

The seroprevalence of human CMV among children aged 1-5 years

Pakiza Fouad Mohammed*¹, Nuha Mumin Wahid², Dunya Mahmoud Abd Alrahman³,
and Zaid Mohammed Al-Bayati^{4,5}

¹Microbiology, Al-Kitab University, Iraq

²Kirkuk Health Directorate Kirkuk Teaching Hospital, Kirkuk, Iraq

³University of Kirkuk, College of Pharmacy, Kirkuk, Iraq

⁴University of Liverpool, Liverpool, UK

⁵Azadi Teaching Hospital, Kirkuk, Iraq

ABSTRACT

Background: Most children with congenital CMV are born to women who have cytomegalovirus. The most likely way for a child to become disabled from a congenital infection is if their mother had it when she was pregnant and was not already infected with CMV.

Objectives: to furnish the inaugural national estimate of cytomegalovirus seropositivity among Iraqi children under five years of age and to evaluate the levels of BAFF and VCAM-1 in children infected with CMV.

Materials and Methods: involved a total of 90 children with CMV and 90 healthy individuals, all aged between 1 and 5 years. Five milliliters of blood were obtained via venipuncture using a 5 ml disposable syringe from each participant in this study. Serum from both patients and controls was analyzed using enzyme-linked immunosorbent assays (ELISA) for CMV IgM and IgG, BAFF, and VCAM-1 in accordance with the manufacturer's instructions.

Results: The current data indicate a markedly reduced mean age in CMV-infected patients (2.18 ± 0.84 years). This signifies that younger children are disproportionately impacted by CMV. The current investigation demonstrated a CMV ELISA seroprevalence of 7(7.78%) for IgM positive patients and 13(14.44%) for those positive for both IgG and IgM. Additionally, it indicated significantly increased levels of B-cell Activating Factor (BAFF) and vascular cell

adhesion molecule-1 (VCAM-1) in CMV-infected pediatric patients relative to controls. The study indicated that CMV may enhance long-term vascular risk through immune-mediated endothelial stress.

Keywords: CMV, cCMV, BAFF, VCAM-1, IUGR, HCMV.

1. Introduction

The *Herpesviridae* family of viruses includes human cytomegalovirus (CMV), which is always present but only affects certain people. The organism has a large double-stranded DNA genome of 236 kb that codes for at least 167 gene products. More than 40 of these gene products change the host's immune response after infection. After the first infection, HCMV causes a long-lasting, latent infection by using a number of methods to avoid the immune response. Human Cytomegalovirus is the most common cause of congenital viral infection. It can cause intrauterine growth retardation (IUGR) and hearing, vision, and other neurological problems in fetuses and infants. The most common effect is sensorineural hearing loss. Congenital CMV (cCMV) can happen when a mother is infected with the virus for the first time or when she is infected again with a different strain of the virus. This can happen when the virus is reactivated or when the mother gets a new strain of the virus (1). One of the eight human herpesviruses is human cytomegalovirus. Herpesviruses are very common in animals and can cause serious illnesses in both babies and adults. These include cold sores HSV-1, sexually transmitted infections (HSV-2), and cancers (Epstein-Barr virus, and Kaposi's sarcoma-associated herpesvirus, KSHV) (2). Congenital CMV is among the most common congenital infections worldwide.

Among 85%–90% of cCMV-infected neonates who are asymptomatic at birth, 10%–15% later exhibit sequelae (3), cCMV infection is the most common congenital infection, affecting about 1 in 200 babies in wealthy areas. About one in four children will have lasting effects, such as sensorineural hearing loss and neuro-disability, cCMV is common and can be quite serious for some children, but not many pregnant women, families, or healthcare providers know about it. It is very important to find out about a CMV infection during pregnancy as soon as possible so that valaciclovir treatment can be evaluated. This may lower the risk of fetal transmission or make the results for infected infants less severe. It is very important for neonatologists, pediatricians, and audiologists to know what congenital CMV looks like so they can start testing for cCMV in the first 21 days of life (4). HCMV is passed from mother to kid in utero, during labor, and through nursing. The primary modes of infection include sexual transmission and exposure to bodily fluids, including semen, cervical or vaginal secretions, urine, and blood. HCMV can also be transferred via saliva and breast milk postnatally (5). Similar to all other herpesviruses, HCMV maintains permanent latency after an initial phase of lytic replication. Latency can be interrupted by sporadic episodes of viral secretion. HCMV is the most genetically intricate virus among all human pathogenic viruses, with infection marked by

Citation: Pakiza Fouad Mohammed, Nuha Mumin Wahid, Dunya Mahmoud Abd Alrahman, and Zaid Mohammed Al-Bayati (2026). The seroprevalence of human CMV among children aged 1-5 years.

Journal of American Medical Science and Research.

DOI: <https://doi.org/10.51470/AMSR.2026.05.01.38>

Revised 16 January 2026

Received 15 February 2026

Accepted 06 March 2026

Corresponding Author: **Pakiza Fouad Mohammed**
Email Address: pakizamohammed@uoalkitab.edu.iq

Copyright: © The Author(s) 2026. This article is Open Access under a Creative Commons Attribution 4.0 International License, allowing use, sharing, adaptation, and distribution with appropriate credit. License details: <http://creativecommons.org/licenses/by/4.0/>. Data is under the CC0 Public Domain Dedication (<http://creativecommons.org/publicdomain/zero/1.0/>) unless otherwise stated.

tight species specificity and an extended replication cycle in both infected hosts and cell cultures(6). Maternal infections can cause intrauterine CMV transmission in both primary (when a woman who was not previously infected with CMV becomes infected) and non-primary (when a woman who was already infected with CMV becomes infected again) ways. The percentage of cCMV infections caused by primary or non-primary maternal infections depends on the CMV seroprevalence in the pregnant women being studied. However, countries with high CMV seroprevalence have higher rates of cCMV infection (7). CMV can be passed from the mother to the fetus by the vertical transmission, can occur via three mechanisms: intrauterine, intrapartum, and postnatal (8). Horizontal transmission of CMV between individuals can serve as a substantial source of infection. About one in three children in the United States are infected with CMV by the age of five, with a higher prevalence among those who attend daycare. Young children may serve as a vector for infection to pregnant women. While less prevalent, transmission through sexual contact with an infected individual is nevertheless conceivable (9). People who have chronic infections with human cytomegalovirus (HCMV) have weak immune systems and are more likely to have long-term inflammation and heart disease. A homologue of the anti-inflammatory cytokine encoded by HCMV may change the effects (10). The B cell-activating factor receptor (BAFFR) is very important for B cells to mature. People with common variable immunodeficiency, which is the most frequent symptomatic primary immunodeficiency, have been found to be missing BAFFR. People who don't have BAFFR have a lot of B cell lymphopenia and can't make immunoglobulins (11). B cell-activating factor (BAFF) is involved in the causes of autoimmune diseases (12). In the early stages of life, babies are more likely to get serious viral infections and their antibody responses are weaker, thus local airway immunity needs to develop quickly. Still, we don't know enough about how human airway B-cells form and how they interact with airway epithelial cells (AECs) in early life (13). The vascular endothelium, an essential modulator of hemostasis, promotes vascular dilatation, inhibits adhesion of platelet and diminishes thrombin generation. Endothelial dysfunction caused by acute or chronic inflammation, as seen in atherosclerosis. Persistent infection with human cytomegalovirus (HCMV) has been associated with atherosclerosis. In vivo studies have shown that HCMV infection of the vascular wall affects various cell types, including monocytes/macrophages, smooth muscle cells (SMCs), and endothelial cells (ECs). Human cytomegalovirus-infected smooth muscle cells in vascular lesions demonstrate enhanced proliferation and reduced apoptosis, leading to plaque formation, and restenosis (14).

2. Methodology

2.1. Study setting and population

A cross-sectional study was conducted in Kirkuk city from December 15, 2024, to June 15, 2025. A study included 90 children with CMV and 90 healthy participants aged 1 to 5 years, with the patients exhibiting varying degrees of symptoms. The research project received ethical approval from the Ethical Decision Committee of the College of Health. Verbal consent was acquired from each patient following their decision to participate in the study.

2.2. Patients Methodology

Five milliliters of blood were obtained via venipuncture using a 5 ml disposable syringe from each participant in the present investigation. Blood samples were deposited in sterile test tubes and after thirty minutes of incubation, the sample was spun at 3000 rpm for fifteen minutes. After that, the clot was taken out, and the resultant sera were aspirated using an automated micropipette and transferred into two clean Eppendorf tubes. The label was affixed to each Eppendorf tube, after which the serum was removed and stored at -20°C. Serum from both the patient and control groups was analyzed using enzyme-linked immunosorbent assays (ELISA) for CMV IgM and IgG, Human B-cell activation factor (BAFF), and Human vascular cell adhesion molecule-1 (VCAM-1) following the manufacturer's instructions (SUNLUNG/Biotech, Ltd; China).

2.3. Statistical analysis

The statistical analysis was conducted using Graph Pad Prism version 10.1. The comparison utilized the Chi-square (χ^2) test, and the t-test was employed as necessary to compare groups, with a probability (P value) of less than 0.05 deemed statistically significant (S), a P value below 0.01 classified as highly significant (H.S.), and a P value exceeding 0.05 regarded as non-significant (N.S) (15).

3. Results

The current study indicates that CMV infection primarily impacts children under one year of age, with 45.56% of patients in this demographic, in contrast to 28.89% in the control group. This pronounced disparity indicates a significant vulnerability to CMV infection during infancy, consistent with global and regional patterns.

Table 1: The distribution of study group according to Age group

Age group	Patients		Control	
	No.	%	No.	%
<1	41	45.56	26	28.89
1-2	25	27.78	34	37.78
3-4	14	15.56	17	18.89
4-5	10	11.11	13	14.44
Total	90	100.00	90	100.00

Our data reveal a significantly lower mean age in CMV-infected patients (2.18 ± 0.84 years) compared to healthy controls (3.06 ± 1.06 years). This indicates that younger children, particularly those under three years of age, are disproportionately affected by CMV, underscoring that early childhood, especially infancy, is a critical phase for CMV exposure and infection, as illustrated in Table 2. In the last five years, a number of nationally representative cross-sectional studies with big enough sample sizes have looked at how often children get HCMV using either serological or molecular methods. A study in the US found that 1.1% of children aged one to five had IgM and 20.7% had IgG (16). A study in Germany found that 27% of toddlers and teens aged one to seventeen have HCMV-specific IgG -(17). A different study done in the Netherlands found that 8% of children aged three to five with hearing problems had HCMV infection (18).

Table 2: Mean age of study group

Age	Patients		Control	
	Mean \pm SD		Mean \pm SD	
	2.18 \pm 0.84		3.06 \pm 1.06	
Total	90		90	

This study presents a comparative profile of cases and controls categorized by gender and residency, with each group consisting of 90 patients and 90 controls. The gender distribution in the cases indicates a somewhat greater proportion of females than men, while the control group exhibits a predominance of males. The disparity may indicate possible gender-related exposure or vulnerability, maybe attributed to behavioral, occupational, or biological influences. The current finding corresponds with specific epidemiological studies; Goodwin et al. (2023) observed that females may demonstrate greater health-seeking behavior, hence being more frequently represented in diagnostic "case" groups for various chronic diseases(19). Conversely, males frequently have greater representation in control groups owing to reduced healthcare engagement or varying risk profiles for particular diseases(20). The distribution of dwellings revealed that 62.22% of CMV patients resided in urban regions, as shown in Table 3. HCMV infection demonstrates considerable regional heterogeneity within nations. Preliminary study findings suggest no association between place of residence and the incidence of HCMV infection in children under five years in Iraq, maybe reflecting a homogeneity within the Iraqi population (21). The urban domination in both categories is anticipated owing to elevated urban population density and enhanced access to healthcare services. The increased rural presence among cases, in contrast to controls, may indicate health inequities, including less access to preventive treatment, heightened exposure to environmental hazards, or delayed diagnosis for rural inhabitants (21). This aligns with data indicating that rural populations frequently encounter poorer health outcomes and diminished access to healthcare (22). Furthermore, health disparities between urban and rural areas have intensified in recent years owing to the unequal distribution of healthcare facilities (23). All studies indicated a large disparity in HCMV infection based on geographical variability, which corresponds to racial and ethnic differences within countries. Ethnic and ethnic diversity underscores variations in the determinants affecting HCMV transmission. Examples encompass the duration and frequency of breastfeeding, attendance at daycare, and childcare arrangements(24).

Table 3: Sociodemographic characteristics of the study population

Participant profile		Cases		Control	
		No	%	No	%
Gender	Male	41	45.56	52	57.78
	Female	49	54.44	38	42.22
Total		90	100.00	90	100.00
Residency	Rural	34	37.78	21	23.33
	Urban	56	62.22	69	76.67
Total		90	100.00	90	100.00

The current table displays clinical signs in 90 CMV-positive pediatric patients, of whom 52 (57.78%) demonstrated symptoms. The most prevalent symptoms were lack of appetite (24.44%), followed by fatigue (16.67%), fever (13.33%), and hepatomegaly (3.33%). It's important to remember that all of the children who were called "symptomatic" showed the usual signs and symptoms of overt HCMV congenital and acquired infections when the samples were taken. In a previous study, 15.9% of unwell kids tested positive for particular IgM (21). Habib et al. reported a finding of around 16.1% in symptomatic newborns in Baghdad utilizing specific IgM for detection (25).

Positive IgM frequencies have been recorded at 11.7% among symptomatic children in Palestine (26) and 1.6% of symptomatic newborns in Iran (27).

Table 4: The relation of HCMV positive patients according to symptomatic cases

CMV Symptoms	Patients	
	No	%
Fever	12	13.33
Loss of appetite	22	24.44
Fatigue	15	16.67
Hepatomegaly	3	3.33
Total	52	57.78

The CMV ELISA seroprevalence indicated that 7.78% of patients tested positive for IgM, whereas 14.44% tested positive for both IgG and IgM and absent in healthy control, as illustrated in Table 5. The current findings correspond with a recent case-control study of children with autoimmune and neurological disorders, which identified significantly elevated CMV IgG and IgM positive in patients compared to controls (IgM positivity was 13.2% in patients, akin to the present research's 14.44%). In Kirkuk, Iraq, Al-jumaili et al. (2013) showed that IgM levels were considerably elevated (7.2%) in women compared to control groups (5.3%) (28). Tuma et al. found that 12.4% of women in Baghdad, Iraq, have HCMV IgM antibodies (29). The current HCMV infection in women with a poor obstetric history (BOH) is more likely to serve as a source of infection for their children during pregnancy. Nonetheless, not all contemporary maternal infections result in fetal transmission (30). There is proof that viruses can spread during pregnancy and through breast milk. Also, there is a chance that women who are currently infected could pass the virus on to their newborns and young children through close contact (21).

Table 5: Distribution of Human Cytomegalovirus antibodies among studied groups

CMV ELISA	Patients		Control	
	No	%	No	%
IgM+IgG-	7	7.78	0	0.00
IgM+IgG+	13	14.44	0	0.00
IgM-gG+	24	26.67	5	5.56
IgM-IgG-	46	51.11	85	94.44
Total	90	100.00	90	100.00

Table 6 demonstrates markedly increased concentrations of BAFF and VCAM-1 in pediatric patients infected with CMV relative to controls. These results indicate immune system activation and inflammation, consistent with the established pathophysiology of CMV. A recent study indicated markedly increased levels of ICAM-1 and VCAM-1 in children with acute CMV, identifying these markers as predictors of endothelium damage, particularly in newborns (31). Another study identified increased BAFF levels in children with CMV and EBV, associated with lymphocytosis and hypergammaglobulinemia. BAFF is proposed as a biomarker for early viral reactivation and immunological dysregulation, especially in immunocompromised individuals (32). Yoshida et al. observed elevated levels of BAFF and ICAM-1 during active CMV replication following transplantation. These elevations, restored with antiviral medication, are associated with active viral burden rather than the host's baseline immunity (33-35).

Table 6: Comparison of markers of BAFF and VCAM-1 dysfunction between CMV patients and control

Biomarker	Patients n= (90)			Control n= (90)	P- value
	Mean	SD	Mean	SD	
ICAM-1, ng/mL	186.27	69.41	42.33	19.72	0.0028
BAFF, ng/mL	131.04	43.86	47.89	11.63	0.0095

4. Conclusions

The current study concluded that CMV may promote long-term vascular risk via immune-mediated endothelial stress.

5. References

- Njue A, Coyne C, Margulis AV, Wang D, Marks MA, Russell K, et al. The role of congenital cytomegalovirus infection in adverse birth outcomes: a review of the potential mechanisms. *Viruses*. 2020;13(1):20.
- Pass RF. Human herpesviruses: cytomegalovirus. In: *Viral Infections of Humans: Epidemiology and Control*. Springer; 2022. p. 1–49.
- Smyrli A, Raveendran V, Walter S, Pagarkar W, Field N, Kadambari S, et al. What are the neurodevelopmental outcomes of children with asymptomatic congenital cytomegalovirus infection at birth? A systematic literature review. *Rev Med Virol*. 2024;34(4):e2555.
- Jones CE, Bailey H, Bamford A, Calvert A, Dorey RB, Drysdale SB, et al. Managing challenges in congenital CMV: current thinking. *Arch Dis Child*. 2023;108(8):601–7.
- Xia W, Yan H, Zhang Y, Wang C, Gao W, Lv C, et al. Congenital human cytomegalovirus infection inducing sensorineural hearing loss. *Front Microbiol*. 2021;12:649690.
- Spindler N, Diestel U, Stump JD, Wieggers AK, Winkler TH, Sticht H, et al. Structural basis for the recognition of human cytomegalovirus glycoprotein B by a neutralizing human antibody. *PLoS Pathog*. 2014 Oct;10(10):e1004377.
- Novelli M, Natale F, Di Norcia A, Boiani A, Temofonte S, Calandriello F, et al. Early neurodevelopmental outcomes in children with asymptomatic congenital CMV infection. *Ital J Pediatr*. 2022;48(1):203.
- Chiopris G, Veronese P, Cusenza F, Procaccianti M, Perrone S, Daccò V, et al. Congenital cytomegalovirus infection: update on diagnosis and treatment. *Microorganisms*. 2020;8(10):1516.
- Andronaco DW. Congenital cytomegalovirus and hearing loss. *J Obstet Gynecol Neonatal Nurs*. 2020;49(3):293–304.
- Zhang XJ, Zhang JX, Qu Y, Peng RM, Zhang P, Hong J. Cytokine analysis of aqueous humor in patients with cytomegalovirus corneal endotheliitis. *Graefes Arch Clin Exp Ophthalmol*. 2024;262(8):2593–600.
- Xu HC, Huang J, Khairnar V, Duhan V, Pandya AA, Grusdat M, et al. Deficiency of the B cell-activating factor receptor results in limited CD169+ macrophage function during viral infection. *J Virol*. 2015;89(9):4748–59.
- Giordano D, Kuley R, Draves KE, Elkon KB, Giltiy NV, Clark EA. B cell-activating factor (BAFF) from dendritic cells, monocytes and neutrophils is required for B cell maturation and autoantibody production in SLE-like autoimmune disease. *Front Immunol*. 2023;14:1050528.
- Chorvinsky E, Surajit Bhattacharya, Salka K, Bera BS, Welham A, Mondell E, et al. Human Airway Epithelial BAFF is Reduced in Early Life but Virally-induced via JAK/STAT. *J Allergy Clin Immunol*. 2025.
- Popović M, Smiljanić K, Dobutović B, Syrovets T, Simmet T, Isenović ER. Human cytomegalovirus infection and atherothrombosis. *J Thromb Thrombolysis*. 2012;33:160–72.
- Albarzanji ZNM, Wahid NM, Shaker NB, Al-Bayati ZM. Relation of ICAM-1 and VCAM-1 markers in COVID-19 patients in Kirkuk province. *Hum Antibodies*. 2024;32(4):213–20.
- Lanzieri TM, Kruszon-Moran D, Amin MM, Bialek SR, Cannon MJ, Carroll MD, et al. Seroprevalence of cytomegalovirus among children 1 to 5 years of age in the United States from the National Health and Nutrition Examination Survey of 2011 to 2012. *Clin Vaccine Immunol*. 2015;22(2):245–7.
- Voigt S, Schaffrath Rosario A, Mankertz A. Cytomegalovirus seroprevalence among children and adolescents in Germany: data from the German Health Interview and Examination Survey for Children and Adolescents (KiGGS), 2003–2006. *Open forum infectious diseases*. Oxford University Press; 2016. p. ofv193.
- De Vries JJC, Korver AMH, Verkerk PH, Rusman L, Claas ECJ, Loeber JG, et al. Congenital cytomegalovirus infection in the Netherlands: birth prevalence and risk factors. *J Med Virol*. 2011;83(10):1777–82.
- Goodwin CR, Price M, Goodwin AN, Dalton T, Versteeg AL, Sahgal A, et al. Gender and Sex Differences in Health-Related Quality of Life, Clinical Outcomes and Survival after Treatment of Metastatic Spine Disease. *Spine (Phila Pa 1976)*. 2023.
- Abdullah NN, Arsat MHM, Aziz NRA, Al-Kubaisy W. Men health seeking behaviour: a literature review. *Environ Proc J*. 2022;7(20):247–54.
- Alwan SN, Al-Saffar AJ, Bayati AH, Kadhim HS, Arif HS, Ghaib AH, et al. Prevalence of cytomegalovirus in Iraqi children. *Int J Med Res Heal Sci*. 2017;6(11):113–24.
- Kaye LW. *Handbook of rural aging*. Routledge; 2021.
- Turner J. Reducing Rural Disparities: Revitalizing Rural Healthcare for the Elderly. *Elder Law J*. 2024;32(2).
- Fowler KB, Ross SA, Shimamura M, Ahmed A, Palmer AL, Michaels MG, et al. Racial and ethnic differences in the prevalence of congenital cytomegalovirus infection. *J Pediatr*. 2018;200:196–201.
- Habib MA, Al-Omar LS, Sameh H. Prevalence of HCMV infection among Iraqi infants. *Iraqi J Med Sci*. 2003;2:76–82.
- Neirukh T, Qaisi A, Saleh N, Rmaileh AA, Zahriyeh EA, Qurei L, et al. Seroprevalence of Cytomegalovirus among pregnant women and hospitalized children in Palestine. *BMC Infect Dis*. 2013;13:1–7.
- Golalipour MJ, Khodabakhshi B, Ghaemi E. Possible role of TORCH agents in congenital malformations in Gorgan, northern Islamic Republic of Iran. *East Mediterr Heal J*. 2009;15(2).
- Aljumaili ZKM, Alsamara AM, Najem WS. Cytomegalovirus seroprevalence in women with bad obstetric history in Kirkuk, Iraq. *J Infect Public Health*. 2014;7(4):277–88.
- Tuma FL, Fadhil HY, Moayad D, Anor M, Al-Hamdani FG. Survey for CMV, HSV-2 Infections and their Association with Congenital Anomalies, Baghdad. *Int J Adv Res*. 2013;1(10):310–6.
- Mokhtar SY, Elhag WI. Serofrequency of cytomegalovirus infection in women with bad obstetric history attending routine antenatal clinic at Omdurman Military Hospital. *Eur Acad Res*. 2015;3(6):6270–82.
- Palomo M, Moreno-Castaño AB, Salas MQ, Escibano-Serrat S, Rovira M, Guillen-Olmos E, et al. Endothelial activation and damage as a common pathological substrate in different pathologies and cell therapy complications. *Front Med*. 2023;10:1285898.
- Högelin KA. *Multiple Sclerosis, Viruses, and B Cell Depleting Therapy; A Meeting at the Immune System*. Karolinska Institutet (Sweden); 2024.
- Yoshida M, Yokoyama Y, Kokubun T, Tsuda S, Himori N, Maekawa S, et al. Long-Term Surgical Outcomes and Possible Postoperative Complication with Severe Corneal Endothelial Cell Loss After Trabeculectomy for Cytomegalovirus-Associated Anterior Uveitis with Secondary Glaucoma. *Ocul Immunol Inflamm*. 2024;32(5):690–8.
- Qader SM, Al-Azzawy MA, Tawfiq SK. Possible roles of human cytomegalovirus immunoglobulin G and its avidity to specific human cytomegalovirus antigens in the prevention of abortion among pregnant women. *Medical Journal of Babylon*. 2023;20(S1):S41-S47.
- Rashid HM, Rasheed MA, Omer ZA. Traumatic Head Injuries Among Children Admitted to Azadi Teaching Hospital in Kirkuk/Iraq. *Bahrain Medical Bulletin*. 2025;47(3).