ACUTE ASTHMA EXACERBATION

BACKGROUND
■ 4% of adult population has asthma
■ In general, sx often worsen during 28–36 wk of pregnancy; acute exacerbations rare in last 4 wk or in labor

DIAGNOSIS

History
■ Sx include cough, dyspnea, wheezing; fever, chills, malaise less common
■ Ask about prior attacks, current Rx, baseline peak expiratory flow rate (PEFR), precipitating events (upper respiratory tract infection, allergen exposure)

Physical examination
■ Check temp, pulse oximetry (note: pulse oximetry does not assess pt’s ability to clear CO₂)
■ Examine for cyanosis, hyperinflation, use of accessory muscles, pulsus paradoxus

Diagnostic tests
■ Laboratory tests: ABG, CBC
■ Specific diagnostic tests: decrease in PEFR, FEV1
■ Imaging tests: AP/lateral CXR
■ Screening tests: daily PEFR

DIFFERENTIAL DIAGNOSIS
■ Pulmonary edema
■ Pulmonary embolism
■ Bronchitis
■ Pneumonia

COMPLICATIONS
■ Maternal complications: respiratory failure, preterm birth
Acute Asthma Exacerbation

Fetal complications: prematurity, low birthweight, increased perinatal mortality (esp. w/ severe disease)

PROGNOSIS
- Pregnancy represents a state of compensated respiratory alkalosis; maternal PCO₂ > = 35 mmHg in room air suggests impending respiratory failure

MANAGEMENT

General measures
- O₂ supplementation to maintain O₂ saturation > = 95%, PO₂ > = 70%
- Continuous pulse oximetry to follow oxygenation
- Adequate hydration
- Serial ABGs

Specific treatment
- Inhaled beta-2-agonist (bronchodilator) Rx q 20–30 min × 3 doses
- If initial response adequate (ie, increase in PEFR to > = 70% predicted or baseline, if known), continue bronchodilator Rx & follow as outpatient
- If response inadequate, continue Rx for 2–3 h; consider admission for measured PEFR <70% predicted
- Consider IV corticosteroid Rx for PEFR 40–70% baseline (or predicted) after 2–3 h
- Stress-dose steroids (IV hydrocortisone 80 mg q8h) in labor if history of steroid Rx in last 6 mo

Contraindications
- Cardiogenic disease relative contraindication to beta-agonist Rx

Side effects & complications of treatment
- Maternal adrenal suppression (can be avoided w/ stress-dose steroids in labor)
Follow-up care

- Regular outpatient visits
- Referral to pulmonologist

SUBSEQUENT MANAGEMENT

- Severity & frequency of acute exacerbations similar in subsequent pregnancies

ACUTE CYSTITIS

BACKGROUND

- Most common medical complaint of pregnancy
- Incidence: 1–4% of all pregnancies
- Organisms: *Escherichia coli* (90%), *Staphylococcus saprophyticus* (4–7%)

DIAGNOSIS

History

- Sx may include frequency, dysuria, urgency, suprapubic pain
- *Risk factors*: diabetes, urinary tract anomaly, prior urinary tract infection/pyelonephritis in index pregnancy, sickle cell trait/disease

Physical examination

- Suprapubic tenderness
- Flank pain, costovertebral angle tenderness, fever, systemic complaints usually absent

Diagnostic tests

- Urine dip can be positive for nitrates, leukocyte esterase
- Definitive Dx made by urinalysis (\(\geq 100,000\) colony-forming units/mL of single pathogenic organism in midstream clean-catch urine specimen)
- Imaging studies not indicated
- Check CBC if patient febrile
Acute Cystitis

DIFFERENTIAL DIAGNOSIS
- Mycotic/bacterial vaginosis w/ contamination of urine specimen
- Asymptomatic bacteriuria
- Pyelonephritis

COMPLICATIONS
- Maternal complications: progression to pyelonephritis, urosepsis, ARDS, preterm labor
- Fetal complications: preterm birth, low birthweight

PROGNOSIS
- Full resolution can be expected w/ adequate Rx; increased risk of pyelonephritis/urosepsis if Rx inadequate
- Screening/treatment prevents 80% of pyelonephritis in pregnancy

MANAGEMENT

General measures
- Aggressive oral hydration
- Outpatient Rx acceptable in absence of pyelonephritis

Specific treatment
- Antibiotic Rx for 3 d adequate for otherwise healthy women (consider 5-d course for women w/ concurrent chronic disease); single-dose Rx assoc. w/ increased failure rate in pregnancy
- Rx options include trimethoprim/sulfamethoxazole 160/180 mg po bid, nitrofurantoin monohydrate/macrocrystals 100 mg po bid, cephalexin 500 mg po qid
- Adjust Rx according to culture results, if indicated

Prevention
- Periodic screening urinalysis in women at high risk for urinary infections
Acute Cystitis

**Amniotic Fluid Embolism**

**SUBSEQUENT MANAGEMENT**

- Repeat urine culture in 10 d after completion of Rx (“test of cure”)
- If Rx unsuccessful, consider noncompliance, failed Rx (poor antibiotic selection, antibiotic resistance)
- Consider suppressive Rx for 6 wk if repeat culture positive w/ same organism.

**AMNIOTIC FLUID EMBOLISM**

**BACKGROUND**

- Rare, unpredictable, catastrophic obstetric event
- 10% of maternal mortality in U.S.
- *Incidence*: 1/8,000–1/85,000 births

**DIAGNOSIS**

**History**

- Prodromal sx may include sudden chills, sweating, anxiety
- *Risk factors*: multiparity, advanced maternal age, hypertonic labor, male fetus, intrauterine fetal demise, oxytocin, amniotomy, abruption, intrauterine pressure catheter, chorioamnionitis, cesarean, preeclampsia, intrauterine saline injection (abortion)

**Physical examination**

- *Clinical*: Dx characterized by acute-onset respiratory distress, cyanosis, hypotension, tachycardia, hypoxemia, neurologic manifestations (seizures, coma), hemorrhage in labor/delivery or early puerperium

**Diagnostic tests**

- *Laboratory tests*: check CBC, DIC panel
- *Specific diagnostic tests*: clinical Dx, identification of amniotic fluid (mucin, fetal squames) in pulmonary vasculature at postmortem not pathognomonic
Amniotic Fluid Embolism

**Imaging:** check CXR, V/Q scan (shows decreased perfusion); of little value in acute setting. Transesophageal ultrasound useful in acute assessment of pulmonary embolism in intubated pt.

**Differential Diagnosis**
- Pulmonary embolism
- Pulmonary edema
- Venous air embolism (assoc w/ ruptured uterus, placenta previa, persistent atrial septal defect)
- Aspiration
- Eclampsia
- Drug overdose/withdrawal
- Other causes of DIC

**Complications**
- Maternal complications: shock, DIC, blood transfusion; very high maternal mortality rate (60–90%), permanent neurologic sequelae (85% of survivors)
- Fetal complications: intrauterine fetal demise, hypoxic ischemic cerebral injury if fetus undelivered

**Prognosis**
- Death not inevitable if early Dx, aggressive management, including intubation and possible pulmonary bypass

**Management**

General measures
- High index of suspicion, early Dx
- Monitor vital signs, O₂
- Anesthesia consult, central hemodynamic monitoring, IV access
- Immediate delivery regardless of gestational age
- Rx primarily supportive
Amniotic Fluid Embolism

Specific treatment
- CPR, Rx hypoxemia (supplemental O₂, mechanical ventilation)
- Control bleeding (correct DIC, uterotonic Rx)
- Correct anemia/coagulopathy w/ aggressive blood product transfusion
- Maintain arterial PO₂ >60 mmHg, O₂ saturation >90%; Rx bronchospasm (terbutaline, aminophylline, ? steroids)
- Maintain SBP >90 mmHg, urine output >25 mL/h; inotropic support (dopamine) as needed

Contraindications
- Regional anesthesia contraindicated in acute setting; general endotracheal anesthesia for cesarean
  - Airway management crucial and intubation highly likely
  - Pressor support likely to be acutely needed
  - May require pulmonary bypass
- Avoid heparin in established DIC

SUBSEQUENT MANAGEMENT
- Recurrence rate not clear, likely low.

ANTENATAL FETAL TESTING

BACKGROUND
- Goal: early identification of fetus at risk for preventable morbidity due to hypoxemia
- Assumptions: (1) hypoxemia leads to permanent injury; (2) tests discriminate between asphyxiated, nonasphyxiated fetuses; (3) early detection can prevent adverse outcome
- At most, 15% of cerebral palsy due to intrapartum hypoxemia

DIAGNOSIS

History
Indications for testing:
Antenatal Fetal Testing

1. **Maternal factors:** diabetes, hypertension, hyperthyroidism
2. **Fetal factors:** intrauterine growth restriction, increased fetal activity, oligo/polyhydramnios
3. **Pregnancy-associated:** placental abruption, postterm pregnancy

**Physical examination**
- Usually unhelpful

**Diagnostic tests**
1. **Fetal movement charts** ("kick counts"): count all movements in 1 h or count time for 10 kicks; 2–3 times/d; any decreased movement requires further evaluation
2. **Contraction stress test** (CST): measures response of fetal heart rate to contractions (3/10 min required to interpret test); (+) CST defined as decelerations w/ ≥50% contractions
3. **Nonstress test** (NST): changes in fetal heart rate pattern w/ time; reflects maturity of fetal autonomic nervous system; absence of reactivity (2 accelerations of 15 bpm × 15 sec in 20 min) depends on gestational age: 50% at 24–28 wk, 15% at 28–32 wk
4. **Biophysical Profile** (BPP): NST + 4 sonographic variables: breathing ≥30 sec/30 min, movements ≥3/30 min, tone (flexion/extension) ≥1/30 min, amniotic fluid volume ≥2 cm single vertical pocket

**DIFFERENTIAL DIAGNOSIS**
Causes of irreversible cerebral injury other than hypoxia:
- Congenital abnormalities
- Intracerebral hemorrhage
- Infection
- Drugs
- Trauma
- Hypotension
- Metabolic (thyroid, hypoglycemia)
COMPLICATIONS

- Maternal complications: increased cesarean delivery rate
- Fetal complications: iatrogenic prematurity due to false-positive testing

PROGNOSIS

- Negative predictive value (intrauterine fetal demise < 1 wk following (−)/reassuring testing) consistent for all tests at 0.3–1.9/1,000 pregnancies
- Positive predictive value varies widely; severely abnormal fetal testing associated with adverse outcome in only 25–40% of cases
- Interpret testing in light of gestational age, underlying clinical risk factors, congenital anomalies

MANAGEMENT

General measures

- All antenatal tests probably equally efficacious

Contraindications

- Contraindications to CST: preterm premature rupture of membranes, previa, preterm labor, prior cesarean

SUBSEQUENT MANAGEMENT

- Specific to suspected pathology.

ANTIPHOSPHOLIPID ANTIBODY SYNDROME

BACKGROUND

- Autoimmune disorder characterized by circulating antibodies against membrane phospholipid & one or more specific clinical syndromes
- Incidence depends on population screened (0.5–3% of non-pregnant, 2–4% of pregnant, & 4–5% of women w/ prior pregnancy loss have low-titer anticardiolipin antibody [ACA] IgG;
Antiphospholipid Antibody Syndrome

among women w/ recurrent pregnancy loss, 5–20% have moderate to high titer ACA, & 5–10% are + for lupus anticoagulant [LAC])

DIAGNOSIS

Two elements are required for Dx:

1. **Appropriate clinical setting:**
   - Recurrent pregnancy loss
   - Unexplained thrombosis
   - Autoimmune thrombocytopenia
   - ? Preeclampsia
   - ? Intrauterine growth restriction
   AND

2. **A confirmatory serologic test:**
   - *LAC* is an unidentified antibody causing increases of phospholipid-dependent coagulation tests (aPTT, Russel Viper Venom test) by binding to prothrombin-activator complex; in vivo, LAC causes thrombosis; LAC results reported as present or absent (no titers)
   - Specific antiphospholipid antibodies measured by ELISA (most commonly ACA) assoc. w/ anticoagulant activity in vitro but procoagulant activity in vivo; ACA IgM alone &/or low-positive IgG may be nonspecific; moderate to high levels of ACA IgG required for Dx
   - ACA & LAC similar but not identical antibodies; may coexist in vivo (70–80% of women w/ LAC are ACA (+); 10–30% of ACA (+) women have LAC)
   - False-positive test for syphilis common but not sufficient to make Dx of antiphospholipid antibody syndrome (APS)

DIFFERENTIAL DIAGNOSIS

- SLE (10–30% of women w/ SLE have antiphospholipid antibodies; 60–90% of women w/ APS are ANA (+) but w/ insufficient criteria for Dx of SLE)
- Other causes of thrombocytopenia

© Cambridge University Press