICATIONS**—untreated hypothyroidism can lead to neurodevelopmental abnormalities in the child

**GRAPES DISEASE**—most common cause of hyperthyroidism in pregnancy (95%). TSH receptor antibodies can cross placenta to cause thyrotoxicosis in fetus and fetal goiter. Exacerbations may happen in T1 and postpartum. Improvement may happen in T3. Classically improves in pregnancy

**POSTPARTUM THYROIDITIS**—clinically just like subacute thyroiditis, but autoimmune in origin and goiter is painless. Usually begins with a hypothyroid phase followed by a hyperthyroid phase. If patient has postpartum depression, consider this diagnosis and perform thyroid uptake study. Nursing mothers who had the radioactive uptake study should pump and dump breast milk for 72 h before refeeding

**DIAGNOSIS**—once hyperthyroidism is diagnosed during pregnancy (high free T4, low TSH), the cause may be difficult to establish. Postpartum follow-up may help. Thyroid radionuclide scan is contraindicated in pregnant women. Consider anti-TSH receptor antibody if suspect Graves disease

**TREATMENTS**

- **GRAVES’ DISEASE**—β-blockers (avoid atenolol) can be safely used in pregnancy and lactation. PTU is the anti-thyroid agent of choice before and during the first trimester (as methimazole is associated with fetal abnormalities during this period), while methimazole should be used for the remainder of the pregnancy. Use the lowest dose of PTU possible. Graves’ generally improves in pregnancy. β-Blockers may lead to bradycardia, hypoglycemia, and IUGR

**POSTPARTUM THYROIDITIS**—may not require treatment if mild symptoms. For significant hyperthyroidism symptom, give β-blocker. For hypothyroidism, give L-thyroxine 50–100 μg PO daily ×8–12 weeks and then reassess

**COMPLICATIONS**—decreased fertility, ↑ miscarriage, premature labor, thyroid storm (especially during labor and delivery), IUGR, and perinatal mortality

**COMPLICATIONS**—decreased fertility, miscarriage, premature labor, thyroid storm (especially during labor and delivery), IUGR, and perinatal mortality

**Other Disorders in Pregnancy**

**SEIZURES IN PREGNANCY**

**PATHOPHYSIOLOGY**—for women with known epilepsy, 25% will have ↑ frequency, 25% will have ↓ frequency, and 50% will not change in pregnancy. It is important to be seizure free, ideally off medications, for at least 6 months prior to conception to ensure good outcome. Risk of seizures in offspring is elevated at 5%. Eclampsia, intracerebral bleed, and cerebral vein thrombosis may lead to seizures in pregnancy

**TREATMENTS**—valproic acid has a relatively high risk of neural tube defects and should be switched to alternate antiepileptic pre-pregnancy if possible. Phenytoin,
carbamazepine, and phenobarbital are all teratogenic but may be used if indicated and after appropriate counseling. Lamotrigine seems to have reasonable data in pregnancy. *Folic acid* 0.4 mg PO daily should be prescribed to all women on antiepileptics in the childbearing age. Those planning a pregnancy should take *folic acid* 5 mg PO daily in the preconception period and in first trimester, then 1 mg PO daily throughout remainder of pregnancy. *Vitamin K* may be recommended during the last month of pregnancy to reduce the risk of hemorrhagic complications in newborns.

**LUPUS IN PREGNANCY**

**LUPUS EXACERBATIONS**—may have increased flares during pregnancy and postpartum if not in remission for >6 months prior to conception. Plaque-nil, azathioprine, and corticosteroids may be used during pregnancy. Avoid NSAIDs in T3

**COMPLICATIONS**—increased risk of prematurity and in utero fetal death. Patients with nephritis may have severe exacerbations with acute renal failure, preeclampsia, and maternal death. Children of patients with anti-SSA and anti-SSB are at increased risk for congenital heart block and neonatal lupus. Patients with antiphospholipid antibodies are at increased risk for preeclampsia, miscarriage, and possibly thrombosis.

**BREAST CANCER IN PREGNANCY**

**DIAGNOSIS**—staging workup similar to non-pregnant women. Use MRI (without gadolinium) or U/S instead of CT if imaging of abdomen required

**TREATMENTS**—mastectomy preferred over lumpectomy to avoid radiation. If adjuvant radiation indicated, it should be deferred until after delivery. Anthracycline containing adjuvant chemotherapy can usually be safely given during 2nd and 3rd trimesters, but not in 1st trimester or within 2 weeks of delivery. Methotrexate is absolutely contraindicated and taxane/dose dense regimens should be avoided. Hormonal therapy is contraindicated during pregnancy. Breast feeding contraindicated in women on hormonal therapy or chemotherapy. Stage by stage, gestational breast cancer has similar prognosis to non-pregnant counterpart.

**PAIN CONTROL IN PREGNANCY**

**ACCEPTABLE**—acetaminophen, opioids

**CONTRAINDICATED**—NSAIDs in T3 (may use in T1 or T2)

**THROMBOCYTOPENIA IN PREGNANCY**

**GESTATIONAL THROMBOCYTOPENIA** (T3)—asymptomatic and resolves after pregnancy. May be difficult to distinguish from ITP except platelet count usually higher (>70 × 10^9/L) in gestational thrombocytopenia. Follow platelet counts regularly.

**THROMBOCYTOPENIA IN PREGNANCY (CONT’D)**

**ITP** (T1–3)—may use prednisone and IVIG in pregnancy. Platelet transfusion if acute. Monitor closely. Splenectomy is last resort (safest in T2). Epidural is generally performed if platelet >80 × 10^9/L. Cesarean delivery safe if platelet >50 × 10^9/L; 5% of newborns may also have thrombocytopenia, requiring close monitoring in first few days

**HELLP** (T2–3)—supportive, early delivery, steroids for lung maturity if delivered <34 weeks (see earlier sections)

**TTP/HUS**—plasma exchange, dialysis as needed

**OTHERS**—DIC, bone marrow disease, vitamin B12 deficiency, drugs, autoimmune diseases, and hypersplenism

**ANTI PHOSPHOLIPID ANTIBODY SYNDROME IN PREGNANCY**

**PATHOPHYSIOLOGY**—antibody against phospholipids or cell surface proteins bound to anionic phospholipids. These include lupus anticoagulants, anti-cardiolipin antibody (false-positive VDRL), and anti-β2GP1 antibody — most lead to hypercoagulable state, some may inhibit coagulation

**CLINICAL FEATURES**—venous and arterial thrombosis and rarely hemorrhage affecting the lungs, heart, CNS, GI, kidneys, skin, and eyes. Also thrombocytopenia (via ITP, TTP), Raynaud’s phenomenon, ↑ risk of preeclampsia/eclampsia, recurrent fetal losses or >10-week losses and intrauterine growth restriction

**CAUSES**—primary APS, secondary APS (various rheumatic diseases such as SLE, infections such as HIV and drugs)

**DIAGNOSIS**—clinical criteria of VTE or arterial thrombosis, or 3 unexplained consecutive T1 losses, or 1 or more unexplained morphologically normal T2 loss, or <34-week preeclampsia/eclampsia/placental insufficiency; plus laboratory criteria of elevated anticardiolipin antibodies, or lupus anticoagulant, or anti-β2GP1 antibodies, confirmed >12 weeks apart. Diagnosis requires at least one clinical and one laboratory criteria

**TREATMENTS**—for women with APS associated with adverse obstetric outcomes, give prophylactic LMWH and low-dose ASA during pregnancy. For women with APS associated with VTE, same antenatal treatment plus anticoagulation prophylaxis postpartum for 6 weeks (see p. 157 for more details on ANTI PHOSPHOLIPID ANTIBODY SYNDROME)

**Related Topics**

Antiphospholipid Antibody Syndrome (p. 157)

Breast Cancer (p. 189)

Lupus (p. 279)

Thrombocytopenia (p. 151)

Seizures (p. 309)
