437 Team: Obstetrics and Gynecology

Prenatal Infection

Objectives:

- Describe the route of transmission and common complications of perinatal infections including group B beta-hemolytic streptococci, toxoplasmosis, varicella zoster, rubella, cytomegalovirus, HSV, HIV, syphilis, and hepatitis B.

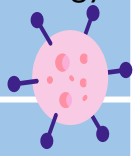
References: Kaplan

Done by: Norah Alkadi, Munira AlHadlag & Rahaf AlShammari

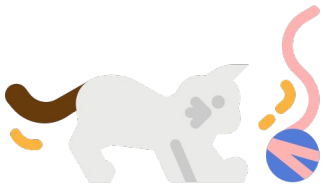
Revised by: Sondos AlHawamdeh

1- NONSEXUALLY TRANSMITTED

GROUP B β-HEMOLYTIC STREPTOCOCCI (GBS)

Pathophysiology 	GBS is a bacterium commonly found in normal GI tract flora. 30% of women have asymptomatic vaginal colonization with GBS, with the majority having intermittent or transient carrier status. Most neonates delivered to colonized mothers will be culture positive.
Significance	One in 500 neonates will develop serious clinical infections or sepsis. • Early onset infection is the most common finding, occurring within a few hours to days of birth, and is characterized by **fulminant pneumonia and sepsis**. This is usually **vertical transmission** from mother to neonate with a 30% mortality rate at or **before 33 weeks** but less than 5% at term. • Late-onset infection is less common, occurring after the **first week of life**, and is characterized by **meningitis**. This is usually hospital acquired, with a 5% mortality rate.
Prevention	The purpose is to decrease early-onset infection only. **Intrapartum** antibiotic prophylaxis of neonatal GBS sepsis is given with **IV penicillin G.** If the patient is penicillin allergic → clindamycin or vancomycin. Candidates for antibiotic prophylaxis are selected as follows: • No screening—**All women with a positive GBS urine culture or a previous baby with GBS sepsis** will receive intrapartum prophylaxis. Prophylaxis of other women is based on either of the following two protocols, each of which will prevent 70% of neonatal sepsis. • Screening by vaginal culture—**Third-trimester vaginal and rectal cultures** are obtained at 35–37 weeks gestational age, and intrapartum prophylaxis is administered **only to those with positive GBS cultures**. Antepartum treatment is not given. • Screening by intrapartum risk factors—No vaginal cultures are obtained. Intrapartum prophylaxis is given on the basis of risk factors being present: **preterm gestation (<37 weeks)**, **membranes ruptured** >18 h, or maternal **fever** (>100.4°F) (**38**°C). Online meded: Antibiotics are NOT needed with C-section if there was NO rom at onset of labor even if positive for GBS.

TOXOPLASMOSIS

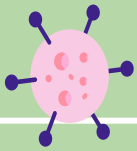


Pathophysiology	Toxoplasmosis is caused by a parasite (Toxoplasma gondii) transmitted most commonly in the United States from exposure to **infected cat feces**. Infections can also occur from **drinking raw goat milk** or **eating raw or undercooked infected meat.** Toxoplasmosis presents as a mononucleosis-like syndrome, but most infections are subclinical. ➢ Vertical transmission from mother to fetus or neonate can only occur during the parasitemia of a primary infection because the result is residual lifelong immunity. ➢ Up to 40% of pregnant women are toxoplasmosis IgG seropositive. ➢ First-trimester infection risk is low (15%), but infections are most serious, even lethal. ➢ **Third-trimester** infection risk is **high** (50%), but infections are mostly **asymptomatic**.
Significance	• Fetal infection—Manifestations may include symmetric IUGR, nonimmune fetal hydrops, microcephaly, and intracranial calcifications. • Neonatal findings—Manifestations may include chorioretinitis, seizures, hepatosplenomegaly, and thrombocytopenia.
Prevention	Avoid contact with cat litter or feces, raw goat milk, and undercooked meat.
Treatment	Pyrimethamine and sulfadiazine are used to treat a known infection. **Spiramycin** is used to prevent vertical transmission from the mother to the fetus.

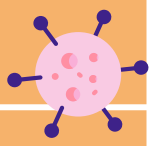
VARICELLA (VZV)

Pathophysiology	Varicella zoster is a DNA virus that is the causative agent of chicken pox and herpes zoster. It is spread by respiratory droplets, but is less contagious than rubeola or rubella. More than 90% of women are immune by adulthood.
Significance	• Fetal infection—Transplacental infection rate is as low as 2% with 25% mortality. • Neonatal findings—Congenital varicella syndrome is characterized by "**zigzag**" skin lesions, mulberry skin spots, optic atrophy, cataracts, chorioretinitis, extremity hypoplasia, and motor and sensory defects. **The greatest neonatal risk is** if maternal rash appears between 5 days antepartum and 2 days postpartum. No passive IgG antibodies are present. • Maternal infection—10% of patients with varicella will develop varicella pneumonia, which has a high maternal morbidity and mortality .Other complications include preterm labor and encephalitis Communicability begins 1–2 days before vesicles appear and lasts until all vesicles are crusted over. Pruritic vesicles begin on the head and neck, progressing to the trunk. The infection can trigger labor.
Prevention	• **Passive immunization:** Administer **VZIG (varicella zoster immune globulin)** to a susceptible gravida within 96 h of exposure. • **Active immunization : Live-attenuated varicella virus (Varivax III)** can be administered to non-pregnant or postpartum to varicella IgG-antibody–negative women.
Treatment	**IV** antiviral treatment with **acyclovir** for varicella pneumonia, encephalitis, or the immunocompromised

RUBELLA (GERMAN MEASLES)

Pathophysiology 	Rubella is a highly contagious RNA virus that is spread by respiratory droplets. Up to 85% of pregnant women are rubella IgG seropositive. • Vertical transmission from mother to fetus or neonate can only occur **during the viremia of a primary infection** because the result is residual lifelong immunity.
Significance	• Fetal infection—Transplacental infection rate is **>90% in the first 10 weeks** of pregnancy, but 5% in the third trimester. Manifestations may include symmetric IUGR, microcephaly, or ventricular septal defect (VSD). • Neonatal infection—Congenital rubella syndrome is characterized by **congenital deafness** (most common sequelae), congenital heart disease, cataracts, mental retardation, hepatosplenomegaly, thrombocytopenia, and "blueberry muffin" rash. • Maternal infection—Rubella infection during pregnancy is generally a mild, low-morbidity condition.
Diagnosis	• Serologic test : -IgM response is rapid . it begins at the onset of the rash and then declines and disappears by 4 to 8 weeks. -IgG response begins at the onset of the rash and remains elevated for life.
Prevention 	➔ All pregnant women should undergo rubella IgG antibody screening because immunity can wane ➔ Rubella-susceptible women should avoid known rubella cases, then receive active immunization after delivery. Because rubella vaccine is made using a live attenuated virus, pregnancy should be avoided for 1 month after immunization.
Treatment	• Rubella is not a contraindication to breastfeeding • No specific treatment. • Rubella has been eradicated from the United States; no cases have been reported here since 2004.

COXSACKIE VIRUS

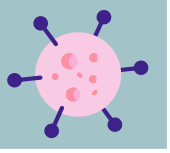
Pathophysiology 	Coxsackie is an enterovirus commonly known as hand, foot and mouth disease (HFMD). It is common, and pregnant women are frequently exposed to it, especially in summer and fall months. Infections are spread by fecal-oral and respiratory routes, with the majority of infections mild or asymptomatic mostly affecting children.
Significance	Fetal infection: Enteroviruses rarely cross the placenta and cause disease in the fetus. There is no evidence of infection causing increased miscarriages, stillbirths, or malformations. Vertical transmission may occur at birth with exposure of the fetus to virus-containing maternal secretions. Neonatal presentation: Newborns who acquire infection from mothers at delivery are at risk of severe disease including sepsis, encephalitis, myocarditis, and pneumonia. Maternal infection: Most enterovirus infections during pregnancy cause mild or no illness in the mother. Clinical findings, when they occur, can include fever, oral vesicles of the mouth and tongue, as well as lesions on the hands and feet. Infection in the third trimester can trigger labor.
Prevention	Avoiding individuals with possible disease. Maintain good handwashing practices and wear a mask if contact with an infected person is unavoidable.

ZIKA VIRUS

Pathophysiology	A mosquito-borne RNA flavivirus. Vertical transmission is transplacental; however, because the virus can persist longer in the serum of a pregnant woman as compared to that of one who is not, the fetus is at risk for infection and major CNS anomalies even if the mother is asymptomatic.
Significance	Fetal infection: The greatest risk of serious perinatal sequelae appears to be with 1st and 2nd trimester infections. Ultrasound abnormalities seen with congenital infections include fetal growth restriction, ventriculomegaly, microcephaly, and intracranial calcifications. Neonatal presentation: Newborn findings other than listed above include ocular abnormalities (e.g. retinal atrophy, microphthalmia), hearing loss, and neurologic abnormalities (e.g. hypertonia, hypotonia, seizures). Maternal infection: Clinical signs consistent with Zika infection are maculopapular rash, arthralgias, conjunctivitis and fever. Only 20% of infected women will have these findings which are often mild. Zika can also be transmitted though sex without a condom with an infected person even if there are no symptoms.
Prevention 	➔ Pregnant women in endemic areas should follow steps to prevent mosquito bites. ➔ Avoid unprotected sex with an infected partner. ➔ Symptomatic or Zika-exposed women should undergo serum and urine nucleic acid test and IgM serology as soon as possible through 12 weeks after. ➔ Positive blood tests should be followed up by prenatal ultrasound and repeated monthly looking for findings listed above.

* Both Coxsackie & Zika have No specific maternal treatment.

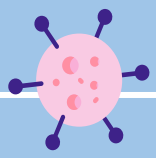
PARVOVIRUS B19

Pathophysiology 	• Parvovirus B-19 is a DNA virus also known as fifth disease. It is a common childhood illness characterized by a “slapped cheek” appearance on the face. • When infection occurs in adults it is most often asymptomatic or mild. It preferentially infects rapidly dividing cells such as RBC precursors and stimulates apoptosis or cell death. • About 50% pregnant woman have protective IgG antibodies. • Vertical transmission is transplacental at the time of primary viremia.
Significance	• Fetal infection: Almost all fetal losses are linked to infections occurring prior to 20 weeks. Parvovirus B-19 is cytotoxic to fetal RBC precursors and may cause fetal anemia and hydrops fetalis. This non-immune hydrops is seen more commonly with infections **prior to 32 weeks.** Transient isolated fetal pleural or pericardial effusions may be seen that resolve spontaneously prior to delivery. The effusions are thought to be due to direct cardiac/pleural inflammation. • Neonatal presentation: While fetal hydrops can occur, most intrauterine parvovirus infections do not have an adverse outcome. There is no evidence of teratogenicity. • Maternal infection: Maternal parvovirus B-19 infections are mild and generally do not include the rash seen in children. Joint pains and fever may occur but the clinical course is usually self-limited.
Prevention 	➔ Pregnant women exposed to or with symptoms of parvovirus infection should have serologic testing for IgG and IgM antibodies. ➔ A positive IgG and negative IgM is consistent with maternal immunity so the fetus is protected. ➔ A positive IgM antibody is consistent with acute infection and should initiate obstetric ultrasound assessment starting at 22 weeks, looking for evidence of fetal hydrops as well as fetal Doppler screening for anemia.
Treatment	Intrauterine transfusion for severe fetal anemia (only intervention available)

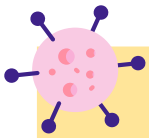
2- SEXUALLY TRANSMITTED

CYTOMEGALOVIRUS (CMV)

Most common congenital viral infection in the United States

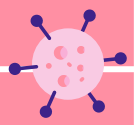
Pathophysiology 	CMV is a DNA herpes virus that is spread by infected body secretions. Up to 50% of pregnant women are CMV IgG seropositive. Vertical transmission from mother to fetus or neonate occurs mainly during the viremia of a primary infection. However, because the result of primary infection is predisposition to a residual lifelong latency, fetal infection can occur with reactivation.
Significance	• Fetal infection—Transplacental infection rate is 50% with maternal primary infections regardless of the pregnancy trimester, but <1% with recurrent infections. Manifestations may include nonimmune hydrops, symmetric IUGR, microcephaly, and cerebral calcifications in a periventricular distribution. • Neonatal infection—From 1 to 2% of newborns have evidence of in utero exposure to CMV. Congenital CMV syndrome is the most common congenital viral syndrome in the United States. **CMV is the most common cause of sensorineural deafness in children.** Only 10% of infected infants have clinical disease, which includes petechiae, mulberry skin spots, meningoencephalitis, periventricular calcifications, hepatosplenomegaly, thrombocytopenia, and jaundice. • Maternal infection—CMV infection during pregnancy is generally a mild, low-morbidity condition appearing as a mononucleosis-like syndrome with hepatitis.
Diagnosis	• Culture: urine culture or by culture of other body secretions or tissues. • Serologic testing: IgM antibodies & IgG antibodies -Problems with serologic testing include: (1) the prolonged elevation in levels of IgM, making delineation of timing of infection difficult (2) a 20% false-negative rate in IgM testing
Prevention	• Follow universal precautions with all body fluids. • Avoid transfusion with CMV-positive blood.
Treatment	- Hyperimmune antiCMV globulin . -Antiviral therapy with **ganciclovir**.

HERPES SIMPLEX VIRUS (HSV)



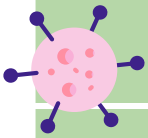
Pathophysiology	Pathophysiology. HSV is a DNA herpes virus that is spread by intimate mucocutaneous contact. Up to 50% of pregnant women are HSV IgG seropositive. • Most genital herpes results from HSV II, but can also occur with HSV I. • Transplacental transmission from mother to fetus can occur with viremia during the primary infection but is rare. HSV infection predisposes to a residual lifelong latency with periodic recurrent attacks. • The most common route of fetal infection is **contact with maternal genital lesions** during a recurrent HSV episode.
Diagnosis	• The definitive diagnosis is a positive HSV culture from fluid obtained from a ruptured vesicle or debrided ulcer, but there is a 20% false-negative rate. • PCR is 2–4x more sensitive and is best to detect viral shedding.
Significance	• Fetal infection—The transplacental infection rate is 50% with maternal primary infections. Manifestations may include spontaneous abortions, symmetric IUGR, microcephaly, and cerebral calcifications. • Neonatal infection—With passage through an HSV-infected birth canal, the neonatal attack rate is 50% with a primary infection, but <5% with a recurrent infection. Neonatal mortality rate is 50%. Neonatal infection can be classified as : (1) disseminated disease with involvement of multiple major organs: pneumonia ,hepatosplenomegaly, and jaundice (2) central nervous system disease with encephalitis: meningoencephalitis , seizures , and mental retardation (3) skin, eye, or mouth infection with localized involvement: petechiae • Maternal infection (2 types): ○ Primary herpes results from a viremia and has systemic manifestations: fever, malaise, adenopathy, and diffuse genital lesions (vagina, cervix, vulva, and urethra). Transplacental fetal infection is possible. However, in 2/3 of cases, the infection is mild or subclinical. ○ Recurrent herpes results from migration of the virus from the dorsal root ganglion but is localized and less severe with no systemic manifestations. Fetal infection results only from passing through a birth canal with lesions present.
Prevention	A cesarean section should be performed in the presence of genital HSV lesions at the time of labor. If membranes have been ruptured >8–12 h, the virus may already have infected the fetus and cesarean delivery would be of no value.
Treatment	Antenatal antiviral prophylaxis (valacyclovir and acyclovir) from 36 weeks until delivery

HUMAN IMMUNODEFICIENCY VIRUS (HIV)

Pathophysiology 	HIV is an **RNA retrovirus** that is spread by infected body secretions. Sharing contaminated needles, having sexual intercourse with an infected partner, and **perinatal** transmission are the most common ways of transmission. The infected patient develops acquired immunodeficiency syndrome (AIDS). The clinical course from HIV to AIDS is a gradual but relentless immunosuppression during a period of years, resulting in death caused by overwhelming infection from opportunistic diseases.
Significance	• Fetal infection—Transplacental infection occurs, but the major route of vertical transmission is **contact with infected genital secretions at the time of vaginal delivery.** Maternal azidothymidine (AZT) prophylaxis, decreased the vertical transmission rate from 30% to 10% with vaginal delivery. With elective cesarean section without labor and before membrane rupture, the perinatal infection rate may be <5%. The greatest benefit to the fetus of **cesarean delivery** is probably in women with **low CD4** counts and **high RNA viral loads**, making infection through a vaginal delivery much more likely. • Neonatal infection—At birth neonates of HIV-positive women will have positive HIV tests from transplacental passive IgG passage. HIV-infected breast milk **can** potentially transmit the disease to the newborn. Progression from HIV to AIDS in infants is **more rapid** than in adults. • Maternal infection—Pregnancy in an HIV-positive woman does **not** enhance progression to AIDS.
Diagnosis	Specific treponemal tests (confirmatory test): such as the fluorescent treponemal antibody absorption test (FTA-ABS)
Prevention 	• Antiviral prophylaxis— to help protect mom's health and prevent passing the infection on to their babies. Infected pregnant women should take **triple-drug therapy** including the drug zidovudine (ZDV) as part of their drug regimen, starting at 14 weeks and continuing throughout pregnancy, intrapartum, and after delivery. • Mode of delivery—Vaginal delivery should be planned at 39 weeks. The guidelines for vaginal delivery are 1) to avoid amniotomy as long as possible, 2) do not use scalp electrodes in labor, 3) avoid forceps or vacuum extractor operative delivery 4) use gentle neonatal resuscitation. Cesarean section is offered at 38 weeks without amniocentesis if viral load is > 1,000 copies/mL. • Breastfeeding—This is probably **best avoided** in HIV-positive women. • Universal precautions—Pay careful attention to handling of all body fluids.
Treatment	All HIV-positive pregnant women should be on **combination triple antiviral HAART therapy.** This includes 2 nucelotide reverse transcriptase inhibitors (NRTIs) with either an NNRTI or a protease inhibitor. An example would be zidovudine, lamivudine, or ritonavir.

SYPHILIS

Pathophysiology



Syphilis is caused by Treponema pallidum, a motile anaerobic spirochete that cannot be cultured. Syphilis does not result in either a state of immunity or latency. The infection can be eradicated by appropriate treatment, but reinfection can occur over and over again. It is spread as a sexually transmitted disease by intimate contact between moist mucous membranes or congenitally through the placenta to a fetus from an infected mother.

Significance

• Fetal infection—Transplacental infection is common with vertical transmission rates of 60% in primary and secondary syphilis. The rate of fetal infection with latent or tertiary syphilis is lower. Without treatment, manifestations of **early congenital syphilis** include nonimmune hydrops, hepatosplenomegaly, profound anemia and thrombocytopenia,macerated skin,osteitis and periostitis, pneumonia, and hepatitis. Fetal death rates are high, with perinatal mortality rates approaching 50%. The placenta is typically large and edematous.

• Neonatal infection—Late congenital syphilis is diagnosed after age 2 years and includes “Hutchinson” teeth, “mulberry” molars, “saber” shins, “saddle” nose, and 8th nerve deafness.

• Maternal infection (4 types):

– Primary syphilis is the first stage after infection. Papules become painless ulcers with rolled edges (chancres) which appear 2–3 weeks after contact at the site of infection, most commonly the vulva, vagina, or cervix. Dark-field microscopy of lesion exudate is positive for the spirochete, but the nonspecific serologic tests VDRL or rapid plasma reagin [RPR] test) are not yet positive. Without treatment the chancre spontaneously disappears.

– Secondary syphilis is characterized by systemic spirochetemia. Two to three months after contact, fever, malaise, general adenopathy, and a maculopapular skin rash (“money spots”) are seen. Broad exophytic excrescences (condyloma lata) appear on the vulva. These physical findings also spontaneously disappear without treatment. Dark-field microscopy of condyloma exudate is positive for treponema. The VDRL or RPR test will be positive, but a diagnosis of syphilis must be confirmed with a treponema-specific test, such as the fluorescent titer antibody absorption (FTA-ABS) or microhemagglutination assay for antibodies to T. pallidum (MHA-TP). The treponema-specific tests do not correlate with disease activity and remain positive in spite of treatment.

– Latent syphilis is characterized by absence of symptoms or physical findings. One third of cases proceed to tertiary disease. The nonspecific and treponema-specific tests remain positive.

– Tertiary syphilis is a symptomatic stage with symptoms dependent on which organ system is affected by the classic necrotic, ulcerative nodules (gummas). Lesion location may include the cardiovascular system (aortitis, saccular aneurysms), CNS (meningitis, tabes dorsalis, dementia, ataxia), or bone (osteitis). Not only are the blood tests positive, but also the cerebrospinal fluid will be positive with CNS involvement.

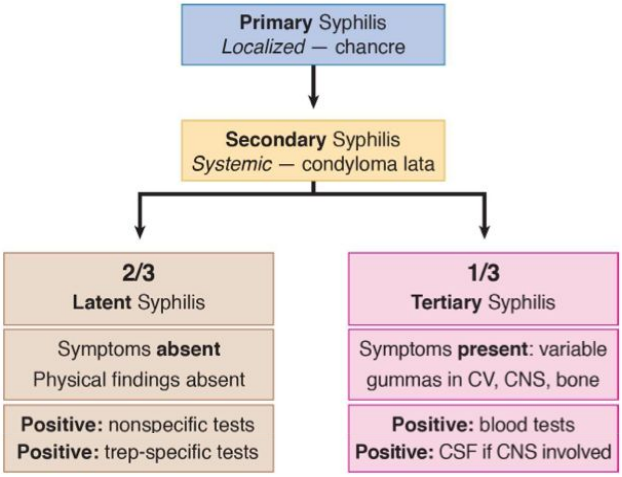


Figure I-7-2. Maternal Syphilis

CNS involvement.

Characteristic	Primary	Secondary
Classic lesion	Chancre	Condyloma lata ("money spots")
Extent of disease	Localized	Systemic
Lab tests (VDRL, Darkfield, FTA-ABS)	VDRL (–) Darkfield (+) FTA-ABS (+)	VDRL (+) Darkfield (+) FTA-ABS (+)
Fetal infection rate	60%	60%
Treatment of choice	Penicillin	Penicillin

Table I-7-1. Syphilis in Pregnancy

Prevention	• Vaginal delivery is appropriate with cesarean section only for obstetric indications. • Follow the principles of avoiding multiple sexual partners, and promote use of barrier contraceptives.
Management	**Benzathine penicillin** 2.4 million units **IM** × 1 is **given in pregnancy** to ensure adequate antibiotic levels in the fetus. Other antibiotics do not cross the placenta well. Even if the gravida is penicillin-allergic, she should still be given a full penicillin dose using an oral desensitization regimen under controlled conditions. Follow serology titers at 1, 3, 6, 12, and 24 months. Titers should be decreased fourfold by 6 months, and should be negative in 12-24 months. The **Jarisch-Herxheimer reaction** is associated with treatment and occurs in half of pregnant women. It starts in 1-2 hours, peaks in 8 hours, and resolves in 24-48 hours. It is associated with acute fever, headache, myalgias, hypotension, and uterine contractions. Management is supportive care.

HEPATITIS B (HBV)

Pathophysiology 	Hepatitis B is a DNA virus that is spread by infected body secretions (blood, saliva, vaginal secretions, semen, and breast milk and across the placenta). Sharing contaminated needles, having sexual intercourse with an infected partner, and **perinatal** transmission are the most common ways of transmission. **Vertical** transmission accounts for 40% of all chronic HBV infections. Most HBV infections are asymptomatic.
Significance	• Fetal infection—Transplacental infection is rare, occurring mostly in the third trimester. The main route of fetal or neonatal infection arises from exposure to or ingestion of infected genital secretions at the time of vaginal delivery. There is no perinatal transmission risk if the mother is positive for HBV surface antibodies but negative for HBV surface antigen. Chronic active hepatitis is associated with an increased risk for prematurity, low birth weight, and neonatal death. • Neonatal infection—Neonatal HBV develops in only 10% of mothers positive for HBsAg but in 80% of those positive for both HBsAg and HBeAg. Of those neonates who get infected, 80% will develop chronic hepatitis, compared with only 10% of infected adults. • Maternal infection (3 types): – Asymptomatic HBV. The majority of all infected patients fall into this category with no impact on maternal health. Hepatitis B surface antigen **(HBsAg) is the screening test** used for identifying existing infection and is obtained on all pregnant women. A positive HBsAg test is followed up with a complete hepatitis panel and liver enzymes assessing for active or chronic hepatitis. – Acute hepatitis. Acute and chronic HBV infections can result in right upper quadrant pain and lethargy varying according to the severity of the infection. Laboratory studies show elevated bilirubin and high liver enzymes. The majority of patients with acute hepatitis will recover normal liver function. – Chronic hepatitis. Cirrhosis and hepatocellular carcinoma are the most serious consequences of chronic hepatitis.
Prevention 	• Vaginal delivery is indicated with cesarean section only for obstetric indications. • Avoid scalp electrodes in labor as well as scalp needles in the nursery. • Neonates of HBsAg-positive mothers should receive passive immunization with hepatitis B immunoglobulin (HBIg) and active immunization with hepatitis B vaccine. Breastfeeding is acceptable after the neonate has received the active immunization and HBIG. • HBsAg-negative mothers at high risk for hepatitis B should receive HBIg passive immunization. Active immunization is safe in pregnancy because the agent is a killed virus.
Treatment	There is no specific therapy for acute hepatitis. Chronic HBV can be treated with interferon or lamivudine.

	Lifelong	Treatment	Delivery
Group β beta streptococcus	Colonization	Penicillin G	Vaginal
Toxoplasmosis	Immunity	Pyrimethamine sulfadiazine	Vaginal
Rubella	Immunity	None	Vaginal
Cytomegalovirus	Latency	Ganciclovir	Vaginal
Varicella/HSV	Latency	Acyclovir	Cesarean section if active HSV
HIV	Latency	Triple Rx antivirals	Cesarean section if high viral load

Table I-7-2 HBV in Pregnancy

	Findings	Findings
Toxoplasmosis[*+]	Intracranial calcifications	Chorioretinitis
Varicella[+]	Zig zag lesions	Small eyes
Rubella[*+]	Deafness	Congenital heart disease
Cytomegalovirus[*+]	Petechiae	Enlarged liver, spleen
Syphilis[+]	Hydrops	Macerated skin
HSV, HIV, HBV[Δ]	None	

[*]Associated with IUGR
[+]Transplacental vertical transmission
[Δ]Vaginal delivery vertical transmission

Table I-7-3 Key Phrases in Perinatal Infections

OB Triads

Congenital Toxoplasma
- Chorioretinitis
- Intracranial calcifications
- Symmetrical IUGR

Congenital Varicella
- "Zig-zag" skin lesions
- Microphthalmia
- Extremity hypoplasia

Congenital Rubella
- Congenital deafness
- Congenital cataracts
- Congenital heart disease

Cytomegalovirus
- Most common congenital viral syndrome
- Most common cause of deafness in children
- Neonatal thrombocytopenia and petechiae

EXTRA Cases

GBS	A 20-year-old woman G2 P1 is admitted to the birthing unit at 35 weeks' gestation in active labor at 6-cm dilation. Her prenatal course was unremarkable with the exception of a positive first-trimester urine culture for GBS. Her first baby was hospitalized for 10 days after delivery for GBS pneumonia.
Toxoplasmosis	A 26-year-old primigravida was admitted to the birthing unit at 39 weeks' gestation in active labor at 6-cm dilation. During her second trimester she experienced a mononucleosis-like syndrome. Uterine fundal growth lagged behind that expected on the basis of a first-trimester sonogram. Serial sonograms showed symmetrical intrauterine growth retardation (IUGR). She delivered a 2,250-g male neonate who was diagnosed with microcephaly, intracranial calcifications, and chorioretinitis.

Varicella	A 29-year-old woman (G2 P1) is at 34 weeks' gestation. She complains of uterine contractions every 5 min. During the last few days she has developed diffuse pruritic vesicles on her neck that appear to be also developing on her chest and breasts. She has a fever and complains of malaise.
Rubella	An 18-year-old primigravida is at 30 weeks' gestation and is employed in a childcare center. One of the children had a rash that was diagnosed as rubella. The patient's rubella IgG titer is negative. She is concerned about the possibility of her fetus getting infected with rubella.
CMV	A 31-year-old neonatal intensive care unit nurse has just undergone an uncomplicated term spontaneous vaginal delivery of a 2,300-g female neonate with a diffuse petechial rash. At 12 weeks' gestation she experienced a flulike syndrome with right upper quadrant pain. Obstetric sonograms showed fetal growth was only at the fifth percentile.
HSV	A 21-year-old multipara was admitted to the birthing unit at 39 weeks' gestation in active labor at 6-cm dilation. The bag of water is intact. She has a history of genital herpes preceding the pregnancy. Her last outbreak was 8 weeks ago. She now complains of pain and pruritis. On examination she had localized, painful, ulcerative lesions on her right vaginal wall.
HIV	A 22-year-old multigravida is a former IV drug user. She was diagnosed as HIV positive 12 months ago during her previous pregnancy. She underwent vaginal delivery of an infant who is also HIV positive. She is now pregnant again at 15 weeks' gestation.
Syphilis	A 34-year-old multigravida presents for prenatal care in the second trimester. She admits to a past history of substance abuse but states she has been clean for 6 months. With her second pregnancy, she experienced a preterm delivery at 34 weeks' gestation of a male neonate who died within the first day of life. She states that at delivery the baby was swollen with skin lesions and that the placenta was very large. She was treated with antibiotics but she does not remember the name or other details. On a routine prenatal panel with this current pregnancy she is found to have a positive VDRL (Venereal Disease Research Laboratory) test.
HBV	A 29-year-old multigravida was found on routine prenatal laboratory testing to be positive for hepatitis B surface antigen. She is an intensive care unit nurse. She received 2 units of packed red blood cells 2 years ago after experiencing postpartum hemorrhage with her last pregnancy.