CE 1.5 HOURS Continuing Education

# Congenital Syphilis

## *A Discussion of Epidemiology, Diagnosis, Management, and Nurses' Role in Early Identification and Treatment*

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**ABSTRACT**

**Background:** Syphilis is caused by the spirochete bacterium *Treponema pallidum*. Syphilis left untreated, or inadequately treated during pregnancy, can result in congenital syphilis (CS). Congenital syphilis can lead to severe sequelae or fetal, neonatal, or infant death.

**Purpose:** To discuss the epidemiological trends, pathophysiology, diagnosis, and management of CS; the implications of CS upon the infant; as well as the importance of the nurse's role in the prompt identification of CS and the timely interventions needed to minimize sequelae.

**Methods:** A literature search was completed using ProQuest, CINAHL, Google Scholar, and PubMed. Articles published within the past 10 years were included.

**Findings:** Epidemiological trends of CS in the United States indicate that maternal syphilis infection and CS are on the rise. Risk factors include ethnicity, socioeconomic status, access to prenatal care, and sexual behaviors, as well as compliance with prenatal syphilis screening by prenatal providers. Risks of CS to the developing fetus begin at approximately 14 weeks. Timely treatment is necessary to minimize or eliminate mortality and morbidity.

**Implications for Practice:** Evidence-based, interprofessional strategies, which promote a collaborative perinatal/neonatal preventative approach to care of the pregnant female, are indicated to reverse the increasing incidence of CS within the United States. Strategies prioritizing early identification and treatment of at-risk neonates are necessary to reduce/eliminate the devastating long-term consequences of CS upon this vulnerable population.

**Implications for Research:** The paucity of research, which focuses on CS, is most likely due to ethical concerns related to infants as research participants and provides an opportunity for future research. Future research could focus on factors that focus on maternal–fetal/maternal–child transmission of CS.

**Key Words:** community health, congenital infections, congenital syphilis, epidemiology, management, pathophysiology, screening, syphilis during pregnancy, treatment

Syphilis is a sexually transmitted bacterial infection. If detected early, syphilis is considered both treatable and curable, with little risk for comorbidities. Despite proactive efforts by the World Health Organization (WHO) (2007-2012) to curb the spread of syphilis, cases of primary and secondary syphilis continue to trend upward.[1-3] More specifically, rates of primary and secondary syphilis infections rose from 0.9 to 1.9 cases per 100,000 females (2012-2016), with rates of syphilis highest among females living within the Western and Southern regions of North American.[4] Similarly, rates of congenital syphilis (CS) infection rose from 8.4 to 15.7 cases per 100,000 live births from 2012 to 2016, an 86.9% increase.[4]

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Ethnic disparities, low socioeconomic status, unsafe sexual practices, inadequate treatment during pregnancy, and partial or no prenatal care due to limited access to medical care in certain North American regions are positively associated with an increased risk for syphilis infection during pregnancy and subsequent CS.[2,5,6] Furthermore, the stigma and discrimination associated with sexually transmitted infections often deter at-risk females from seeking appropriate prenatal care.[2,5,6] Inconsistent maternal syphilis screening during pregnancy contributes to missed diagnostic and curative opportunities, fetal infection, and resultant mortality and morbidity risks.[2,5,6]

Congenital syphilis impacts both perinatal and neonatal care. Congenital syphilis can be acquired transplacentally, as early as the 14th week of fetal development, or by direct skin-to-skin contact with a vaginal syphilitic lesion during delivery.[7] Syphilis left untreated during pregnancy can lead to severe fetal neurological, developmental, and musculoskeletal impairments, as well as fetal demise.[8,9] Morbidity and mortality risks during the perinatal period are estimated at 33.6% and 6.5%, respectively.[10] Maternal syphilis infection may be without obvious

clinical manifestations and hidden behind a shroud of shame; therefore, properly timed prenatal screening is crucial.[11]

Increased awareness of CS is essential for the obtainment of optimal neonatal outcomes. The purpose for this article is to present pertinent epidemiological trends, as well as the pathophysiology, diagnosis, and management of CS, as it relates to collaborative nursing and medical care of the affected family unit.

## EPIDEMIOLOGICAL TRENDS: 2007 TO 2016

In 2007, 12 million individuals were infected with syphilis annually, with pregnant females accounting for 1.9 million of all cases.[1,2] Due to those staggering statistics, the WHO launched an 8-year global initiative aimed at eliminating mother-to-child transmission of syphilis by 2015. Scheduled prenatal testing in the first and third trimesters and treatment of seropositive women and their infected partners were adopted as standards of practice, to promote early detection and timely treatment of CS.[1,2]

The WHO initiative resulted in a temporary regression in the incidence of CS. Over the past 5 years, however, failed adherence to prenatal screening, risky sexual behaviors, and late or limited prenatal care have contributed to the aggregate increased rates of CS infection within the United States, from 8.4 to 15.7 per 100,000 live births (2012-2016) (Figure 1).[4] The majority of maternal and CS infections are observed within the Western and Southern United States, with an alarming 42.3% increase in cases of CS (2012-2016) in the West (Figure 2).[4]

Ethnic disparities are well reported. The incidence of syphilis among African Americans is 43.1 cases per 100,000 live births compared with American Indians with 31.6 cases per 100,000 live births.[3] Hispanics account for 20.5 cases per 100,000 live births, while Asian and Pacific Islanders account for 9.2 cases per 100,000 live births, and whites with 5.3 cases per 100,000 live births.[4] Thoughtful consideration of ethnicity as a risk factor for CS infection is important.

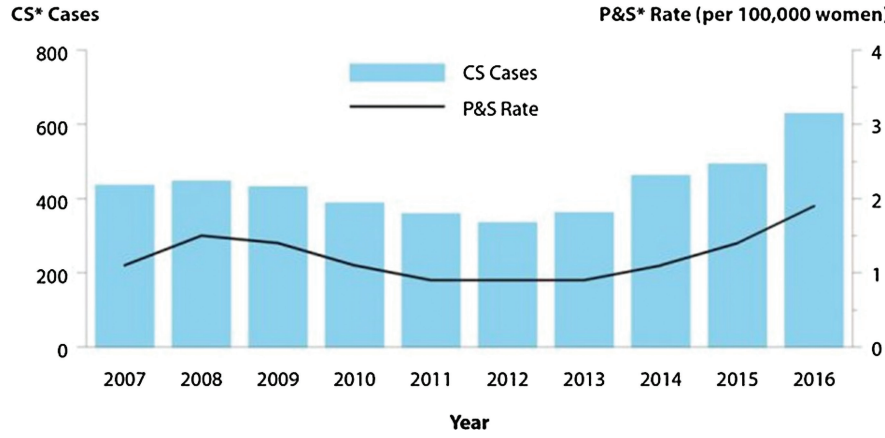
## MATERNAL SYPHILIS INFECTION

Syphilis is caused by the spirochete bacterium *Treponema pallidum* and is predominately transmitted by sexual contact with an infected lesion.[9] Major risk factors for the acquisition of syphilis include sexual contact with men who are sexually active with other men, African American ethnicity, a history of sexually transmitted infections, multiple sexual partners, illicit drug use, poverty, poor education, and uninsured or underinsured medical status.[12,13] Syphilis infection is divided into 4 sequential stages based upon clinical findings: primary, secondary, latent, and tertiary.

### Primary Infection

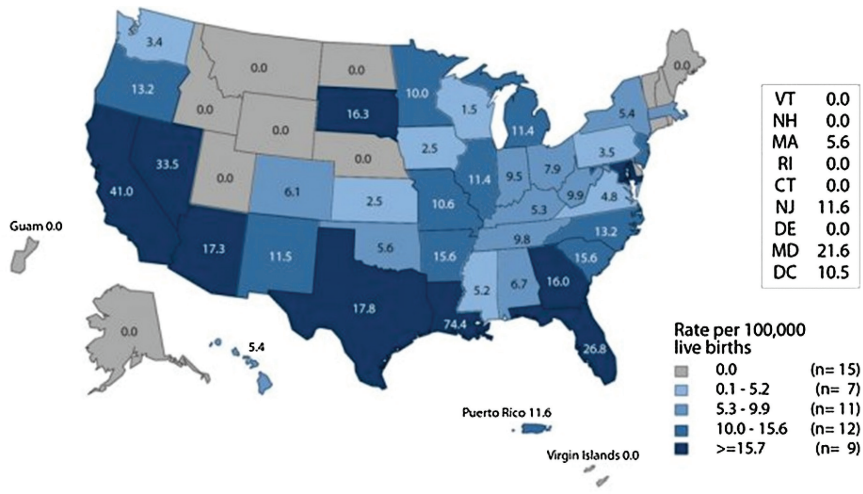
The highly contagious primary stage of syphilis occurs within the first 90 days of exposure.[9,14] Clinical manifestations are usually limited to one or few lesions at the origin of infection.[14] For pregnant females, primary infection often occurs intravaginally, as a result of sexual intercourse. If detected by prenatal providers, primary infection can be treated and cured. If undiagnosed, congenital infection is likely.[9]

FIGURE 1




Congenital syphilis—reported cases by year of birth and rates of primary and secondary syphilis among women.[4] CDC report showing in graph format, the reported cases of congenital syphilis and primary and secondary syphilis among women in the United States from 2007 to 2016. CDC materials available on the CDC web site are in the public domain and free of copyright restrictions unless otherwise noted.

FIGURE 2




Congenital syphilis—rates of reported cases among infants per 100,000 live births.[4] CDC report formatted as a map of the United States and outlying areas illustrating the rate of reported cases of congenital syphilis per 100,000 live births in 2016. CDC materials available on the CDC web site are in the public domain and free of copyright restrictions unless otherwise noted.

### Secondary Infection

If left untreated, primary syphilis will progress to secondary syphilis. Manifestations may include maculopapular rashes on the palms of the hands and soles of the feet, as well as sores on the genitals, anus, or mouth. Other commonly reported clinical manifestations include fever, malaise, lymphadenopathy, and myalgia. These clinical findings may mimic other commonly occurring diseases, such as flu and the common cold, increasing the risk for missed diagnostic opportunities by primary care and prenatal care providers.[9,15]

### Latent Infection

Untreated secondary infection will progress to latent syphilis. During the stage of latent syphilis, the infection is concealed. No visible clinical manifestations are evident.[9,16]

### Tertiary Infection

Some affected persons will develop tertiary syphilis often decades after primary infection. In this case, the infection lies dormant (patient is seropositive but asymptomatic) within the body and can present decades after the initial infection.[7] The tertiary stage is associated with life-threatening comorbidities including multiorgan damage and failure.[16] Comorbidities may include infiltrative tumors of the skin, bones, and liver, occurring in up to 15% of all cases.[7] Most often seen in the liver, these proliferative lesions are believed to occur as an inflammatory response to the treponemes.[17] Neurological complications include meningovascular and parenchymatous damage and neurosyphilis, which manifests as irritability, insomnia, memory loss, and personality changes.[9,17] A common cardiovascular presentation is proximal aortic aneurysm with aortic regurgitation.[17]

## PATHOPHYSIOLOGY OF CONGENITAL SYPHILIS INFECTION

Although CS can be acquired at any time during pregnancy or birth, transplacental transmission commonly occurs during the early fetal period of development (14-16 weeks of gestation), a time when *T. pallidum* spirochetes that are transmitted across the placenta are able to pass through the chorionic layers of the amniotic sac and infiltrate the developing fetus.[7,18] After fetal infection occurs, pervasive spirochetal dissemination can affect and disrupt multiple organ systems.[7] Approximately 21% to 40% of untreated or inadequately treated maternal syphilis cases result in fetal loss.[2] Fetal death is mainly caused by placental infection and the resultant decrease in placental blood flow.[7] Vertical transmission occurs when the mother transmits the bacteria to the infant during birth; although less common, transmission is possible if the infant comes in contact with an infected lesion when passing through the birth canal.[9]

### Clinical Manifestations of Congenital Syphilis

Infants may have typical signs and symptoms or they may be asymptomatic.[19] The stage of maternal syphilis, prenatal treatment, fetal immunological response, and gestational age influence the degree of clinical manifestations.[7,9] The disease may manifest

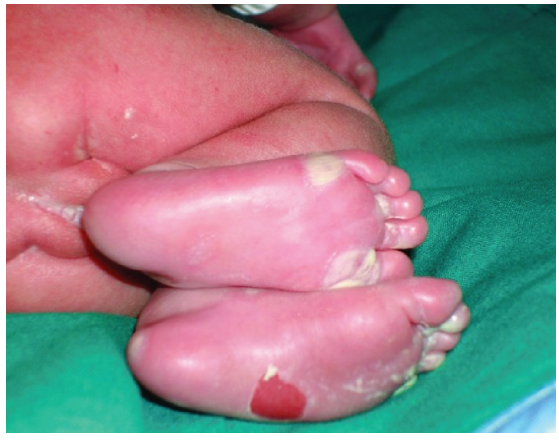
in 2 categories: early congenital syphilis (ECS) and late congenital syphilis (LCS).

### *Early Congenital Syphilis*

ECS is usually identified by 3 months of age, but symptoms may present as late as 2 years of age.[7,19] Typical features seen in ECS include organomegaly, jaundice, anemia, thrombocytopenia, mucocutaneous lesions, generalized edema, and abnormalities of the eyes, ears, and nose.[7] Hepatomegaly presents in nearly all infants, while splenomegaly presents in approximately half of cases.[7] Jaundice occurs due to direct hyperbilirubinemia with elevated serum transaminase and alkaline phosphatase concentrations as a result of syphilitic hepatitis and hemolytic anemia.[7] The presence of petechial lesions may be a result of thrombocytopenia from abnormal spleen and liver functions.[7]

Mucocutaneous involvement may present at birth or within the first few weeks of life as small copper-red maculopapular lesions on the body, with the hands and feet being the most likely and most severely affected areas (Figure 3).[7,9,20,21] Mucocutaneous lesions and the associated discharge are highly infectious and contain a large amount of the spirochete.[7] Rhinitis with bloody mucus discharge (snuffle) may be present during the first week of life or as late as 3 months of age.[7,9] Ocular and vestibular involvement, although rare in the early stage, may include chorioretinitis, glaucoma, uveitis, cataracts, eyelids lesions, and hearing loss.[7] Ulceration of the nasal mucosa may spread to the nasal cartilage, causing a *saddle nose* deformity, or collapse of the nasal bridge.[7] Other general symptoms may include feeding difficulties, failure to thrive, irritability, fever, malaise, and pneumonia.[15]

FIGURE 3



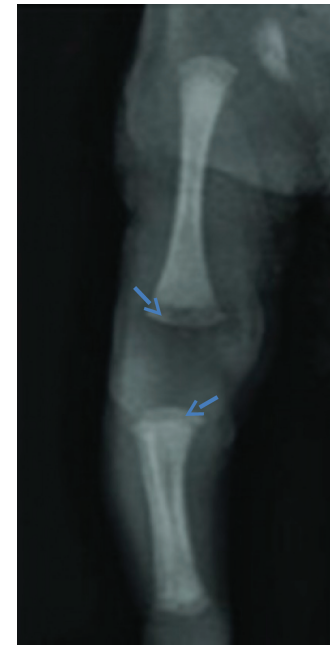
Congenital syphilis skin rash/copper-red maculopapular lesions.[7,9,20,21] Cutaneous findings can appear at birth or within the first few weeks of life as desquamated copper-red maculopapular skin rash mainly on the face, palm, and soles.

### *Late Congenital Syphilis*

Untreated ECS may lead to LCS, which is diagnosed any time after 2 years of age.[22] Late CS develops in approximately 40% of untreated children; symptoms include syphilitic rhinitis, syphilitic vasculitis, interstitial keratitis, and neurological and musculoskeletal abnormalities.[7,9] Syphilitic rhinitis can affect the central portion of the face, causing deformities of the nose, cartilage, and maxilla.[7] Syphilitic vasculitis is responsible for dental abnormalities such as peg-shaped, wide-spaced teeth known as Hutchinson's teeth; mulberry molars, which are characterized by hypertrophy and pitting of the enamel; and increased risk of cavities.[7,9] A classic finding is perforation of the hard palate.[23] Interstitial keratitis is usually manifested as secondary glaucoma or corneal clouding and may not present until the second decade of life.[7]

Neurological involvement (neurosyphilis) can result in hydrocephalus, seizure disorders, developmental delays, deafness, blindness, and juvenile general paresis.[7] Musculoskeletal manifestations are rare at this stage but may include frontal bossing, a bulging of the forehead; saber shin, a bowing of the tibia; osteochondritis and periostitis at the epiphysis and metaphysis, resulting in lucent epiphyseal bands (Figure 4); Higoumenakis' sign, a unilateral enlarging of the clavicle; and Clutton's joints, a bilateral

FIGURE 4



Congenital syphilis long bone x-ray. Osteochondritis and periostitis resulting in lucent epiphyseal bands.[24] Syphilitic changes of the skeletal system demonstrated on radiographic examination as diaphyseal periostitis, osteochondritis, and lucent epiphyseal bands.

swelling and/or inflammation of the elbows and knees.[7,24] Early diagnosis is important to prevent progression to LCS and severe, long-term sequelae.[25,26]

### *Lifelong Implications of Congenital Syphilis*

Infants affected by CS are at risk for severe, lifelong sequelae associated with the disease. If unidentified and/or left untreated, the disease can progress to organ damage, including heart failure; brain damage and infections, which can result in seizures and paralysis; and deformities of the arms and legs resulting in immobility.[7,8] Other detrimental consequences may include growth restrictions, hearing loss, blindness, and death.[7,8] These medical implications exacerbate the emotional and economic burdens faced by families due to the need for frequent follow-ups aimed at limiting these sequelae; this highlights the importance of the nurse's role in early identification of the disease.[8]

## DIAGNOSIS OF CONGENITAL SYPHILIS

The easy transfer of immunoglobulin (Ig) G antibodies across the placenta to the fetus makes the diagnosis of CS challenging in the fetal and early neonatal period, as it can complicate the interpretation of serologic tests in the neonate.[1,11] Prenatal testing for CS can also be complicated by the inability to successfully culture the *T. pallidum* bacteria.[2] Direct visualization of the spirochete and serologic testing continue to be the gold standard for diagnosing the infection due to these perplexities, with serologic testing the more common due to cost-effectiveness, ease of use, and reliability.[2,27] Imaging studies and percutaneous umbilical cord blood sampling can also aid in the diagnosis.[28]

### Prenatal Diagnosis

Prenatal screening results in decreased fetal mortality and is the rationale for serologic testing in the early trimesters.[23] Positive maternal serologic testing during any stage of pregnancy is concerning for ECS, mandating neonatal testing.[23] The Centers for Disease Control and Prevention (CDC) recommends routine testing at the first prenatal visit for all pregnant women, and in the second trimester and at delivery for high-risk women and those living in high prevalence areas.[11] Laboratory serologic testing falls into 2 categories: nontreponemal and treponemal. Direct detection in the form of DNA assessment and visualization of the bacteria are confirmatory testing methods as well.[9]

Nontreponemal tests are nonspecific and may produce similar results in the presence of other viral and bacterial infections, or autoimmune conditions.[23,27] Pregnancy can also cause false-positive results. The nontreponemal serology tests are used to both screen and monitor the status of the infection. Nontreponemal tests include the venereal disease research laboratory (VDRL) and the rapid plasma reagin (RPR), which measure the levels of IgG and IgM antibodies.[27] These antibodies can be detected as soon as 6 days after bacterial invasion.[27] The RPR is predominantly used for serum testing and the VDRL for cerebrospinal fluid testing.[25] A positive screen should be followed by a treponemal test, which is more specific.[23,27]

The treponemal serology tests are used for confirmation of the presence of the bacteria and detect antibodies against *T. pallidum* antigens.[23,27] Treponemal tests include fluorescent treponemal antibody-absorbed test, microhemagglutination assay for *T. pallidum*, enzyme-linked immunosorbent assays, and Treponemal IgM in serum.[27,29] Treponemal tests can detect both IgM and IgG antibodies that are specific to *T. pallidum*.[27] These antibodies remain positive for life; therefore, positive results need to be verified with nontreponemal testing to determine past versus active infection.

Although used less commonly, polymerase chain reaction techniques used to amplify and detect the *T. pallidum* DNA in tissue and body fluid samples are more reliable due to rapid turnaround times, validity, sensitive detection of *T. pallidum*, and diagnostic accuracy.[27,30] These characteristics of polymerase chain reaction enable rapid diagnosis and implementation of treatment.[30] Dark-field microscopy or immunofluorescent staining can be performed to directly visualize the bacteria in fluid samples from lesions and infected tissues.[27]

The presence of *T. pallidum* in amniotic fluid or fetal blood can confirm the diagnosis in utero. Prenatal ultrasounds may also reveal features that are suggestive of CS including hepatomegaly, splenomegaly, placentomegaly, and fetal growth restriction.[28] Prenatal screening allows for prompt treatment and reduction in the sequelae of CS, highlighting the importance of serologic screening at different stages of pregnancy.[23]

### Postnatal Diagnosis

The diverse clinical features of CS can make the diagnosis daunting. Assessment should begin with a thorough physical examination for skin lesions, jaundice, mucous membrane fissures or patches, and thick or bloody nasal discharge.[19,23] Next, detailed palpation should occur to assesses for organomegaly.[19]

All infants suspected of having CS should be tested with the same nontreponemal tests that were performed on the mother, and the results should be analyzed for the difference in titers.[4,23] A positive serum IgM in the infant, detected through RPR, is reflective of active syphilis infection because maternal IgM does not cross the placenta.[23] Congenital syphilis is indicated when the nontreponemal serologic titer in the infant is fourfold higher than that of the

mother.[11,23] The American Academy of Pediatrics and the CDC recommend that all infants born to mothers who were inadequately treated during pregnancy should be further evaluated with complete blood counts and cerebrospinal fluid analysis for protein, cell count, and quantitative VDRL.[23] Other diagnostic tests include eye examinations to assess for structural abnormalities; chest and long bones radiography, which may show radiolucency, osteochondritis, periostitis, bone destruction, and opacities; and liver function tests.[23,25] Once diagnosis is made, prompt and adequate treatment is necessary to minimize sequelae.

## MANAGEMENT OF CONGENITAL SYPHILIS

Parenterally administered penicillin G is the only known effective antimicrobial to treat maternal syphilis and prevent maternal transfer to the fetus or newborn.[7,9,11] No other antibiotic efficaciously destroys the *T. pallidum* bacteria; therefore, desensitization has to be instituted and the therapy continued in cases of penicillin allergy.[2,9,13,23] The efficacy of intramuscularly administered benzathine penicillin G against syphilis is credited to its slow release into the body tissues.[13] Management and treatment of CS depend on the stage and treatment of maternal disease, clinical manifestations, and evaluation of the findings in the infant (Table 1).[23]

Diagnosis made later in infancy is subject to more aggressive and more frequent dosing due to the risk of neurosyphilis.[23] The infant diagnosed with CS should have structured follow-up that includes nontreponemal serologic testing every 3 months until the tests are nonreactive or the titers are less than fourfold.[4]

## IMPLICATIONS OF CONGENITAL SYPHILIS

Congenital syphilis is a preventable disease; timely, adequate treatment of the pregnant woman affected with syphilis can limit the associated emotional, social, economic, and medical burdens.[10,31] The emotional loss of a fetus or child can be traumatizing for parents.[31] When losses are caused by congenital infections, such as syphilis, the psychological effect of the traumatic event can be escalated by feelings of guilt, blame, and in some cases, depression in the mother.[31] Therefore, implementation of programs that place focus on the prevention of CS is warranted.[31]

The economic burden associated with the treatment of CS affects not only families, but society as a whole.[31] The hospitalization cost for an infant affected by CS is as much as 7 times higher, and the length of hospital stay is approximately 8 days longer than for a healthy infant.[32] These cost increase estimates do not include postdischarge medical expenses related to late CS.[32] In comparison, prenatal screening offers a long-term cost-benefit for public health entities.[31]

## IMPLICATIONS FOR NURSING PRACTICE

Nurses play an integral role in the detection, early implementation of treatment, effective management, and elimination of CS.[33] The expert knowledge

TABLE 1. Management of Confirmed, Suspected, and Asymptomatic-Confirmed Congenital Syphilis

| Criteria | Treatment Regimen |
|---|---|
| Confirmed or highly probable CS[4,6,9,23]<br>• Abnormal PE or<br>• Serological titer >4-fold maternal titer or<br>• Positive DFM or PCR | Aqueous crystalline penicillin G 50,000 μ/kg/dose IV<br>• Q12 h × 7 d followed by<br>• Q8 h × 3 d *OR*<br>Procaine penicillin G 50,000 μ/kg/dose IM daily × 10 d |
| Possible CS[4,6,9,23]<br>• Normal PE, titers ≤4-fold and inadequately treated mother | As above *OR*<br>Benzathine penicillin G 50,000 μ/kg/dose IM × 1 dose |
| Less likely CS[4,6,9,23]<br>• Normal PE and adequately treated mother | Benzathine penicillin G 50,000 μ/kg/dose IM × 1 dose |
| Unlikely CS[4,23]<br>• Normal PE, adequately treated mother, and low maternal serologic titer | No treatment *OR*<br>Benzathine penicillin G 50,000 μ/kg/dose IM × 1 dose |
| Symptomatic diagnosed CS[4,23]<br>• Reactive serologic tests with clinical manifestations | Aqueous crystalline penicillin G 50,000 μ/kg/dose IV<br>• Q4-6 h × 10 d |
| Asymptomatic diagnosed CS[4,23]<br>• Reactive serologic test without clinical manifestations | Benzathine penicillin G 50,000 μ/kg/dose IM weekly μ 3 wk |

*Abbreviations: CS, congenital syphilis; DFM, dark-field microscopy; IM, intramuscular; IV, intravenous; PCR, polymerase chain reaction; PE, physical examination.*

| Summary of Recommendations for Practice and Research | |
|---|---|
| **What we know:** | • Congenital syphilis has remained persistent and is on the rise in the United States.<br>• Syphilis left untreated during pregnancy can increase morbidity in infants and mortality of the fetus, neonate, or infant.<br>• Penicillin G effectively treats syphilis and congenital syphilis. |
| **What needs to be studied:** | • Why syphilis is more prevalent in certain geographical areas and certain ethnicity.<br>• Reasons for lack or inadequacy of treatment during pregnancy.<br>• The effects of penicillin G on the fetus. |
| **What we can do today:** | • Follow WHO and CDC guidelines for testing during pregnancy.<br>• Treat infected mothers with penicillin as recommended by the WHO and the CDC.<br>• Ensure all mothers have been screened for syphilis before the infant is discharged from the hospital.<br>• Structured follow-up of affected infants. |

possessed by neonatal nurses and neonatal nurse practitioners empowers them to be vigilant in gathering comprehensive pertinent maternal history and performing careful detailed physical examinations of the newborn. Ordering the appropriate diagnostic tests; correctly interpreting results; initiating timely treatment and management strategies; and preparing the infant for structured follow-up while effectively communicating the plan of care to the family are all activities that neonatal nurse practitioners are efficient at.[7,8,15,33]

The maternal history will provide pertinent information regarding the need for further diagnostic evaluation of the neonate. Newborns of mothers with a reactive nontreponemal or treponemal serologic test or who never received syphilis screening during pregnancy should have serologic testing done in the form of RPR or VDRL prior to hospital discharge.[9,11] Bedside registered nurses are influential in ensuring that laboratory tests are collected in a timely manner, and neonatal nurse practitioners are leaders in guaranteeing judicious results review and interpretation.

Nurses must be aware that the nonspecific clinical manifestations of CS, including edema, rash, organomegaly, anemia, thrombocytopenia, and snuffles, can be overlooked or misinterpreted for other illnesses such as pneumonia.[15] Of equal importance is the need for nurses to be knowledgeable about inpatient and outpatient management strategies so they can educate parents on the plan of care and expected outcomes. The obtainment of best outcomes can be achieved through parental education on the necessity for compliance with treatment and adherence to follow-up appointments to evaluate and confirm the effectiveness of treatment.[8]

## CONCLUSION

Congenital syphilis, a disease caused by the *T. pallidum* bacterium, continues to persist in the United States despite being preventable with appropriate prenatal screening and adequate penicillin treatment.[19,20,25] Syphilis left untreated during pregnancy poses the greatest risk of severe irreversible sequelae and/or fetal, neonatal, and infant death.[4] Vigilant prenatal and at delivery screening, treatment of the infected mother during pregnancy, meticulous assessment of the newborn, and prompt initiation of treatment with benzathine penicillin G when indicated, along with appropriate follow-up postdischarge, are crucial in reducing the incidence of CS and constraint of negative sequelae.[4,11,15,25] Neonatal nurses are experts in newborn care, advocates at the bedside, and knowledgeable about infectious diseases; as such, they have a critical role in the interprofessional strategic approach to decrease the incidence and limit the severe sequelae of CS.

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