



Comorbidity of Transplacental Congenital Pathogen Infection among Mothers and Neonates in Kogi State, North Central, Nigeria

By

Akor Egbunu Shedrack^{1,2,3*}, Mathias A. Emokpae², Yahaya F. Abuh³, Akubo Ojodale Andrew¹, Ogboi Johnbull Sonny^{1,3}, Akogu SPO⁴, Christian Egwumah¹, Ekpe Christian Atuluku⁵.

¹Department of Medical Laboratory Sciences, Prince Abubakar Audu University Anyigba, Kogi, Nigeria

²Department of Medical Laboratory Science, School of Basic Medical Sciences, University of Benin, Benin City, Edo State, Nigeria

³Nigo Rite Diagnostic and Research Centre, Anyigba, Kogi State

⁴Department of Obstetrics and Gynecology, Prince Abubakar Audu University Anyigba, Kogi, Nigeria.

⁵Department of Human Physiology, Prince Abubakar Audu University Anyigba, Kogi, Nigeria



Article History

Received: 02/10/2026

Accepted: 08/10/2026

Published: 10/10/2026

Vol –4 Issue – 10

PP: -01-05

ABSTRACT

Transplacental congenital pathogen infections, commonly grouped under the TORCH complex (*Toxoplasma gondii*, Others such as *Treponema pallidum*, Rubella virus, Cytomegalovirus, and Herpes simplex virus), remain major contributors to adverse pregnancy outcomes and neonatal morbidity in low- and middle-income countries. These infections can cross the placenta during pregnancy and result in miscarriage, stillbirth, preterm birth, intrauterine growth restriction, congenital anomalies, and neonatal death. In Nigeria, limited epidemiological data exist regarding the burden and co-occurrence of these infections among mothers and their newborns, particularly in North-Central states such as Kogi State. This research investigated the comorbidity of transplacental congenital pathogen infections, maternal and neonatal risk factors, and the implications for maternal and child health in Kogi State. Maternal factors such as poor antenatal care attendance, low immunization coverage, malnutrition, coinfections, poverty, and inadequate laboratory screening contribute significantly to the transmission. Neonatal consequences include congenital cytomegalovirus infection, congenital syphilis, congenital toxoplasmosis, and congenital rubella infection. The coexistence of multiple pathogens in the same mother or neonate may worsen clinical outcomes through placental inflammation, immune dysregulation, and micronutrient competition. Therefore, strengthening antenatal screening, vaccination, public health education, and laboratory diagnostic capacity is critical for reducing the burden of congenital infections in Kogi State and Nigeria as a whole.

Keywords: Transplacental congenital infections; TORCH pathogens; maternal risk factors; neonatal outcomes; pathogen comorbidity; antenatal screening.

Introduction

Congenital infections are pathogenic infectious organisms transmitted from a pregnant woman to her fetus either transplacentally during gestation, intrapartum and delivery, or postnatally through breastfeeding (Evans-Gilbert, 2020; Akor *et al.*, 2021). Among these, transplacental infections are particularly important because they can disrupt fetal organogenesis and cause lifelong disabilities or fetal loss. The most recognized group of transplacental pathogens is the TORCH complex, which includes *Toxoplasma gondii*,

Treponema pallidum (syphilis), Rubella virus, Cytomegalovirus (CMV), Herpes simplex virus (HSV), and additional pathogens such as Human Immunodeficiency Virus (HIV), hepatitis B virus, Parvovirus B19, and Varicella zoster virus. (Türkmen Albayrak, 2020)

Globally, congenital infections account for approximately 2–3% of all congenital anomalies and contribute substantially to miscarriage, stillbirth, neonatal mortality, neurodevelopmental impairment, blindness, deafness, and chronic disease. The burden is highest in low- and middle-income countries where



access to vaccination, antenatal screening, and effective treatment is limited. ((Adeniyi *et al.*, 2021).

In sub-Saharan Africa, including Nigeria, maternal exposure to multiple infectious agents is common due to endemicity of sexually transmitted infections, malaria, HIV, and poor sanitation (Ubesie *et al.*, 2013). These conditions increase the risk of vertical transmission and simultaneous infection with more than one congenital pathogen. Such comorbidity can intensify placental damage, trigger chronic inflammation, and worsen maternal and neonatal outcomes.

Kogi State, located in North-Central Nigeria, has a mixed urban-rural population with variable access to quality healthcare services. Pregnant women in many communities' face challenges such as delayed antenatal booking, inadequate diagnostic facilities, limited awareness of congenital infections, and socioeconomic barriers to care. These factors predispose mothers and newborns to undiagnosed and untreated congenital infections.

The term "comorbidity" in this context refers to the coexistence of two or more transplacental congenital infections or their simultaneous presence with other maternal conditions such as anemia, malnutrition, malaria, and HIV (Akor *et al.*, 2021). For example, a pregnant woman may be infected with both CMV and syphilis, while her neonate may present with low birth weight, hepatosplenomegaly, jaundice, and sensorineural hearing loss. Multiple infections may interact biologically through immune suppression, placental inflammation, and competition for essential micronutrients such as zinc, selenium, iron, and vitamin A.

Therefore, the study of comorbidity of transplacental congenital pathogen infections among mothers and neonates in Kogi State is imperative for several reasons, as it will provide baseline epidemiological data where little currently exists and guide development of targeted screening programs and preventive interventions that will support policies on rubella immunization, syphilis control, HIV prevention, and newborn screening, and finally, contribute to reducing neonatal morbidity and mortality in line with the World Health Organization Sustainable Development Goals.

Materials and Methods

Study Area: The study was carried out at the Prince Abubakar Audu University Teaching Hospital in Anyigba, Kogi State, while samples were processed at Nigo Rite Diagnostic and Research Centre, Anyigba

Study Population and Design: A total of 368 participants were involved in this cross-sectional study. This comprised 184 mothers' arterial blood samples and 184 umbilical cord blood samples of the newborn babies.

Ethical Approval: The study was approved by the ethical review committee of the Prince Abubakar Audu University Teaching Hospital, Anyigba.

Inclusion and Exclusion Criteria; The selection of participants followed specific criteria. Healthy expectant mothers attending the ante-natal clinic who delivered at the

healthcare center and consented to participate were included. In contrast, those with chronic illnesses, poor obstetric histories showing clinical signs, or who declined consent were excluded.

Data and sample collection: 4.5 ml of each newborn's umbilical cord arterial blood and their mother's venous blood were aseptically collected during delivery into separate labeled containers. The serum was separated from the clotted blood and preserved at -80°C for subsequent analyses.

Molecular Identification: DNA extraction of transplacental congenital pathogen was done, followed by nested PCR amplification using PCR kits from Shanghai Ideal Medical Technology Co Ltd. The resulting amplified products were confirmed through agarose gel electrophoresis at a concentration of 2%. Visualization of DNA bands was accomplished using a UV transilluminator.

Result:

Pairwise Associations of Detected Pathogens among Mothers

Table 1. shows the pairwise associations of pathogens detected in the mothers using Chi-square p-values. Significant associations were found between *Cytomegalovirus* and *Herpes Simplex Virus* ($p = 0.001$) as well as *Cytomegalovirus* and *Plasmodium falciparum* ($p = 0.001$). In contrast, its associations with *Toxoplasma gondii* ($p = 0.623$), *Rubella* ($p = 0.055$), and *Treponema pallidum* ($p = 0.055$) were not significant. *Herpes Simplex Virus* showed a strong relationship with *Plasmodium falciparum* ($p = 0.001$), while its associations with *Toxoplasma gondii* ($p = 0.105$), *Rubella* ($p = 0.075$), and *Treponema pallidum* ($p = 0.075$) were not statistically significant. For *Toxoplasma gondii*, no significant associations were observed with *Cytomegalovirus* ($p = 0.623$), *Herpes Simplex Virus* ($p = 0.105$), or *Plasmodium falciparum* ($p = 0.899$). However, significant associations were recorded with *Rubella* ($p = 0.027$) and *Treponema pallidum* ($p = 0.027$). *Plasmodium falciparum* notably exhibited strong associations with *Rubella* ($p = 0.000$) and *Treponema pallidum* ($p = 0.000$). Similarly, *Rubella* was significantly associated with *Treponema pallidum* ($p = 0.000$).

Pairwise Associations of Detected Pathogens among Neonates

Table 2. shows the pairwise associations of pathogens detected in Neonates using Chi-square p-values. Significant associations were observed between *Cytomegalovirus* and *Herpes Simplex Virus* ($p = 0.000$), as well as between *Cytomegalovirus* and *Plasmodium falciparum* ($p = 0.000$). Likewise, *Herpes Simplex Virus* showed significant associations with *Toxoplasma gondii* ($p = 0.027$), *Plasmodium falciparum* ($p = 0.000$), *Rubella* ($p = 0.004$), and *Treponema pallidum* ($p = 0.004$). Although *Toxoplasma gondii* demonstrated associations with other pathogens, only the pair with *Herpes Simplex Virus* ($p = 0.027$) was significant, while its association with *Plasmodium falciparum* ($p = 0.055$), *Rubella* ($p = 0.196$), and *Treponema pallidum* ($p = 0.196$) was not statistically significant. There were strong associations found between *Plasmodium falciparum* and *Rubella* ($p =$

0.000) as well as *Treponema pallidum* ($p = 0.000$). Similarly, *Rubella* showed a significant association with *Treponema pallidum* ($p = 0.000$).

Comorbidity of Transplacental Congenital Pathogens among Mothers and Neonates

Comorbidity of Transplacental Congenital Pathogens among Mothers shows the distribution of Transplacental Congenital Pathogens comorbid infections among mothers (Fig 1). A substantial proportion of the mothers examined had no infection (34.8%). The level of infection decreased with the number of comorbidities. Single infections represent (30.4%), as double infections accounted for (17.4%), while both triple and quadruple infections were observed in (8.7%) each.

Figure 2. presents the distribution of Transplacental Congenital Pathogens infection patterns in babies' cord blood.

The majority of neonates had **no detectable infection (56.5%)**. The level of infection also decreased as the comorbidity increased. **Single infections were observed in (21.7%)** of the cases. **Double infections accounted for (13%)**, while both **triple and quadruple infections were less frequent, occurring in (4.3%) each**.

Table 1. Pairwise Associations (Chi-square p-values) Among Pathogens in Pregnant Women

Pathogens	Cytomegalovirus	Herpes Simplex Virus	<i>Toxoplasma gondii</i>	<i>Plasmodium falciparum</i>	Rubella	<i>Treponema pallidum</i>
Cytomegalovirus		0.000	0.623	0.000	0.055	0.055
Herpes Simplex Virus	0.000		0.105	0.001	0.075	0.075
<i>Toxoplasma gondii</i>	0.623	0.105		0.899	0.027	0.027
<i>Plasmodium falciparum</i>	0.000	0.001	0.899		0.000	0.000
Rubella	0.055	0.075	0.027	0.000		0.000
<i>Treponema pallidum</i>	0.055	0.075	0.027	0.000	0.000	

Table 2. Pairwise Associations (Chi-square p-values) Among Pathogens in Neonates

Pathogens	Cytomegalovirus	Herpes Simplex Virus	<i>Toxoplasma gondii</i>	<i>Plasmodium falciparum</i>	Rubella,	<i>Treponema pallidum</i>
Cytomegalovirus		0.000	0.105	0.000	0.105	0.105
Herpes Simplex Virus	0.000		0.027	0.000	0.004	0.004
<i>Toxoplasma gondii</i>	0.105	0.027		0.055	0.196	0.196
<i>Plasmodium falciparum</i>	0.000	0.000	0.055		0.000	0.000
Rubella	0.105	0.004	0.196	0.000		0.000
<i>Treponema pallidum</i>	0.105	0.004	0.000	0.000	0.000	

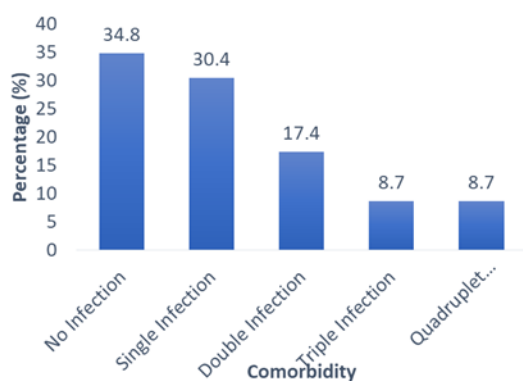


Figure 1. Maternal comorbidity pattern

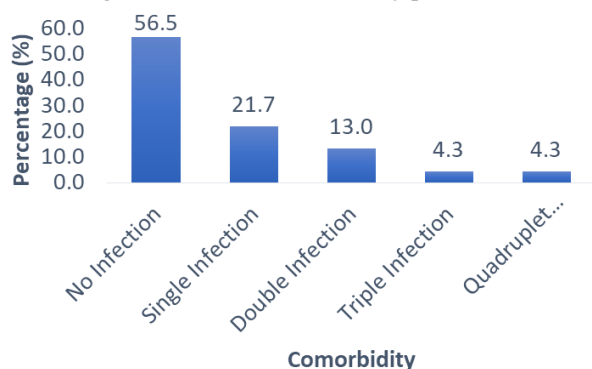


Figure 2. Neonatal comorbidity pattern

Discussion

The present study demonstrates a substantial burden and notable comorbidity of transplacental congenital pathogen detection among mother–neonate pairs in Anyigba, Kogi State, Nigeria. The progressive reduction in frequency with increasing numbers of concurrent pathogens is epidemiologically plausible because simultaneous exposure to multiple organisms is less likely than exposure to a single pathogen. Nevertheless, the substantial proportion of mothers with multiple infections highlights the importance of considering congenital infections as a potentially complex infectious burden rather than isolated pathogen-specific events.

The significant pairwise associations between CMV/HSV, CMV/*P. falciparum*, and *P. falciparum*/Rubella/treponema pallidum are indeed unlikely to represent direct biological synergy. Thus, the observed statistical associations are more likely due to shared risk factors, immune status influences, or co-endemicity rather than direct biological cooperation or synergy between these diverse pathogens (Gangemi *et al.*, 2021). This epidemiological finding suggests that public health interventions should include education and screening for other pathogens that cluster in the same vulnerable populations as the pathogens studied are not merely opportunistic invaders but active agents that reshape the metabolic and nutritional landscape of the mother and her unborn child, with consequences that likely extend far beyond birth.

The maternal detection rate is consistent with evidence that several congenital pathogens are common among Nigerian women. For instance, (Karshima and Karshima, 2020) reported a pooled *Toxoplasma gondii* prevalence of 40.25% among pregnant women in Nigeria, while Bigna *et al.* (2020) demonstrated substantial geographical variation in *T. gondii* seroprevalence globally. These findings provide an epidemiological context for the high maternal pathogen burden observed in the present study. However, direct numerical comparison should be made cautiously because previous studies frequently relied on serology and examined individual pathogens, whereas the present investigation employed molecular detection of multiple pathogens simultaneously.

The lower frequency of pathogen detection in cord blood compared with maternal blood is biologically tenable and may reflect the protective role of the placenta. Maternal infection does not inevitably result in fetal infection because vertical transmission depends on pathogen characteristics, maternal immune status, gestational age, placental susceptibility, pathogen burden, and the integrity of the maternal–fetal interface (Creisher & Klein, 2024). The placenta can therefore act as both a barrier and a site of pathogen-induced inflammation and tissue injury. Importantly, detection of pathogen DNA in cord blood should be interpreted as molecular evidence of fetal/neonatal exposure rather than automatically as clinically manifest congenital disease. Pathogen-specific diagnostic algorithms may require additional specimens and confirmatory testing.

The occurrence of double, triple, and quadruple infections in both mothers and neonates is particularly significant. Multiple infections may potentially produce overlapping inflammatory and immunological effects at the placenta, thereby affecting trophoblast function, maternal–fetal exchange, and fetal development. Such mechanisms provide a plausible basis for investigating whether pathogen comorbidity is associated with altered micronutrient homeostasis, particularly because infection and inflammation can modify nutrient utilization and distribution. However, causality cannot be inferred from the present cross-sectional findings.

Overall, these results provide important baseline molecular evidence of transplacental pathogen comorbidity in Kogi State and support integrated antenatal screening, improved molecular diagnostic capacity, maternal health education, appropriate vaccination and targeted neonatal follow-up. Future multicentre longitudinal studies should identify specific pathogen combinations and determine their relationships with micronutrient status, birth outcomes and long-term neurodevelopment.

References

- Adeniyi, O. A., Adeyemo, A. A., and Odusanya, O. (2021). Maternal infections and perinatal outcomes in Lagos, Nigeria. *African Journal of Reproductive Health*, 25(4), 73–81
- Akor, S. E., Onoja-Alexander, M. O., Agbanna, B. E., Musa, D. A., Priscilla, O. O., Agama, O. G., and

- Faith, F. J.; Infectious diseases co-morbidities among patients attending Kogi State University Teaching Hospital: a ten-year retrospective study. *African Journal of Clinical and Experimental Microbiology*. ISSN 1595-689X Apr 2022; Vol.23 No.2 AJCEM/2160. <https://www.ajol.info/index.php/ajcem>
3. Bigna, J. J., Tochie, J. N., Tounouga, D. N., Bekolo, A. O., Ymele, N. S., Youda, E. L., Sime, P. S., & Nansseu, J. R. (2020). Global, regional, and country seroprevalence of *Toxoplasma gondii* in pregnant women: A systematic review, modelling and meta-analysis. *Scientific Reports*, 10, 12102. <https://doi.org/10.1038/s41598-020-69078-9>
 4. Creisher, P. S., & Klein, S. L. (2024). Pathogenesis of viral infections during pregnancy. *Clinical Microbiology Reviews*, 37(2). <https://doi.org/10.1128/cmr.00073-23>
 5. Evans-Gilbert, T. (2020). Vertically transmitted chikungunya, Zika and dengue virus infections: The pathogenesis from mother to fetus and the implications of co-infections and vaccine development. *International Journal of Pediatrics and Adolescent Medicine*, 7(3), 107–111.
 6. Gangemi, A. C., Choi, S. H., Yin, Z., and Feurdean, M. (2021). Cytomegalovirus and Herpes Simplex Virus Co-Infection in an HIV-Negative Patient: A Case Report. *Cureus*, 13(2), e13214.
 7. Karshima, S. N., & Karshima, M. N. (2020). Human *Toxoplasma gondii* infection in Nigeria: A systematic review and meta-analysis of data published between 1960 and 2019. *BMC Public Health*, 20, 877. <https://doi.org/10.1186/s12889-020-09015-7>
 8. Türkmen Albayrak, H. (2020). Evaluation of *Toxoplasma gondii*, rubella virus and cytomegalovirus infections in pregnant women. *The Anatolian Journal of Family Medicine*.
 9. Ubesie, A. C, Cole, C. R, Nathan, J. D, Tiao, G.M, Alonso, M.H, Mezzoff, A.G, Henderson, C.J, and Kocoshis, S.A. (2013). Micronutrient deficiencies in pediatric and young adult intestinal transplant patients. *Pediatr Transplant.*,17(7):638-45