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CYTOMEGALOVIRUS INFECTION IN PREGNANT WOMEN AND NEWBORNS: ETIOLOGY, TRANSMISSION ROUTES, AND CLINICAL MANIFESTATIONS

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ABSTRACT:

Cytomegalovirus (CMV) infection represents the most prevalent congenital viral infection worldwide, affecting 0.5-2% of live births and constituting the leading non-genetic cause of sensorineural hearing loss and neurodevelopmental disability in children. This comprehensive review examines the multifaceted aspects of CMV infection in pregnant women and newborns, with particular emphasis on etiological factors, transmission pathways, and clinical presentations. Primary maternal infection during the first trimester carries the highest risk of vertical transmission (30-40%) and severe fetal sequelae, although non-primary infections also contribute significantly to disease burden due to maternal seroprevalence rates exceeding 50% in many populations. Transmission occurs through contact with infected bodily fluids, with young children serving as primary reservoirs. Clinical manifestations in congenitally infected newborns range from asymptomatic courses (approximately 85-90%) to symptomatic disease characterized by

hepatosplenomegaly, petechiae, thrombocytopenia, jaundice, microcephaly, and neurological involvement. Long-term sequelae, particularly sensorineural hearing loss affecting 30-50% of symptomatic and 8-15% of initially asymptomatic infants, underscore the public health significance of this infection. This review synthesizes current evidence regarding pathogenesis, risk factors, diagnostic approaches, and clinical outcomes to enhance understanding of this clinically important congenital infection.

Introduction

Cytomegalovirus (CMV), a ubiquitous DNA virus belonging to the Herpesviridae family, represents the most common cause of congenital infection globally, with an estimated 350,000 children born annually with long-term sequelae [Bailey et al., 2025, p. 450]. Despite its significant public health impact, CMV remains underrecognized among healthcare providers and the general public, leading to missed opportunities for prevention and early intervention [Williamson-Leon et al., 2025, p. 8]. The clinical spectrum of congenital CMV (cCMV) infection is remarkably diverse, ranging from completely asymptomatic newborns to those with severe multiorgan involvement and permanent neurological damage [Zhang et al., 2025, p. 2026]. Approximately 10-15% of infected infants exhibit clinically apparent symptoms at birth, while the remaining 85-90% appear healthy initially. However, even among asymptomatic neonates, 8-15% will subsequently develop late-onset sequelae, particularly sensorineural hearing loss (SNHL), which may manifest months or years after birth [Zhang et al., 2025, p. 2026].

The pathogenesis of cCMV infection is intricately linked to gestational timing, with first-trimester maternal primary infection conferring the highest risk of severe fetal consequences [Moutsopoulou et al., 2025, p. 929]. The virus's ability to establish latency, reactivate, and cross the placental barrier complicates our understanding of transmission dynamics and clinical outcomes. Furthermore, the absence of universal screening programs in most countries means that many affected infants remain undiagnosed until irreversible neurological damage has occurred [Williamson-Leon et al., 2025, p. 9].

This review aims to provide a comprehensive analysis of the causes, transmission routes, and clinical manifestations of CMV infection in pregnant women and newborns, synthesizing current evidence to inform clinical practice and guide future research directions.

Literature review

Epidemiology and Global Burden - The global birth prevalence of congenital CMV infection ranges from 0.5% to 2.0% in developed countries, with higher rates observed in developing regions [Moutsopoulou et al., 2025, p. 929]. A longitudinal Australian study spanning 1999-2023 identified 586 cases of cCMV infection, corresponding to 8.15 per 100,000 births, although this likely represents significant underascertainment compared to expected prevalence estimates [Egilmezer et al., 2025, p. 611]. Similarly, Japanese claims data from 2011-2019 demonstrated a diagnosed birth prevalence ranging from 6.88 to 22.56 per 100,000 births, highlighting substantial variations in case identification across healthcare systems [Buck et al., 2025, p. 1].

Maternal seroprevalence varies considerably by geographic region, socioeconomic status, and age. In high-income countries, seroprevalence increases from approximately 40-50% in higher socioeconomic groups to 70-80% in lower socioeconomic populations [Williamson-Leon et al., 2025, p. 8]. This epidemiological pattern has important implications for the relative contributions of primary versus non-primary maternal infections to congenital disease burden.

Virological Characteristics and Pathogenesis - CMV exhibits characteristic herpesvirus features including latency and reactivation potential. Following primary infection, the virus establishes lifelong persistence in multiple cell types, with periodic reactivation occurring throughout the host's lifetime [Palmetti et al., 2025, p. 381]. In immunocompetent individuals, these reactivation episodes typically remain asymptomatic but facilitate viral transmission to susceptible contacts.

The pathogenesis of fetal infection involves complex interactions between viral factors and the developing fetal immune system. CMV demonstrates particular tropism for neural progenitor cells, disrupting normal neuronal migration and cortical development [Palmetti et al., 2025, p. 381]. This neurotropism explains the predominance of central nervous system manifestations, including microcephaly, intracranial calcifications, and subsequent neurodevelopmental impairment.

Discussion

Causes and Risk Factors for Maternal Infection - Sources of Infection. CMV transmission occurs through direct contact with infected bodily fluids, including urine, saliva, blood, tears, semen, and cervical secretions [Williamson-Leon et al., 2025, p. 9]. Unlike many viral pathogens, CMV is not transmitted through airborne routes or casual contact, requiring close interpersonal contact for effective transmission.

Young children serve as the primary reservoir for CMV transmission to pregnant women. Infected children typically shed the virus for months to years following primary infection, with viral concentrations in urine and saliva reaching levels substantially higher than those observed in immunocompetent adults [Moutsopoulou et al., 2025, p. 930]. This prolonged shedding pattern, combined with frequent close contact during diaper changes, feeding, and shared utensil use, creates significant exposure opportunities for caregivers.

Maternal Immune Status. The risk of vertical transmission and subsequent fetal disease is profoundly influenced by maternal immune status at the time of infection. Primary maternal infection during pregnancy carries approximately 30-40% risk of vertical transmission, with the highest rates observed when infection occurs during the first trimester [Fourgeaud et al., 2025, p. 2].

Historically, maternal pre-existing immunity was considered protective against fetal infection. However, contemporary evidence demonstrates that non-primary infections (reactivation or reinfection with different viral strains) account for a substantial proportion of cCMV cases, particularly in highly seropositive populations [Bailey et al., 2025, p. 452]. This finding has significant implications for prevention strategies, suggesting that seropositive women remain at risk for delivering infants with cCMV.

Gestational Timing. The relationship between gestational age at maternal infection and fetal outcomes follows a paradoxical pattern. First-trimester infections, while associated with lower transmission rates (approximately 30%), carry the highest risk of severe fetal sequelae when transmission occurs [Moutsopoulou et al., 2025, p. 931]. Conversely, third-trimester infections transmit more frequently (up to 70%) but typically result in asymptomatic or mildly affected newborns.

This phenomenon reflects the developmental vulnerability of the early gestation fetus, particularly regarding central nervous system organogenesis. CMV infection during critical windows of neuronal migration and cortical formation produces irreversible structural damage, whereas later infection may allow for more complete immune-mediated viral clearance with less permanent tissue destruction [Palmetti et al., 2025, p. 382].

Transmission Routes to the Fetus and Newborn

Vertical Transmission. Transplacental transmission represents the primary route of congenital CMV infection. Following maternal viremia, the virus reaches the placental interface, where it must overcome multiple barriers including trophoblast layers and fetal endothelial cells [Raptopoulou et al., 2025, p. 770]. Viral replication within placental tissues may occur before fetal infection is established, with placental involvement serving as both a prerequisite for transmission and a potential source of fetal infection independent of maternal viremia.

The precise mechanisms governing transplacental transmission remain incompletely characterized. However, emerging evidence suggests that viral glycoprotein interactions with cellular receptors on trophoblasts facilitate viral entry, while maternal inflammatory responses may enhance placental permeability [Fourgeaud et al., 2025, p. 3].

Intrapartum and Postnatal Transmission. In contrast to transplacental transmission, intrapartum exposure to infected cervical secretions and postpartum transmission through breast milk typically result in asymptomatic or minimally symptomatic infection in term infants. The presence of maternally derived antibodies provides substantial protection against invasive disease in this setting [Williamson-Leon et al., 2025, p. 10].

However, for very low birth weight preterm infants, particularly those born before 32 weeks' gestation, postnatal CMV acquisition through breast milk may produce symptomatic disease resembling congenital infection [Bailey et al., 2025, p. 453]. This observation underscores the importance of maternal immune status and gestational age in determining clinical outcomes following perinatal exposure.

Clinical Manifestations in Newborns

Symptomatic Congenital Infection. Approximately 10-15% of congenitally infected newborns exhibit clinically apparent manifestations at birth [Zhang et al., 2025, p. 2026]. The classic symptom constellation includes:

Hepatobiliary manifestations: Hepatomegaly occurs in 15-40% of symptomatic infants, often accompanied by splenomegaly and conjugated hyperbilirubinemia [Egilmezer et al., 2025, p. 613]. Australian surveillance data documented liver disease with jaundice in 27.1% of definite cCMV cases, hepatomegaly in 15.7%, and hepatitis in 14.7% [Egilmezer et al., 2025, p. 613].

Hematological abnormalities: Thrombocytopenia represents one of the most frequent findings, occurring in approximately 29% of symptomatic infants, and manifests clinically as petechiae or purpura (18.6% of cases) [Egilmezer et al., 2025, p. 613]. These findings

result from viral suppression of bone marrow precursors and peripheral platelet consumption.

Central nervous system involvement: Neurological manifestations constitute the most clinically significant aspects of cCMV infection. Microcephaly, reflecting impaired brain growth, occurs in 18.6% of symptomatic infants [Egilmezer et al., 2025, p. 613]. Intracranial calcifications, typically periventricular in distribution, represent characteristic neuroimaging findings resulting from necrotizing encephalitis and subsequent dystrophic calcification [Palmetti et al., 2025, p. 383].

Intrauterine growth restriction: Infants with cCMV are frequently small for gestational age, with Australian data demonstrating intrauterine growth restriction in 28.2% of definite cases [Egilmezer et al., 2025, p. 613]. This finding reflects placental insufficiency and direct viral effects on fetal growth regulation.

Asymptomatic Infection and Late-Onset Sequelae. The majority (85-90%) of congenitally infected infants exhibit no clinically apparent abnormalities at birth [Zhang et al., 2025, p. 2026]. However, the designation "asymptomatic" may be misleading, as these infants remain at risk for significant long-term complications.

Sensorineural hearing loss represents the most common sequela of asymptomatic cCMV, affecting 8-15% of initially asymptomatic children [Zhang et al., 2025, p. 2027]. Unlike congenital hearing loss from other etiologies, CMV-associated SNHL may be progressive, fluctuating, or late-onset, with initial presentation occurring months to years after birth. This unpredictable pattern necessitates prolonged audiological surveillance for affected children.

Beyond hearing impairment, emerging evidence suggests that 5-10% of initially asymptomatic children may develop neurodevelopmental abnormalities including mild intellectual disability, attention deficit disorders, learning difficulties, and motor coordination problems [Palmetti et al., 2025, p. 384]. Given the substantial population of asymptomatic cCMV cases, these delayed complications account for 30-40% of all cCMV-related long-term morbidity [Zhang et al., 2025, p. 2027].

Long-Term Outcomes. The prognosis for infants with cCMV varies considerably based on clinical presentation at birth. Symptomatic infants face 40-60% risk of permanent neurological sequelae, including SNHL, cognitive impairment, cerebral palsy, and seizure disorders [Moutsopoulou et al., 2025, p. 932]. The severity of neurological involvement correlates with neuroimaging findings, with extensive intracranial calcifications and cortical malformations portending poorer outcomes.

Results

Synthesis of current literature reveals several key findings regarding CMV infection in pregnant women and newborns:

1. **Prevalence and Burden:** Congenital CMV affects 0.5-2% of live births globally, making it the most common congenital infection. Primary maternal infection during the first trimester carries the highest risk of severe fetal sequelae, with 40-50% of affected fetuses sustaining permanent damage [Moutsopoulou et al., 2025, p. 929].
2. **Transmission Dynamics:** Vertical transmission occurs in 30-40% of primary maternal infections, with rates varying by gestational age. Young children serve as primary reservoirs for maternal infection, with viral shedding persisting for months to years following pediatric infection [Williamson-Leon et al., 2025, p. 9].
3. **Clinical Spectrum:** Symptomatic congenital infection (10-15% of cases) presents with hepatosplenomegaly (15-40%), thrombocytopenia (29%), microcephaly (18.6%), and intrauterine growth restriction (28.2%) [Egilmezer et al., 2025, p. 613]. Asymptomatic newborns constitute 85-90% of cases but remain at risk for late-onset sequelae.
4. **Hearing Loss:** SNHL affects 30-50% of symptomatic and 8-15% of initially asymptomatic children with cCMV [Zhang et al., 2025, p. 2027]. The hearing loss may be bilateral or unilateral, progressive, and late-onset, requiring prolonged audiological monitoring.
5. **Neurodevelopmental Outcomes:** Beyond hearing impairment, cCMV contributes significantly to neurodevelopmental disability, including cognitive impairment, motor dysfunction, and behavioral disorders. The pathogenesis involves viral disruption of neuronal migration and cortical development during early gestation [Palmetti et al., 2025, p. 382].
6. **Healthcare Burden:** Children with cCMV demonstrate significantly higher healthcare utilization and costs compared to unaffected children, with increased outpatient visits, hospitalization rates, and long-term specialty care requirements [Buck et al., 2025, p. 3].

Conclusion

Cytomegalovirus infection in pregnant women and newborns represents a significant public health challenge with substantial implications for child health and development. As the most common congenital infection and leading non-genetic cause of sensorineural hearing loss, cCMV demands greater attention from clinicians, researchers, and policymakers. The pathogenesis of cCMV reflects complex interactions between maternal

immune status, gestational timing, and viral factors. Primary maternal infection during the first trimester confers the highest risk of severe fetal sequelae, although non-primary infections contribute substantially to overall disease burden in highly seropositive populations. Transmission occurs primarily through contact with infected bodily fluids, with young children serving as the principal reservoir for maternal acquisition. Clinical manifestations range from asymptomatic courses to severe multiorgan involvement with permanent neurological damage. Even among asymptomatic newborns, the risk of late-onset sequelae, particularly sensorineural hearing loss, necessitates prolonged surveillance and early intervention when abnormalities are detected. The economic burden of cCMV extends well beyond the neonatal period, with affected children requiring increased healthcare utilization and specialty services throughout childhood. Several critical knowledge gaps remain, including optimal screening strategies for maternal and neonatal infection, biomarkers predictive of long-term outcomes, and effective therapeutic interventions for affected infants. Universal newborn screening for cCMV, implemented in some jurisdictions, offers promise for early identification and intervention but requires further evaluation regarding cost-effectiveness and long-term benefits. Prevention remains the cornerstone of cCMV management. Hygiene education for pregnant women regarding exposure to young children's bodily fluids can reduce maternal acquisition risk. Antiviral prophylaxis with valacyclovir for women with primary first-trimester infection demonstrates efficacy in reducing vertical transmission [Fourgeaud et al., 2025, p. 4]. Vaccine development continues to advance, although currently available candidates demonstrate modest efficacy [Moutsopoulou et al., 2025, p. 935].

In conclusion, cytomegalovirus infection in pregnancy and early infancy represents a preventable cause of significant childhood morbidity. Enhanced awareness among healthcare providers and the public, improved diagnostic strategies, and continued research into preventive and therapeutic interventions are essential to reduce the burden of this important congenital infection.

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