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Torch Infection In Pregnancy

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Abstract
TORCH infections (Toxoplasmosis, Other [Syphilis, Varicella-Zoster, Parvovirus B19], Rubella, Cytomegalovirus, and Herpes Simplex Virus) are a group of infections that have the potential to cause serious complications in pregnancy, including miscarriage, premature delivery, and congenital abnormalities in the fetus. Early detection and appropriate management are key to preventing neonatal morbidity and mortality. The purpose of this study is to review the characteristics of TORCH infections, their impact on mothers and fetuses, and effective prevention and management strategies during pregnancy. The research method used was a literature review examining various scientific sources related to the pathophysiology, clinical manifestations, diagnosis, and management of TORCH infections in pregnant women. The results of the study show that TORCH infections can cause various complications depending on the timing of exposure during pregnancy. Toxoplasmosis carries a risk of hydrocephalus and intracranial calcification, rubella can cause congenital rubella syndrome, while cytomegalovirus is the main cause of congenital hearing loss. Management includes the administration of antiviral drugs or antibiotics according to the causative pathogen, as well as supportive measures to prevent vertical transmission. The implications of this study emphasize the importance of prevention, early detection, and appropriate management to minimize the risk of complications in mothers and fetuses. Improved health education for pregnant women and routine laboratory monitoring are expected to improve pregnancy outcomes in at-risk populations.
Keywords: *TORCH infections, pregnancy, congenital complications, early detection, prenatal management*

1. INTRODUCTION

TORCH infections, which consist of Toxoplasmosis, Other (including Syphilis, Varicella-Zoster, Parvovirus B19, and others), Rubella, Cytomegalovirus (CMV), and Herpes Simplex Virus (HSV), are a group of infections that have the potential for serious complications in pregnancy [1]. Transmission of infection from mother to fetus usually occurs transplacentally, especially when infection occurs in the first or second trimester, which is a critical period for fetal organogenesis. The clinical impact of TORCH infection varies widely, ranging from miscarriage and premature birth to congenital abnormalities such as microcephaly, hydrocephalus, developmental delay, visual impairment, and hearing loss. The complexity of these clinical manifestations underscores the importance of early detection, accurate diagnosis, and appropriate intervention to minimize neonatal morbidity and mortality.

Epidemiologically, the distribution of TORCH infections is influenced by various factors, including geographic and socioeconomic conditions, and public awareness of prevention. For example, toxoplasmosis infections are more common in areas with poor hygiene or consumption of unsafely cooked food. While rubella has decreased in incidence in countries with strong immunization programs, it remains a threat in areas with low vaccination coverage. Cytomegalovirus is the most common infection in pregnant women and tends to be asymptomatic, often going undetected, despite the risk of congenital hearing loss and developmental delays in the fetus. Meanwhile, HSV and syphilis remain global health problems, particularly in areas with a high prevalence of sexually transmitted infections, emphasizing the need for a preventive approach and intensive risk education.

Early detection of TORCH infection is key to preventing fetal complications. Serological testing and other diagnostic methods such as PCR and fetal imaging help identify infections early, allowing for prompt intervention. Preventive measures also include education on hygiene, safe food consumption, and recommended vaccinations, particularly for rubella. These timely interventions are crucial to reduce the risk of maternal-fetal transmission and long-term complications in infants, including neurological and sensory impairments.

Research on TORCH infections during pregnancy is highly relevant for understanding risk factors, prevalence, and clinical impact. This information helps healthcare professionals design more effective screening, management, and prevention strategies, particularly for high-risk pregnant women. A good

understanding of the epidemiology and transmission pathways of TORCH infections also aids in the development of appropriate clinical protocols, decisions regarding antiviral or antibiotic therapy, and safe management of pregnancy and delivery for both mother and fetus. The results of this research are expected to inform prevention efforts, improve antenatal care services, and develop more effective maternal and neonatal health policies.

2. METHOD

This research employed a qualitative approach with a literature review method. The study was conducted by reviewing various relevant scientific sources, covering discussions on the pathophysiology, diagnosis, and management of TORCH infections in pregnancy. Data sources included scientific journal articles, medical textbooks, research reports, and official publications issued by national and international health organizations. Literature selection was based on relevance to the research topic and the credibility of the sources, ensuring the data obtained could provide a strong scientific basis.

The analysis process was conducted descriptively through the stages of identification, categorization, and synthesis of key findings from each literature. Based on the review, a summary was compiled that comprehensively describes the mechanisms of TORCH infection in pregnant women, available early detection procedures, and appropriate management strategies. This research is expected to contribute to a deeper understanding of the importance of preventing and managing TORCH infections in pregnancy.

3. RESULT AND DISCUSSION

Pregnancy is a crucial period, so the health of both mother and fetus requires special attention. One health condition that can seriously impact pregnancy is TORCH infection, which includes toxoplasmosis, other infections (such as syphilis, varicella-zoster, parvovirus B19, and hepatitis B), rubella virus, cytomegalovirus (CMV), and herpes simplex virus (HSV) [1]. Further discussion regarding this disorder will be explained in the following sub-chapter.

3.1Toxoplasmosis in Pregnancy

Fetal toxoplasmosis infection occurs primarily through placental transmission of tachyzoites following primary maternal infection. Transmission can occur during the parasitemic phase before the development of a maternal serological response or after trophoblast infection. The mechanism is complex and incompletely understood. Tachyzoites invade the brain and muscle, forming tissue cysts within a short time in immunocompetent animals, although the duration of this infection in fetuses with immature immune systems is unknown. Once the fetus is infected, placental transfer of maternal IgG is insufficient to prevent sequelae [2].

The primary source of maternal infection is consumption of undercooked meat containing bradyzoites, as well as soil, water, fruit, or vegetables contaminated with sporozoites. Other less common but still risky sources include unpasteurized goat's milk, consumption of raw shellfish or oysters, and organ transplantation or blood transfusion. The risk of transmission to the fetus is greater if the mother is infected with sporozoites from cat oocysts than if she is infected with bradyzoites from meat. Acute maternal infection is usually asymptomatic, although a small proportion of pregnant women may experience fever, lymphadenopathy, or ocular abnormalities such as chorioretinitis [2].

The severity of congenital infection is greatly influenced by genotype Toxoplasma gondii. In Europe and North America, the less virulent types 2 and 3 are dominant, while in South America and Africa, the more virulent types are found, increasing the risk and severity of infection. The risk of miscarriage in pregnancies with seroconversion is estimated at around 0.5% and fetal death at 1.3–1.6%. Most babies are born asymptomatic, with only 10–30% showing clinical signs such as chorioretinitis, hydrocephalus, or intracranial calcifications [2]. However, babies with mild or subclinical infections remain at risk of long-term complications, particularly chorioretinitis, which can lead to visual impairment into adulthood, along with the possibility of other complications such as neurological dysfunction, hearing loss, developmental delays, and endocrine disorders [3].

The diagnosis of toxoplasmosis in pregnancy is generally confirmed by serological testing if there is clinical suspicion, such as fever, maternal lymphadenopathy, or fetal sonographic findings of intracranial calcifications and ventricular dilatation. Congenital infection is suspected in infants of mothers with primary infection. Toxoplasma gondii during pregnancy, in immunosuppressed mothers, or in infants with typical signs such as chorioretinitis and cerebrospinal fluid abnormalities. Supporting tests may

include maternal serology and PCR.T. gondii on CSF, neuroimaging with CT or MRI, as well as ophthalmologic evaluation and hearing testing using auditory brainstem response (ABR) [2], [3].
Congenital diagnosis can be confirmed by detecting specific IgM or IgA. Toxoplasma and confirmation of parasite DNA by PCR. Infection is confirmed by a combination of positive antibody, rising IgG titers in the first year of life, or persistent IgG titers until one year of age. Conversely, negative serology results or a gradual decline in IgG without the appearance of IgM/IgA excludes infection [2], [3].
Regarding prenatal screening, serological testing should be tailored to regional conditions. In areas with low prevalence and less virulent strains, such as Western Europe, the benefits of screening may not outweigh the risks and costs. However, in areas with high prevalence and more virulent strains, such as South America, screening is preferred. Screening results should be interpreted with caution, as IgM can persist for a long time and produce false-positive results, while IgG remains detectable long-term. Detection of new infection is most strongly demonstrated by IgG and IgM seroconversion on repeat testing [2]. IgG avidity testing can help differentiate between old and new infections, with high avidity indicating chronic infection (≥4 months) and low avidity suggesting a possible new infection. Additional testing such as IgG/IgM Western blot or evaluation of repeated IgG titer rises can improve accuracy [2].
Treatment for toxoplasmosis in pregnant women should begin immediately upon detection of a possible maternal infection, even before the fetal diagnosis is confirmed by amniocentesis. Early therapy is important because there are window period therapeutic, namely the period before Toxoplasma gondii transition from the tissue-damaging tachyzoite form to the dormant bradyzoite cyst, which is unresponsive to antibiotics. Administration of antibiotics within the first three weeks after maternal seroconversion can prevent or reduce neurologic damage to the fetus, so therapy should not be delayed even if the pregnancy is approaching 18 weeks (Petersen E & Mandelbrot L, 2024).
The treatment regimen is selected based on gestational age at diagnosis. Spiramycin is recommended in the first trimester (<14 weeks) due to its relative safety, while the pyrimethamine-sulfadiazine combination is more effective when given at ≥14 weeks of gestation. Once the PCR amniocentesis result is known, therapy can be modified; if positive, pyrimethamine-sulfadiazine is continued until delivery, while if negative, the treatment decision can be adjusted based on the risk of onward transmission. The dose of spiramycin is generally 1 g three times daily, while pyrimethamine-sulfadiazine is given in stepped doses and requires regular blood monitoring due to the risk of bone marrow suppression. Temporary alternatives such as cotrimoxazole can be used if the primary drug is unavailable (Petersen E & Mandelbrot L, 2024).
In infants with congenital toxoplasmosis, combination therapy with pyrimethamine, sulfadiazine, and folinic acid is given for up to one year, with some centers extending the course to two years. Glucocorticoids, such as prednisolone, are given if CSF protein is high or chorioretinitis is active. Cohort studies have shown that long-term therapy improves neurologic, cognitive, hearing, and visual outcomes compared with infants who are untreated or receive only short-term therapy. The main side effect of pyrimethamine is neutropenia, while sulfadiazine can cause exanthema and bone marrow suppression; monitoring of CBC and liver and kidney function is recommended during therapy [3].
Prenatal care for pregnant women with toxoplasmosis infection is routine, with no specific indications for method or timing of delivery. Fetal ultrasound monitoring is performed monthly to detect abnormalities such as ventricular dilation or hydrocephalus, while postnatal placental examination is not recommended as a diagnostic method due to its low sensitivity. Primary infection prevention focuses on avoiding sources of the parasite, such as consumption of raw or undercooked meat, raw shellfish, unfiltered water, and contact with cat feces, especially from wild animals [2].
The prognosis for infants without treatment is generally poor, with a mortality rate of approximately 12% and serious complications including intellectual disability, seizures, paralysis, and visual impairment. Early and long-term treatment can reduce the risk of neurological and visual complications, although recurrence is possible because drugs are effective only against tachyzoites, not bradyzoites that lie dormant in the eye and brain tissue. Risk factors for severe complications include severe central nervous system infection, extensive brain calcification, untreated hydrocephalus, and delays in diagnosis and treatment. Chorioretinitis can recur in up to one-third of patients between the ages of 3 and 13 years, although further visual impairment is rare [4].

3.2Rubella in Pregnancy

Rubella, or German measles, is a childhood disease whose incidence has declined dramatically in the United States since routine vaccination was introduced. Although usually mild, rubella infection in

pregnant women can have serious consequences for the fetus, thus routine rubella antibody screening is recommended. The rubella virus infects only humans and is transmitted through droplets from nasopharyngeal secretions, spreads through the bloodstream, and can cross the placenta during maternal viremia, causing congenital infection [5].

Congenital rubella infection (CRI) encompasses a spectrum of intrauterine infections ranging from miscarriage and stillbirth to birth defects or asymptomatic infection, while congenital rubella syndrome (CRS) is characterized by birth defects such as sensorineural hearing loss, congenital heart disease, cataracts, and congenital glaucoma. The risk of congenital infection depends on the gestational age at infection, with the highest risk occurring in the first 10 weeks. Infection before 8 weeks typically results in heart and eye defects, while hearing loss can appear as late as 18 weeks [6].

Rubella pathogenesis involves inhibition of cell division, resulting in smaller fetal organ size and reduced cell number, as well as direct cytopathic effects through apoptosis in various cells such as chorionic villi explants and adult lung fibroblasts. Viral persistence in CRS may be influenced by impaired cellular immunity and tolerance to the rubella E1 protein, while primary fetal fibroblasts survive apoptosis, allowing the virus to persist for a long time in the fetus.[7]

The immune response to rubella infection in the fetus and infant includes humoral, cellular, and potentially autoimmune responses. During the first 16–20 weeks of pregnancy, only 5–10% of maternal IgG is transferred to the fetus, while the fetus begins producing IgM, IgG, and IgA at 9–11 weeks of gestation at low levels, with IgM predominating. In infants with congenital rubella infection (CRI), IgM persists for at least six months to two years, while IgG may persist for several years before declining.

Some infants develop hypergammaglobulinemia or hypogammaglobulinemia due to chronic infection. Children with CRI, whose antibody levels are reduced, do not respond to vaccination due to immunological tolerance to the rubella E1 protein. Cellular responses are also impaired, including T-cell abnormalities, decreased cytotoxicity, and interferon production, increasing the risk of autoimmunity. CRI is associated with an increased risk of type 1 diabetes and thyroid disease, although a direct link to rubella has not been proven (Arrieta AC, 2024).

Acquired rubella is usually mild, with an incubation period of 14–21 days, and approximately 25–50% of cases are asymptomatic. Prodromal symptoms include fever, conjunctivitis, coryza, sore throat, cough, headache, malaise, and suboccipital, postauricular, and cervical lymphadenopathy. A maculopapular rash appears on the face, spreads to the trunk and extremities, and resolves within 1–3 days. Polyarthritis or polyarthralgia may occur one week after the rash, particularly in adolescent girls and adult women. Rare complications include thromboembolism, myocarditis, hepatitis, hemolytic anemia, and hemolytic uremic syndrome [5].

Congenital rubella infection (CRI) and congenital rubella syndrome (CRS) can cause spontaneous abortion, stillbirth, or fetal growth restriction. The risk of birth defects is highest if infection occurs within the first 16 weeks of pregnancy, decreases in the second trimester, and is rare after 20 weeks, although it can cause intrauterine growth retardation. The virus spreads through the placenta and fetal vascular system, causing blood vessel damage and organ ischemia. Maternal immunity from vaccination or prior infection generally protects the fetus, although reinfection can cause some cases of CRS, especially if it occurs before 12 weeks of pregnancy [5], [6].

Diagnosing rubella infection, whether acquired or congenital, involves serologic testing, viral culture, and PCR. Serologic testing typically uses an ELISA to detect rubella-specific IgM or IgG antibodies. In acute infection, a fourfold increase in IgG titer between acute and convalescent serum, detection of IgM, or a positive viral culture can establish the diagnosis. However, a positive IgM in asymptomatic pregnant women can be false, so IgG avidity testing can help distinguish recent infection from cross-reactivity. Postnatal diagnosis of CRI is made by detection of rubella-specific IgM or IgG that outlasts maternal antibodies, most effectively in infants under two months of age, with repeat testing recommended in doubtful cases. Monitoring of rubella-specific IgG at 3, 6, and 12 months of age can confirm congenital infection, although this method is less sensitive in infants with impaired antibody production [6].

Rubella virus isolation by culture can be performed from the pharynx, blood, urine, placenta, or cerebrospinal fluid, with the pharynx being the primary source. In some cases, the virus can persist for years in lens tissue or cerebrospinal fluid, particularly in children with eye disorders or epilepsy. PCR is an effective adjunct method for detecting rubella virus RNA in a variety of specimens, including pharyngeal swabs, cerebrospinal fluid, urine, amniotic fluid, products of conception, or lens tissue, although its availability is limited [6].

For prenatal diagnosis, chorionic villi PCR (CVPCR) is more sensitive than amniotic fluid, allowing earlier detection at 10–12 weeks of gestation compared to 18–20 weeks in fetal blood. Although ultrasound can be used to evaluate fetal growth, the characteristic malformations of CRS are not always obvious, so laboratory testing remains the primary method [5].

Management of rubella infection is supportive, including acetaminophen for symptomatic relief and glucocorticoids or platelet transfusions for complications such as thrombocytopenia or encephalopathy. In pregnant women, the maternal prognosis is generally good, but the serious risks to the fetus warrant counseling regarding maternal-fetal transmission and the option of termination of pregnancy before 16 weeks. After 20 weeks, management is individualized, as no in utero or antiviral therapy is effective in altering the clinical course or fetal viral excretion [5], [6].

The clinical outcome of congenital rubella infection (CRI) varies; most infants survive, but perinatal death can occur in cases of prematurity or severe disease. Long-term effects of CRS include progressive hearing loss, congenital heart defects, retinopathy, stunted growth, and an increased risk of diabetes and thyroid disorders in adulthood, as demonstrated by an Australian cohort study of patients born during the 1939–1943 rubella epidemic [6].

Rubella prevention is primarily through vaccination. Preconception vaccination with MMR is given to susceptible children, adolescents, and adults, with the recommendation to delay pregnancy 28 days after vaccination. Although the vaccine virus can cross the placenta, no cases of CRS associated with vaccination have been reported. Postpartum vaccination is also recommended for seronegative mothers, including those receiving anti-D immunoglobulin, with rubella immunity testing performed three months after vaccination [5]. Post-exposure management includes preventing further exposure, CRS risk counseling, and serologic monitoring, as immunoglobulin is not recommended for routine prophylaxis. Transmission prevention involves droplet precautions, particularly in infants with CRS/CRI, who are contagious for up to one year, and ensuring that caregivers or caregivers are immune to rubella [6].

3.3Cytomegalovirus in Pregnancy

Cytomegalovirus (CMV) is a DNA herpesvirus that can remain latent after primary infection and undergo reactivation, with the possibility of secondary infection via different routes [7]. In immunocompetent individuals, infection is u sually mild or asymptomatic, but in immunocompromised individuals, including transplant recipients or HIV patients, CMV can cause high morbidity and mortality. In pregnant women, infection is often asymptomatic but carries a risk of congenital CMV in the infant, which is the most common congenital viral infection with a birth prevalence of approximately 0.67%. Approximately 90% of infants born with congenital infection are asymptomatic, but both symptomatic and asymptomatic infants are at risk for complications, particularly sensorineural hearing loss [8], [9].

CMV infection is spread through contact with saliva, urine, genital fluids, blood, breast milk, or tissue from an infected individual. The risk of vertical transmission is highest when seroconversion occurs during pregnancy, with the frequency of fetal infection increasing from 5.5% preconceptionally to 66.2% in the third trimester. However, the likelihood of clinical symptoms in the infant decreases with advancing gestation, with the risk of clinical symptoms highest periconceptionally (28.8%) and declining dramatically in the second and third trimesters. Seroimmunity before pregnancy provides partial protection against fetal infection, but most cases of congenital CMV result from nonprimary maternal infection, except in HIV-infected mothers with advanced immunodeficiency [7].

CMV seroprevalence is high globally, approximately 86% in women of childbearing age, and increases with demographic, social, behavioral, and environmental factors, such as contact with young children, high parity, age over 25–30 years, residence in high-prevalence areas, and economic and sanitary conditions. Infection can occur through various routes, including sexual contact, blood transfusion, transplantation, contact with children in daycare, as well as transplacental transmission, delivery, and breastfeeding. Premature or low-birth-weight infants are at higher risk of complications, and the virus can persist on moist surfaces, facilitating environmental spread [7].

Cytomegalovirus (CMV) infection can occur in either primary or non-primary forms. After primary infection, CMV can remain latent and potentially reactivate or reinfect with a new strain, although natural immunity does not completely prevent transmission [8]. Secondary infection often occurs in seropositive women within a few years after delivery, characterized by new antibodies to CMV-specific determinants. Reactivation can occur at any time, particularly in individuals with immunosuppression, including those with glucocorticoid use or diseases such as AIDS, although CMV-specific IgG titers do

not always predict this risk. T-cell responses, both CD4+ and CD8+, play a crucial role in controlling viral replication, although they are unable to completely eliminate CMV from the body.

In pregnancy, primary CMV infection occurs in previously seronegative women, with diagnosis confirmed by seroconversion of CMV-specific antibodies. Non-primary infection occurs in women who already had CMV IgG antibodies before pregnancy, either through reactivation of latent virus or reinfection with a different strain. Reactivation or reinfection during pregnancy can cause transient viremia in the mother and increase the risk of transmission to the fetus [7].

Most primary infections in pregnant women are asymptomatic (approximately 90%), but 10% of cases can cause mild symptoms such as fever, rhinitis, pharyngitis, muscle and joint pain, headache, and fatigue, resembling infectious mononucleosis. Non-primary infections or reactivation in mothers who already have antibodies are usually asymptomatic. The severity of infection is influenced by the condition of the mother's immune system, with immunocompromised individuals being more susceptible to severe and widespread infections [7].

CMV serological screening for pregnant women will not be universally recommended until 2023, although some advocate targeted screening for high-risk women, such as those with frequent contact with young children. In 2024, the European Society of Clinical Virology recommended serological screening in the first trimester for seronegative women, with repeat testing every 4 weeks until 14–16 weeks of gestation. This screening can help seronegative pregnant women practice better hygiene and allow for antiviral intervention in primary infections to reduce the risk of transmission to the fetus.

The diagnosis of CMV infection in pregnant women is suspected if symptoms such as a mononucleosis- or influenza-like syndrome are present, especially if Epstein-Barr and influenza tests are negative, or if signs of hepatitis are present. Typical fetal ultrasound findings, such as periventricular calcifications or ventriculomegaly, also raise suspicion. The diagnosis of primary infection is confirmed by CMV-specific IgG seroconversion within three to four weeks, while a combination of IgG, IgM, and IgG avidity tests helps distinguish recent from past infections. IgM alone is not always reliable, as it can remain positive for a long time or appear due to reactivation (Boppana SB & Hui L, 2024).

Fetal CMV infection can be asymptomatic or cause prenatal manifestations. Amniocentesis is recommended to confirm infection if maternal serology or ultrasound findings are suspicious. Amniotic fluid samples obtained at least eight weeks after the mother's primary infection increase the test's sensitivity (up to 95.8%) and specificity (100%). A positive result confirms congenital infection, while a negative result rules out the possibility. Close fetal monitoring with ultrasound and brain MRI is recommended if infection is confirmed, although some infants remain at risk for hearing loss or developmental delays [7], [9].

Serial ultrasound examinations every 2–4 weeks are important to detect abnormalities that arise after maternal infection, including periventricular calcifications, ventriculomegaly, microcephaly, fetal growth restriction, hydrops, and changes in organs and the placenta. A normal ultrasound is reassuring but does not completely rule out the risk of postnatal neurologic or hearing impairment. Abnormal findings indicate a high risk of severe disease, especially in first-trimester infections, while third-trimester infections rarely cause hearing loss [7], [10].

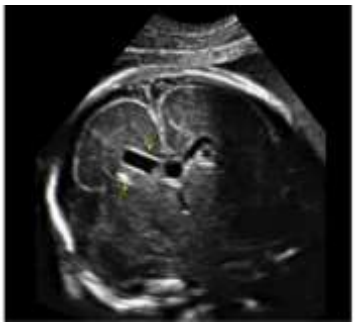

Figure 1. Fetal brain ultrasound shows ventriculomegaly and bilateral periventricular hyperechogenicity (calcifications) [7].

Furthermore, additional tests such as amniotic fluid viral load, MRI, and cordocentesis are not routinely recommended. Viral load can help predict symptomatic disease, but a reliable threshold has not been widely validated. MRI is useful if ultrasound suspects an abnormality, but its predictive value for neurodevelopmental outcomes is limited. Cordocentesis carries an approximately 2% risk of miscarriage

and does not add significantly to the prediction of long-term outcomes compared to an abnormal ultrasound. Liver function tests, hematology, and beta-2 microglobulin are useful only for detecting severe disease but are not more predictive than ultrasound.

Therefore, screening and diagnosis of CMV in pregnant women should be tailored to the risks, symptoms, and ultrasound findings. Amniocentesis remains the gold standard for confirming fetal infection if strongly indicated, while serial ultrasound monitoring and MRI evaluation are used to assess fetal development. These measures help guide prenatal interventions and plan postnatal care, although some complications, particularly hearing loss, can still occur despite normal imaging findings.

Most primary CMV infections in immunocompetent individuals are mild or asymptomatic, so antiviral therapy is usually unnecessary. However, in primary infections early in pregnancy, high-dose valacyclovir may be considered to reduce the risk of vertical transmission to the fetus. Other antiviral agents, such as ganciclovir, foscarnet, and cidofovir, have generally been studied in immunocompromised patients, and their effectiveness in the immunocompetent population remains unclear. The decision to administer antivirals to immunocompetent pregnant women should consider the severity of the infection and the risk of drug toxicity [8].

For pregnant women with symptomatic CMV infection, management is supportive, such as administering acetaminophen to reduce fever. Counseling and psychosocial support are important to reduce anxiety caused by the uncertainty of CMV infection. Referral to a maternal-fetal medicine specialist is recommended if necessary, given the limited experience with antiviral use during pregnancy.

High-dose valacyclovir (8 g/day) is recommended for primary CMV infection in early pregnancy after a discussion of the benefits and risks. This medication can reduce the risk of vertical transmission and neonatal infection, especially if started early and evaluated by amniocentesis at least eight weeks after primary infection. Serious side effects, such as reversible renal failure, can occur, requiring close monitoring. This therapy can also delay the maternal immune response, so consideration should be given to the interval between subsequent pregnancies.

Randomized trials and meta-analyses have shown that valacyclovir effectively reduces vertical transmission, neonatal infection, and termination of pregnancy due to severe CMV-related fetal abnormalities. Therapy can also be used as tertiary prevention in already infected fetuses, increasing the proportion of asymptomatic babies, although it does not affect other perinatal outcomes such as death or neurological impairment. Adherence to therapy is high despite the high dose requirement, and long-term follow-up of infants is needed to assess the full effect [7], [11].

CMV-specific hyperimmune immunoglobulin therapy is not recommended for pregnant women with primary infection, as randomized trials have shown it to be ineffective in reducing congenital infection, death, preterm birth, or developmental delays. Observational studies are promising but require further evaluation in controlled trials.

During labor, standard obstetric indications should be followed, and the presence of CMV in the cervix or urine is not a reason for cesarean delivery. Infants at high risk for congenital CMV infection should be examined within the first 21 days of life, including a hearing and vision evaluation. Breast milk is still recommended for full-term infants, while pasteurization or freezing is recommended for premature or sick infants to reduce the risk of transmission.

Prevention of CMV infection during pregnancy is primarily achieved through behavioral interventions and personal hygiene. Education about hygiene has been shown to reduce the risk of seroconversion in pregnant women, including washing hands after contact with a child's bodily fluids, avoiding sharing food or utensils, and not kissing a child on the lips. Healthcare workers are not at higher risk than the general public, likely due to adherence to standard precautions.

Women who have recently recovered from primary CMV infection are advised to delay pregnancy for at least 3–6 months to reduce the risk of congenital infection, although evidence-based guidelines are lacking. Furthermore, blood transfusions for seronegative individuals, including pregnant women, fetuses, or newborns, should use CMV-seronegative blood or leuko reduced blood products to prevent transmission

3.4Herpes Simplex in Pregnancy

Neonatal herpes simplex virus (HSV) infection has a variable incidence globally, estimated at 3–30 cases per 100,000 live births, with approximately 14,000 cases per year globally. In the United States, the annual incidence is estimated at 8–10 per 100,000 live births [12], [13].

Maternal genital HSV infections are classified into three types based on immune status and infection history: primary infection (first episode without HSV-1 or HSV-2 antibodies), first-episode non-primary infection (first lesion in an individual who already has antibodies to a different HSV), and recurrent infection (lesion of the same HSV type with pre-existing antibodies). Determining the type of infection is especially important during pregnancy because primary or non-primary infection near delivery increases the risk of neonatal transmission, while recurrent infection carries a lower risk [13].

The clinical manifestations of HSV infection vary. Primary infections are usually more severe, characterized by painful genital ulcers, fever, inguinal lymphadenopathy, and systemic symptoms, although many patients are asymptomatic. Non-primary infections tend to be milder, while recurrent infections are usually preceded by prodromal symptoms and mild lesions. Intrauterine infection due to primary maternal infection is rare but can lead to serious complications, including fetal death, hydrops fetalis, and central nervous system damage [12].

Neonatal HSV infections are classified into three categories: skin, eye, and mouth (SEM) disease; central nervous system (CNS) disease; and disseminated disease. SEM is usually associated with HSV-1, CNS is more often associated with HSV-2, and disseminated disease involves multiple systems and has the worst prognosis. CNS disease appears between the second and sixth weeks of life, with symptoms such as seizures, lethargy, and difficulty feeding [12].

Disseminated disease occurs in 25–30% of cases, mimicking sepsis with liver involvement, ascites, and hyperbilirubinemia. Infants may exhibit hypothermia, apnea, abdominal distension, and hepatomegaly. Rapid diagnosis and prompt antiviral therapy are crucial to reduce the high morbidity and mortality associated with neonatal HSV infection [12].

Risk factors for perinatal transmission include the type of maternal HSV infection, maternal antibody status, duration of rupture of membranes, use of fetal scalp monitoring, and mode of delivery. Approximately 10% of neonatal infections are acquired postnatally through direct contact with an individual with active HSV, and infants born to mothers without a history of infection are at higher risk because they do not receive specific antibodies transplacentally [12], [13].

The diagnosis of genital herpes simplex (HSV) infection in pregnant women is generally based on clinical findings of vesicular or ulcerative lesions, but laboratory confirmation is still necessary. Diagnostic methods include PCR, viral culture, direct fluorescent antibody testing, and type-specific serology [13]. In pregnant women without a history of genital HSV, active genital ulcers should be tested by PCR from the vesicle or ulcer base, along with type-specific serologic testing to determine whether the maternal infection is primary, non-primary, or recurrent. If the initial result is negative but clinical suspicion is high, serology should be repeated after 3–4 weeks to detect seroconversion [13].

Women with a history of laboratory-confirmed HSV do not require additional testing. However, if the previous history is based solely on symptoms without laboratory confirmation, active genital ulcers should still be tested with PCR to confirm recurrent infection. PCR is more sensitive than viral culture in detecting recurrent infection. Serology may be used if the viral test is negative but clinical suspicion remains.

Rapid PCR testing to detect asymptomatic HSV shedding during labor is not routinely performed because its benefit in determining delivery method and reducing the risk of neonatal infection is unclear.

Vertical transmission of HSV in neonates generally occurs during delivery through direct contact with the virus from the mother's genital area. The risk is highest in primary infections near delivery, lower in first-time non-primary infections, and very small in recurrent infections. Viral shedding can be asymptomatic, with HSV-2 more frequently causing central nervous system (CNS) infections, while HSV-1 is more frequently associated with skin disease. Intrauterine infection is rare but can lead to miscarriage, congenital abnormalities, premature birth, or fetal growth retardation [13].

Neonatal HSV is classified into intrauterine (5%), perinatal (85%), and postnatal (10%) infections. The perinatal form is most common, occurring during delivery through contact with the virus in the genital tract. Postnatal infection is usually acquired through direct contact with a symptomatic individual and is more severe in infants without transplacental antibodies. Since 2010, HSV-1 has become the more common cause of neonatal HSV, with presentations of skin, eye, and mouth (SEM) disease. Early detection and treatment have reduced the neonatal HSV mortality rate from 20% to 5% (Demmler-Harrison et al., 2024).

Management of pregnant women with genital herpes infection aims to reduce the risk of vertical transmission and control maternal symptoms. Management strategies include suppressive antiviral

therapy starting at 36 weeks' gestation and consideration of cesarean section in selected cases. This approach is tailored to the type of infection (primary, non-primary first episode, or recurrent), the severity of symptoms, and the timing of infection relative to delivery, as recommended by ACOG [13].

In primary infections or non-primary first episodes, empiric antiviral therapy is given while awaiting virologic test results. Oral acyclovir (400 mg three times daily for 7–10 days) is the preferred treatment, with extensions if lesions persist. In severe cases or organ involvement, intravenous acyclovir (5–10 mg/kg every 8 hours) is used. Symptomatic therapy, such as acetaminophen and sitz baths, helps relieve pain and fever.

Suppressive therapy is initiated at 36 weeks' gestation until delivery for all pregnant women with genital herpes lesions, whether primary, non-primary, or recurrent. This approach reduces recurrence during labor, decreases the need for cesarean delivery, and reduces viral shedding. Women with a history of symptomatic recurrence during pregnancy benefit most, while seropositive women without active lesions are not recommended to receive suppressive therapy.

Most recurrent infections are mild and do not require intervention. Episodic or suppressive antiviral therapy is usually avoided before 36 weeks to reduce unnecessary drug exposure, except in cases with severe symptoms or frequent relapses.

Acyclovir has the most extensive clinical experience during pregnancy and is considered safe in all trimesters, for both acute and suppressive therapy. Valacyclovir may be an alternative due to the convenience of twice-daily dosing, although safety and efficacy data are more limited. Famciclovir is less recommended due to the limited data in pregnancy. Furthermore, additional monitoring, such as weekly genital cultures or serial PCR, is not recommended because it does not predict viral shedding during labor. Maternal HSV infection is also not an indication for special antepartum fetal monitoring, as the fetus and placenta are generally uninfected.

Antepartum obstetric procedures in women with genital herpes infection should consider the risk of viral transmission. Transcervical procedures such as cerclage or chorionic villus sampling should be avoided if active lesions are present, but may be performed in asymptomatic patients. Transabdominal procedures such as amniocentesis and fetal blood sampling are not contraindicated even if the mother has active genital lesions.

The route of delivery is determined based on the presence of active lesions or prodromal symptoms. According to CDC and ACOG recommendations, cesarean delivery is recommended immediately after the onset of labor or rupture of membranes in women with active genital lesions, including crusted lesions, or prodromal symptoms such as pain and burning. If the membranes have been ruptured for more than six hours, cesarean delivery may still be considered, although its benefit is less clear (Riley & Wald, 2008).

In primary or non-primary first-episode infections, virus shedding may persist for a prolonged period, and maternal antibodies may not yet have developed. Therefore, cesarean delivery is recommended if infection occurs in the final weeks of pregnancy, although the final decision still depends on the patient's preference. Expert groups, including the Royal College of Obstetricians and ACOG, emphasize cesarean delivery for first-term infections in the third trimester, especially if symptoms appear within the six weeks leading up to delivery (Riley & Wald, 2008).

Women with a history of recurrent HSV without active lesions or prodromal symptoms have a very low risk of neonatal transmission, so cesarean delivery is unnecessary. The same applies to nongenital lesions; the risk of transmission is low, and lesions can be covered with an occlusive dressing during labor. The use of fetal scalp electrodes should be avoided as they may increase the risk of HSV transmission to the infant. External fetal heart rate monitoring is preferred as long as it provides adequate information.

Additionally, other invasive procedures, such as vacuum or forceps delivery, should be minimized to reduce fetal skin abrasion and the risk of neonatal infection. A minimally invasive vaginal delivery approach is recommended to reduce the risk of HSV transmission (Riley & Wald, 2008).

Postpartum and neonatal care emphasize preventing transmission of herpes simplex virus (HSV) from parents or caregivers to infants. Adults with active lesions should cover the lesions and wash their hands before touching the infant, as approximately 5–15% of neonatal herpes cases are acquired postnatally through contact with an infected family member. Breastfeeding is permitted as long as there are no herpetic lesions on the breast, and the use of acyclovir or valacyclovir during breastfeeding is considered safe. It is important for parents to communicate with their pediatrician about possible neonatal exposure and to monitor the infant for signs of HSV infection.

Serologic screening can be used to identify women with a history of HSV to determine the need for suppressive antiviral therapy and to evaluate lesions at delivery. However, universal screening is not recommended because the accuracy of commercial antibody tests is limited, and HSV does not meet the criteria for effective prevention strategies like HIV or hepatitis B. If screening is performed, a type-specific glycoprotein G (gG)-based test is recommended to differentiate HSV-1 from HSV-2, with additional confirmation if a low index result (1.1–3.5) indicates a possible false-positive [13].

Pregnancy management is tailored to the partner's serologic status. If both partners are seronegative, routine treatment is sufficient. If the woman is seronegative for HSV-2 but the partner is seropositive, condom use is recommended during the first and second trimesters, along with abstinence from sexual intercourse in the third trimester. If the woman is seronegative for HSV-1 and the partner is seropositive, sexual and oral-genital contact should be avoided in the third trimester to reduce the risk of neonatal infection.

Suppressive therapy in infected male partners using valacyclovir 500 mg/day can reduce the risk of HSV-2 transmission by up to 48% and is likely effective against HSV-1. However, evidence of the effectiveness of suppressive therapy during pregnancy or in same-sex couples is limited.

Conversely, women seropositive for HSV-1 or HSV-2 without a history of genital lesions are not recommended to receive suppressive therapy because there is no evidence to support its use. This strategy emphasizes a risk-based approach, good communication with healthcare providers, and neonatal monitoring to ensure early detection and appropriate management of HSV infection [13].

4. CONCLUSION

TORCH infections during pregnancy are a health problem that can seriously impact both the mother and the fetus. Various studies have shown that these infections can cause complications such as miscarriage, premature birth, and congenital abnormalities that can potentially affect the quality of life of the unborn child. Key risk factors include a lack of awareness of prevention, low immunization coverage, and limited access to serological testing, which plays a crucial role in early detection.

Preventive measures, such as rubella vaccination, hygiene education to prevent toxoplasmosis, and screening for sexually transmitted infections such as syphilis and herpes simplex, are crucial in reducing the incidence of TORCH infections. Furthermore, optimal pregnancy monitoring with serological testing can facilitate early detection and appropriate management to prevent more severe complications. With increased understanding of the epidemiology, transmission mechanisms, and prevention and management strategies for TORCH infections, it is hoped that the incidence and adverse effects of these infections can be minimized.

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