



Review Article

A review of maternal TORCH-S infections

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ABSTRACT

TORCH-S is a medical acronym for a set of perinatal infections with known adverse impact on fetal developmental and pregnancy outcome. This includes infections with *Toxoplasma gondii*, Rubella virus, Cytomegalovirus, Herpes simplex virus (1 and 2) and *Treponema pallidum* (Syphilis). TORCH-S infections group of prenatal illnesses that have been linked to adverse outcomes in fetal development and pregnancy. Infections caused by TORCH-S can affect anybody, including children, men, and non-pregnant women. However, because they may be passed to the embryo while it is still in the womb, major fetal problems can arise if a mother is exposed during the first 5 months of pregnancy. The cornerstone of congenital infection prevention is the primary prevention of maternal infections during pregnancy. Early identification of TORCH-S infection will help in appropriate treatment and management of these infections.

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1. Introduction

TORCH-S Syndrome refers to infections in neonates caused by the TORCH-S infective agents that cross the placenta while pregnancy. The medical acronym of TORCH-S refers to *Toxoplasma gondii*, Rubella virus, Cytomegalovirus, Herpes Simplex, and syphilis. If the developing fetus is affected by the TORCH-S infective agent, miscarriage, abortion, delayed fetal development and maturation (delayed development within the uterus) or premature delivery may be the consequences of pregnancy. Newborns exposed to any of the TORCH-S agents may have a variety of symptoms and results. It included inattention, fever, difficulty feeding, liver and spleen (hepatomegaly), and decreased levels of the oxygen-carrying pigment in the blood. Infected neonate may also have bleeding, which manifests as red or purple blotches (purpura). Jaundice is a yellow coloring of the skin and eyes; chorioretinitis

is an inflammation of the middle and inner layers of the eyes. It's depending on the stage of fetal development at the time of infection. Each infectious agent may induce further defects of varying degrees and severity. The TORCH agents are described in further detail below. Toxoplasmosis is an infection caused by *Toxoplasma gondii*, a tiny parasitic parasite. Toxoplasmosis can cause microcephaly, inflammation of the middle and deepest layers of the eyes, intracranial calcifications.¹ Fever, upper respiratory infection, lymph node enlargement, skin rash, and joint discomfort are all symptoms of rubella virus infection.² Cytomegalovirus (CMV) can arise during pregnancy, after delivery, or at any age. Growth retardation and other signs and findings in severely afflicted babies are possible.³ Neonatal Herpes is an uncommon condition that affects neonates who have been infected by the Herpes virus (HSV1 and HSV2). This condition can range from moderate to severe. In the majority of instances, the condition is passed down to a newborn from a woman who is sick and has active vaginal sores at the time of birth.⁴ Congenital syphilis is

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spread by *Treponema pallidum* subspecies *pallidum* through sexual contact or vertical transmission during pregnancy.⁵

2. Discussion

2.1. *Toxoplasma gondii*

This is an obligate intracellular protozoan parasite that is a real public health threat. This parasite belongs to the Apicomplexa phylum, Coccidiasina subclass, and Sarcocystidae family.⁶

The duration of illness ranges from 3 to 25 days (with a mean of 11 days).⁷ More than 90% of pregnant mothers who contract a primary infection during their pregnancy are asymptomatic,⁸ and the infection manifests as intrauterine growth retardation, spontaneous abortion, stillbirth, preterm deliveries, and fetal anomalies such as ocular damage, hydrocephalus with mental retardation, and stillbirth that is visible only in the third trimester or after delivery.⁹ As a result, early diagnosis of toxoplasmosis infection is critical to initiate proper treatment in time to decrease transplacental spread.

Congenital toxoplasmosis develops when a pregnant woman gets infected with *Toxoplasma gondii*. Tachyzoites circulating inside the mother's bloodstream may infiltrate and proliferate in the placenta, infecting the fetus. In prenatal parasite infection may result in congenital abnormalities or spontaneous miscarriage.^{10,11}

This parasite has a sexual and asexual life cycle. The sexual cycle takes place exclusively in the infected cat, which serves as the only host.¹² The parasite's infectious stages may assume three distinct forms: sporozoites, tachyzoites, and bradyzoites.¹³ Tachyzoites are also the parasite's rapid reproducing stage, which is spread throughout the body through the bloodstream and infects several organs. The fast multiplication and release of tachyzoites from host cells produce tissue damage and severe inflammatory response and is therefore responsible for illness clinical symptoms.¹⁴

Bradyzoites are parasites that reside in muscle, brain, and other tissues and infect animals for the remainder of their lives.¹⁵ Gametogony causes oocyst development in the gut of the ultimate host (the cat) (sexual reproduction). The adult oocyst includes two sporocysts, each with four infective sporozoites.¹⁶

The cysts may survive for up to 18 months in water or warm wet soils.¹⁷ The clinical signs and symptoms of infection during pregnancy vary depending on the number of parasites, strain pathogenicity, and the mother's age.¹⁸ During the first trimester, infection causes miscarriage, stillbirth, or severe fetal illness. The illness severity reduces with infection in the second or third trimester, but the chance of transmission increases.¹⁹

Lethargy, enlarged lymph nodes, and headache are common symptoms.⁷ The most frequent clinical symptom

of human toxoplasmosis is enlarged lymph nodes.²⁰ The infection is transplacental and may affect the eyes or nervous system. The bacteria may develop tissue cysts in the brain or muscle. However, if the mother has toxoplasmosis later in pregnancy, the baby is more likely to be affected.²¹

When a pregnant woman is infected with a disease, her immune system produces IgM antibodies first, then IgG antibody. IgM antibody to TORCH-S organisms last approximately three months, while IgG antibody last for a life, giving protection and preventing the severity of reinfection. If a pregnant woman has IgM antibody, she is likely to have had a recent or current infection with the pathogen.²² Placenta, serum, and cerebrospinal fluid may be used to identify the causal organism.²³ Using polymerase chain reaction (PCR) amplification of the *Toxoplasma gondii* B1 gene, diagnostic testing for the pathogenic organism in an infant whose mother has acute infection symptoms can begin as early as 18 weeks of pregnancy. To determine the causal organism, specific diagnostic procedures such as differential scanning calorimetric, IgM ELISA, IgM agglutination assay, and anti-P30 IgM were used for estimation antibody against *Toxoplasma gondii*.²⁴ The computed tomography scan of the head may identify calcifications. During toxoplasmosis, the cerebrospinal fluid shows a rise in protein levels as well as pleocytosis.²³ Toxoplasmosis infection is also indicated by rising IgG and IgM antibody levels in the cord or newborn serum.

Prevent *Toxoplasma gondii* from spreading horizontally via food to human's raw or undercooked meat. *Toxoplasma gondii* is also killed by freezing meat at -20°C/4°F. To avoid cross-contamination, wash hands and utensils thoroughly after handling raw meat or vegetables. Before eating raw fruits and vegetables, make sure they're clean.²⁵ Meat should never be eaten while it is being prepared or uncooked.²⁶

Toxoplasmosis treated by spiramycin for preventing intrauterine toxoplasmosis infection and combination of pyrimethamine, folinic acid and sulfadiazine used for fetal toxoplasmosis infection.²⁷

2.2. *R- Rubella virus*

Rubella is a togaviridae virus. Rubella, sometimes called German measles, is a virus that causes a maculopapular rash, lymphadenopathy, and fever. It's a highly infectious yet relatively harmless illness with little side effects. Rubella virus infections can cause during the first trimester of the pregnancy the rubella virus infection malformation called congenital rubella syndrome (CRS). The virus is made up of single-stranded RNA. It enters the cell through the endocytic route. For 7-9 days, viral replication occurs in limited regions of the nasopharynx and regional lymph nodes, followed by viremic dissemination to numerous sites throughout the body. The incubation time ranges from 14 to 21 days.²⁸ The methods through which the

rubella virus causes prenatal harm are unknown. The virus travels via the circulation before the maternal immune response develops and may damage various maternal tissues, including the placenta. Damaged tissue examination indicates two potential pathways for rubella cytopathology and direct cytopathic impact, which may include rubella virus induced death, and a virus induced suppression of cell division.²⁹ Vascular damage and subsequent insufficiency have a larger role in the etiology of CRS (Congenital Rubella Syndrome).³⁰ Placental infection causes focally scattered necrotic areas in the chorionic villae epithelium and endothelial cells of its capillaries during maternal viremia. These cells must be desquamated into vessel lumens, implying that rubella virus is transmitted into the fetal circulation as infected endothelial cell emboli, posing a risk of infection and organ damage. Organs that have been harmed are typically hypoplastic.³¹

Rubella is a contagious disease transmitted via the respiratory system.³² It is disseminated by direct contact with virus-containing.³³

At the time of infection, pregnant women feel different types of symptoms, malaise, UTI (urinary tract infection) fever, lymphadenopathy, early rash, and respiratory problems.³⁴ Pregnant women with rubella are most infectious while the rash is erupting, and infectivity is from seven days before to fourteen days after the rash erupts.³⁵

Infections can be diagnosed using a variety of methods using body fluids, serum used to detect the presence of specific IgM and IgG. Virus from amniotic fluid can be detected using real-time PCR technique.³⁶

In women 28 days before pregnancy, vaccination is the greatest strategy to avoid Rubella virus infection. Although a vaccination is not suggested for pregnant women, it is possible to vaccinate lactating mothers. Women who are not immune to rubella should avoid contact with affected people.³⁴

2.3. C- Cytomegalovirus

Human Cytomegalovirus (CMV) is a herpesviridae that infects humans. In healthy children and adults, most CMV infections are asymptomatic. However, CMV causes severe disease in immunocompromised individuals and fetuses. Congenital CMV infection is the most common non-genetic cause of sensorineural hearing loss in children, as well as a significant contributor to neurodevelopmental delays.^{37,38}

Most infections are asymptomatic, but once infected; the virus may reactivate later in life when the body's protection is weakened.³⁹ Clinical manifestation of CMV infection includes fever, weakness, fatigue, and flu-like,⁴⁰ Most instances are symptomatic, although serious fetal injury and death related to miscarriage are uncommon.⁴¹

CMV infection is passed on to a baby via the mother's milk, through direct contact with urine and saliva, and other

means such as breastfeeding. It is readily transmitted in daycare facilities and households with a large number of little children. It is possible for transplant recipients who are immunosuppressed to develop a severe disease as a result of endogenous reactivation of the virus.²⁴

In order to identify this virus, bodily fluids such as urine and pharyngeal discharge must be used in the first three weeks following birth. After 3 weeks of birth, distinguishing between congenital and postnatal infection will be challenging.³⁴ This virus is often detected using the PCR method. Postnatal seizures are more common in pregnant women who have congenital CMV infections. Postnatal seizures were found to occur 10 to 56% of the time in infants with symptomatic congenital CMV infection, but only 0.9% of the time in pregnant women with asymptomatic congenital CMV infection.⁴²

Primary CMV infections are extremely dangerous to one's health during pregnancy. As a result, increased attention should be placed on women of reproductive age, including pre-pregnancy CMV screening programmes, limiting contact with CMV-infected children during pregnancy, and appropriate sexual behaviours. In order to reduce the risk of abortion, a successful CMV immunisation regimen should be completed.⁴³

Patient symptomatic CMV infection treated using by valganciclovir and ganciclovir. Neonatal CMV symptomatic disease associated with CNS (Central Nervous System) treated with valganciclovir (Oral suspension) continues for six months, dosage changed as neonates grows.⁴⁴

2.4. H- Herpes Simplex Virus

Around 70% of infants with HSV infection are given to moms who have never had genital sores.⁴⁵ Even though the maternal infection could be asymptomatic, most neonatal cases are caused by maternal HSV infection all time of delivery, as defined by IgM production. About a third of asymptomatic women with HSV detected in early labor have the primary genital disease, and their newborns are 10% significantly more likely to occur neonatal HSV than babies of asymptomatic reactivated women. Women with primary genital herpes, who are infected with HSV-1 and HSV-2 in the utter lack of prior HSV antibodies, and those with a non-primary disease, who have a first diagnosable genital infection, generally with HSV-2, in the existence of neutralizing HSV IgG antibodies, usually to HSV-1, have been distinguished. Non-primary prenatal infections were formerly believed to be less likely to cause newborn infection. Infection occurred in infants delivered vaginally to asymptomatic mothers who were shown to be shedding HSV by the viral culture at delivery.⁴⁶

Symptoms include ulcers on the genital and oral mucosa. Systemic symptoms such as headache, fever, and myalgia may accompany primary infection in women. Ulcerative rash and systemic symptoms, including malaise,

dysuria, and lymphadenopathy may also accompany primary infection in women. The asymptomatic infection affects about half of all women who have the primary infection. Symptoms such as vulvovaginitis and cervicitis are experienced by about 20% of moms. Approximately 30% of patients appear with vesicular and ulcerated vaginal lesions that are typical of the condition.⁴⁷

The most frequent cause of oral herpes is Herpes simplex virus 1, while the most common cause of genital herpes is Herpes simplex virus 2 and is nearly always transmitted sexually.⁴⁸ Even though the risk of transmission seems to be highest during lesions recurrences the placenta serves as a vital barrier between the maternal and fetal circulations. As a result, transplacental viral transmission is likely to affect the placenta.⁴⁹

Urine, saliva, and nasopharyngeal secretions can be tested for diagnosis. Positive HSV IgM serum, CSF PCR, or lesion culture results indicate infection. After 5 days, CSF PCR may be negative. Viral culture can detect skin, eye, and mouth infections in 24–36 hours.

Preventive strategies such as frequent prenatal examinations, encouraging a healthy lifestyle, and avoiding numerous sex relations should be prioritized in our daily lives.⁴⁶ Reduced disease-related risks will come from a detailed sexual history, increased early detection of HSV infections, screenings, as well as appropriate disease counseling and education. Genital herpes occurs late in pregnancy, the easiest way to avoid prenatal herpes illnesses is to minimize viral exposure to a neonate, while the incidence of serious neonatal infection is low in repeated episodes. At the initial prenatal appointment, all expectant mothers and their partners should have a diagnosis of HSV infection.⁵⁰

2.5. S- Syphilis (*Treponema pallidum*)

The bacteria that causes congenital syphilis, *Treponema pallidum*, is a tiny, motile spirochete that enters the body through direct sexual contact scratched skin or mucous membranes. Infants can contract syphilis through transplacental transmission from a mother who infected with syphilis at any stage of pregnancy; transmission rates for primary and secondary syphilis are estimated to be 60 to 100%, 40 to 83% for early latent syphilis, and 10% for late latent syphilis. Congenital Syphilis is classified as an early or late illness depending on whether symptoms show before or after the age of two. The signs and symptoms of early Congenital Syphilis are many and diverse. Most children with early congenital syphilis are asymptomatic at birth and appear in the neonatal period during the first 4 to 8 weeks of life. Hepatomegaly with or without splenomegaly, rash, rhinitis, lymphadenopathy, pneumonia, anemia, thrombocytopenia, osteochondritis, and pseudoparalysis are reported in 10% to 40% of infants; pseudo The mucous illness is extremely contagious

through direct contact, thus chronic rhinitis or snuffles” is frequently the initial presenting symptom.⁵¹ Other nonspecific findings include hepatomegaly even without splenomegaly, widespread non-tender lymphadenopathy, and rash. Skeletal abnormalities are observed in 60 to 80% of newborns and maybe the only symptom of congenital Syphilis, with long bones being the most often affected. Affected infants may develop osteochondritis a painful restriction of mobility that mimics paralysis. Pneumonia, anemia, thrombocytopenia, and nonspecific anomalies in the cerebrospinal fluid (CSF) are all possible clinical findings.⁵²

Syphilis is transferred from mother to child, and the children are diagnosed with primary and secondary disease stages but rather tertiary stage syphilis. Early disease (before the age of two years) and later disease (beyond the age of two years) are the two stages of congenital syphilis (after two years).⁵³

Hemorrhagic nasal discharge, jaundice, hepatosplenomegaly, elevated liver enzymes, hemolytic anemia, lymphadenopathy, thrombocytopenia, osteochondritis, mucocutaneous dermatitis, and central nervous system abnormalities are some of the early symptoms.³⁴

Because non-treponemal tests have a high false-positive rate, confirmatory testing is usually done with treponemal tests, such as the *Treponema pallidum* haemagglutination assay (TPHA), *Treponema pallidum* particle agglutination test (TPPA), and also the fluorescent treponemal antibody absorption test (FTA-ABS). Treponemal assays use a *Treponema pallidum* specific assay to detect treponema-specific antibodies. Sensitivities for these tests vary from 70% to 100%, with specificities ranging from 94% to 100%. False positives may occur in individuals who have had their syphilis completely treated in the past or who have had their syphilis recently treated. Treponemal tests aren't very helpful for screening or evaluating the efficacy of therapy. Pregnant women with newly positive treponemal tests or an RPR or VDRL titer of 1:8, particularly in high-prevalence regions with limited access to additional testing, should be treated.^{54,55}

There is currently no vaccine available to prevent syphilis, and the most effective method of prophylaxis is the early treatment to prevent the illness from being transmitted sexually and vertically from mother to child, as well as treatment of all sex couples to prevent re-infection. Latex condoms, male circumcision, and avoiding intercourse with infected partners are among more ways to reduce venereal syphilis transmission. To prevent re-infection, exposed sex partners must be treated.⁵⁵

Syphilis should be diagnosed and treated immediately in pregnant mothers. Suspected pregnant mothers should be tested for syphilis during pregnancy and take appropriate treatment, benzathine penicillin G (600,000U/Kg)

intramuscular injection used for treating syphilis.⁵⁶

3. Conclusion

Pregnant women should take prenatal check-ups and close monitoring because for these infections prevention is preferable. Primary prevention strategies are screening for Toxoplasmosis, Rubella virus, CMV, HSV1 & 2, and syphilis in the first prenatal appointment. The health authorities should arrange campaigns for pregnant women and women of childbearing age for the understanding of these infections, mode of transmission, risks, and health complications. Suspects should take an improved serological test to diagnose active TORCH-S infective agents like IgM ELISA, IgG avidity, and PCR assays.

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None.

5. Conflict of Interest


None.

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