PARVOVIRUS

FLAME LECTURE: 102

STELLER 11.16.18

LEARNING OBJECTIVES

- To discuss the potential impact of Parvovirus on the gravid patient and the fetus/newborn, as well as the impact of pregnancy, and the appropriate initial evaluation
- Prerequisites:
  - NONE

BACKGROUND

- Parvovirus B19 is a small, single-stranded DNA virus that infects ONLY humans
- Characterized by lacy reticular rash *(aka erythema infectiosum, or Fifth disease)*

BACKGROUND

- Self-limited
- Lasts 7-10 days
- Also manifested by fever, HA, malaise → joint pain and swelling
- 20-30% of patients have no symptoms

BACKGROUND

- Transmission occurs most commonly via respiratory droplets
  - More common in late Winter and Spring
  - Can be transmitted in contaminated blood or RhoGam
- Incubation period is 5-10 days after exposure
- By the time the rash is present, the patient is usually no longer contagious

EPIDEMIOLOGY

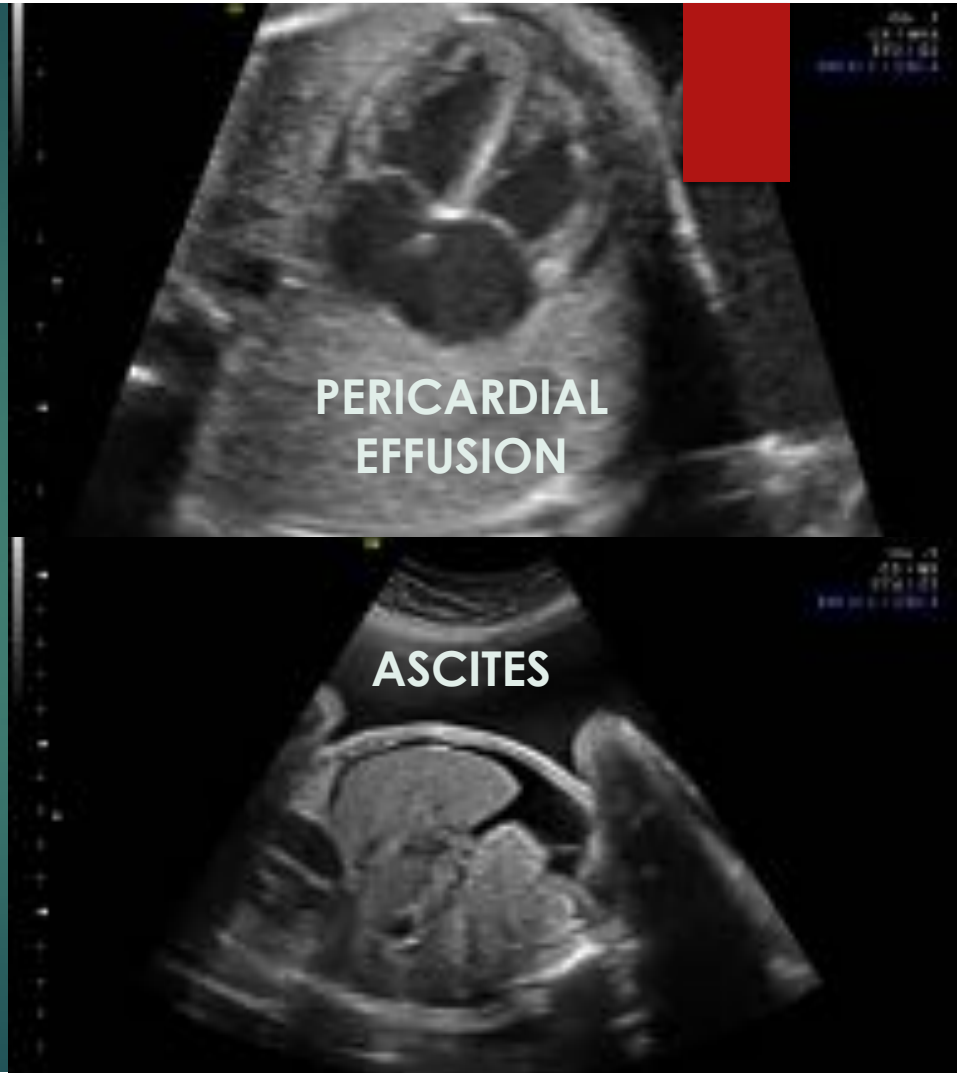
- 50-65% of adults are immune from exposure during childhood
- Primary maternal Parvovirus infection occurs in <1% of pregnancies
- An infected child will infect:
  - 50-80% of household members
  - 20-30% of classroom contacts

PATHOPHYSIOLOGY

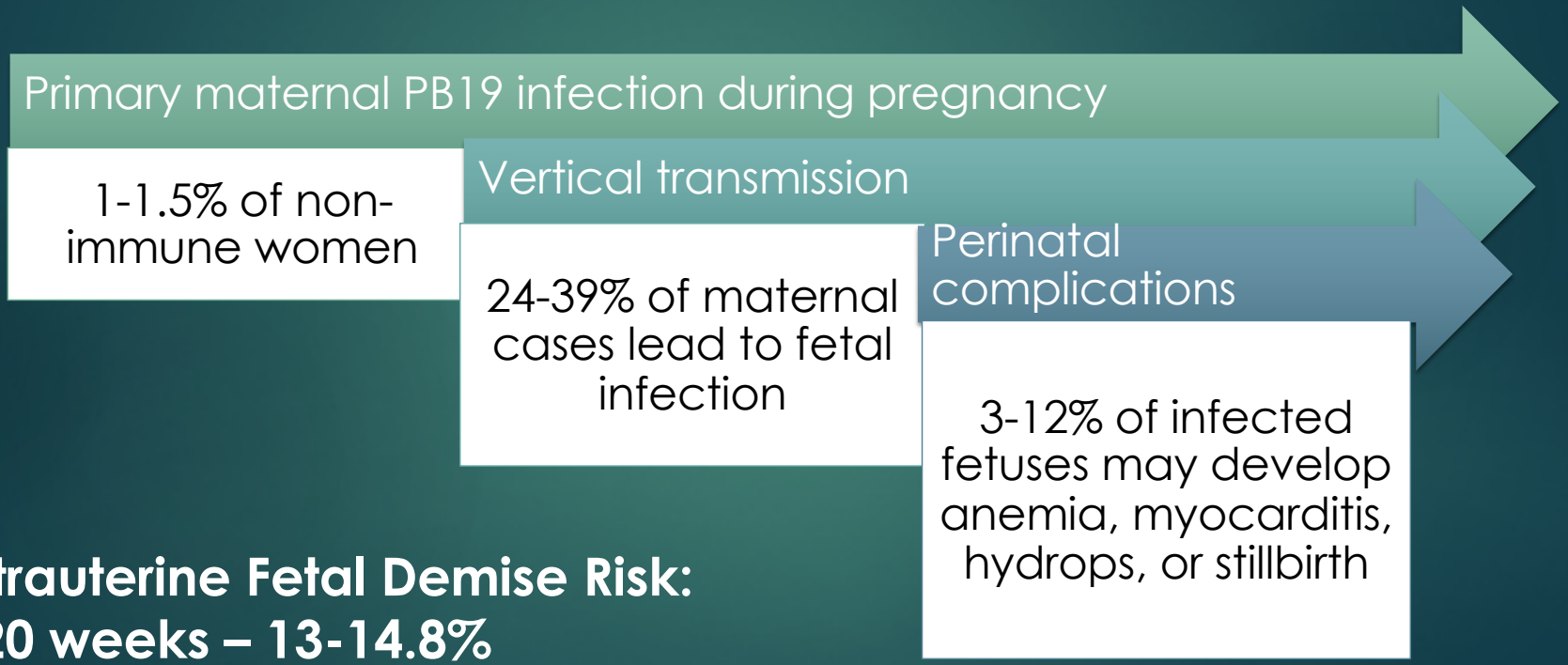
- PB19 preferentially infects rapidly dividing cells and is cytotoxic to **erythroid progenitor cells**
  - Rare cause of transient aplastic crisis in kids/adults with pre-existing hemoglobinopathy or chronic anemia
  - Fetal anemia → hypoxia/high output cardiac failure → impaired venous return → backup in lymph capillaries and veins → increase in interstitial fluid = **hydrops**
  - Can also infect fetal tissues → myocarditis or hepatitis which independently increase fetal interstitial fluid by exacerbating heart failure and impairing protein synthesis in the liver, respectively

BRIEF ASIDE: WHAT IS HYDROPS?

- Abnormal fluid collections in the fetus in >1 compartment including: ascites, pleural effusion, pericardial effusion, or skin/scalp edema
  - Other frequent findings on ultrasound include placental thickening and polyhydramnios (increased amount of amniotic fluid around baby)

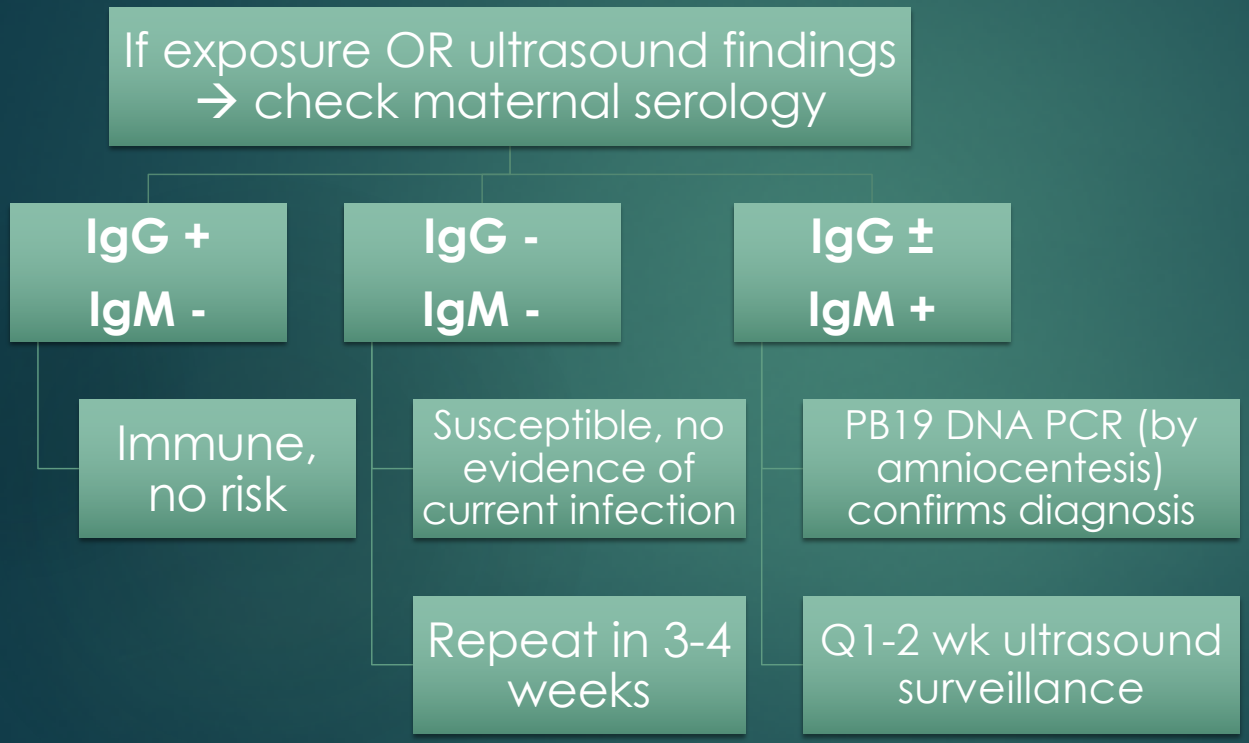


FETAL EPIDEMIOLOGY



Intrauterine Fetal Demise Risk:
<20 weeks – 13-14.8%
>20 weeks – 0.5-2.3%

DIAGNOSIS / MANAGEMENT



- **IgM becomes POS 3-5 days after sx** (or 7 -10 days after infection) and persists for weeks-months
- **IgG becomes POS 7 days after sx** (or 14 days after infection)
- Can also look at B19V DNA in serum/plasma/cord blood/amniotic fluid
  - but this cannot provide time course

FETAL MANAGEMENT

- Hydrops typically develops ~2-6 weeks after seroconversion, but may occur up to 10-12 weeks after
- As a fetus becomes anemic, decreased blood viscosity triggers shunting of blood to critical organs such as the brain
  - Thus, fetal Middle Cerebral Artery (MCA) velocity studies should be monitored via ultrasound q1-2w for 10-12 wks following seroconversion to detect fetal anemia before hydrops develops
- If the MCA peak systolic velocity increases significantly, intrauterine transfusion (IUT) can be considered
  - Frequently only 1 IUT is needed
  - Occasionally RBCs and platelets may need to be transfused
- Limited evidence regarding IVIG (intravenous immunoglobulin)

FETAL MANAGEMENT

- If no signs of hydrops or anemia by 10-12 weeks of exposure, very low likelihood they will develop
- Very few cases of fetal death have been reported in the absence of hydrops
- If compromise noted after 34 weeks → DELIVER
  - Otherwise delivery can be delayed until 37-39 weeks depending upon fetal status
- Long-term neurodevelopmental outcomes in fetus are conflicting
  - With some limited evidence that Parvovirus and/or anemia necessitating IUT may be associated with worse neurologic outcomes

MATERNAL MANAGEMENT

- Frequent handwashing
- Expectant management
  - No medical treatment available
- No vaccine available
- Screen mom for chronic anemia +/- hemoglobinopathy to striate risk of aplastic crisis
- If fetal hydrops develops, monitor mom for mirror syndrome

BRIEF ASIDE: MIRROR SYNDROME?

- Mothers start to mirror their hydropic fetus
  - 80-90% have edema
  - 60% have hypertension
  - 40% have proteinuria
  - 21% have pulmonary edema
- It also mimics pre-eclampsia with signs above and symptoms of headache and visual changes as well
- IUT and/or labor induction are only treatment options

REFERENCES

1. https://www.cdc.gov/parvovirusb19/about-parvovirus.html
2. Feldman et al. 2016. Toxoplasmosis, Parvovirus, and Cytomegalovirus in Pregnancy.
3. Bonvicini et al. 2017. Parvovirus B19 infection in pregnancy-awareness and opportunities.
4. Kobayashi et al 2014. Human parvovirus B19-induced aplastic crisis in adult patients with hereditary spherocytosis: a case report and review of the literature
5. Chauvet et al 2011. Ultrasound diagnosis, management, and prognosis in a consecutive series of 27 cases of fetal hydrops following maternal parvovirus B19 infection.
6. Abbas et al. 2017. Fetal anemia.
7. Sanapo et al. 2016. Fetal anemia, cerebella hemorrhage and hypoplasia associated with congenital Parvovirus infection
8. Isumi et al. 1999. Fetal brain infection with human parvovirus B19
9. Allarakia et al. 2017. Characteristics and management of mirror syndrome: a systematic review (1956-2016).
10. Braun et al. 2009. Mirror syndrome: a systematic review of fetal associated conditions, maternal presentation and perinatal outcome.