Short Communication

Evaluation of Some Immunological Markers in Pregnant Women Infected with Cytomegalovirus in Kirkuk, Iraq

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Abstract

Background: Both innate and adaptive immunological responses may be activated by cytomegalovirus (CMV) infection, leading to significant reactions later in the course of the illness. Innate immune components, which react fast and nonspecifically, combine intricately with specialized immune system components, which may recognize certain antigen epitopes, to initiate immunological responses. In the adaptive immune response, T and B lymphocytes, which are considered more specific and take a few days to become fully activated, were the most prevalent cells. **Objectives:** The aims of this study were to evaluate some immunological markers in CMV-infected pregnant women compared with a control group in Kirkuk City. **Materials and Methods:** Our techniques attempted to estimate the immunological markers CD28, CD80, and CD86. RNA was extracted from blood samples taken from 180 CMV patients and a control group to analyze this marker's gene expression using PCR. **Results:** The sick group displayed significant differences in the immunological markers when compared to the control group for the CD28 and CD80 tests with (*P* value of 0.0007 and 0.0007), respectively. The CD86 test also appeared to demonstrate a significant difference with (*P* value of 0.0007). **Conclusions:** The results of this investigation showed that patients with CMV had considerably higher levels of several immunological markers when compared to the control group.

Keywords: CD28, CD80, CD86, cytomegalovirus, pregnant women

Introduction

The cytomegalovirus (CMV) is known as human betaherpesvirus 5, also called human CMV (HCMV).[1] Belongs to the Herpesviridae family, and the majority of CMV infections are benign, but the virus can produce a variety of symptoms, particularly in immune-compromised individuals; the infection is widespread throughout the world. CMV transmission requires close interaction with contaminated secretions, which happens through bodily fluids and also from infected mother to fetus during pregnancy.[2] If women are informed about CMV transmission and practice preventative hygiene, they can significantly reduce their risk of developing primary CMV infection and possibly reinfections as well.[3] Congenital CMV infection can result from the transfer of the virus to the fetus from the infected mother, and it is the most typical intrauterine infection and the major cause of infectious impairment. Sexual activity and interaction with children are the two main causes of primary maternal CMV infection.[4] Children's hearing loss and problems with their central nervous systems are frequently caused by congenital CMV infection. Despite the fact that most infants with congenital CMV infection are healthy at birth and do not experience any aftereffects.[5] Furthermore, this virus can cause congenital heart failure and myocarditis.[6,7] Primary CMV infection typically affects children and young adults and is asymptomatic or a mild, self-limiting illness in immune-competent patients. CMV virus establishes a latent phase mostly within leukocytes,

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specifically mononuclear cells, after clearance from acute infection.[8] A primary CMV infection during pregnancy is more likely to cause fetal loss and symptomatic congenital infection.[9,10] Abortion may be caused by viral infections such as CMV, Rubella virus, Herpes simplex virus or may be due to other causes such as cancer.[11,12] CMV infection is typically asymptomatic; its symptoms include malaise, headache, sore throat, and fatigue. Infectious mononucleosis is the most prevalent clinical ailment among immune-competent individuals. Fever is a common symptom of CMV.[13]

CMV stays dormant in the host during life and rarely reactivates to cause clinical illness, especially in those with weakened immune systems.[14,15] Early myeloid lineage cells are a key location for human CMV latency.[16]

The diagnosis of CMV is based on serological detection of IgG, which represents chronic infection, and IgM, which indicates acute infection, but a positive test result cannot distinguish between a latent infection and an active disease. Therefore, PCR CMV tests are important.[17] In general, the most common test for diagnosing CMV infection in immune-compromised patients is quantitative real-time PCR.[18] Peripheral T cells have several subsets, and among them are regulatory T (Treg) cells, which regulate immune responses; memory T cells, which are formed from prior antigen activation and sustain long-term immunity; and naive T cells, which can react to new antigens. When naive T cells are exposed to an antigen and costimulatory ligands provided by dendritic cells (DC), an immune response is triggered. This results in the production of interleukin 2 (IL-2) and the proliferation and differentiation of effector cells, which spread to various locations to help clear pathogens.[19] Infection with the cytomegaly virus causes a number of powerful cellular immune reactions, starting with innate Natural Killer cells and progressing to adaptive CD4 and CD8 T cells and B cells.[20] T-cell activation requires additional signals, such as signaling via CD28 or other costimulatory receptors.[21] T cells require two signals in order to completely activate. The first signal is the peptide-major histocompatibility complex on antigen-presenting cells (APCs) ligating the T-cell receptor (TCR), and the second signal is produced when CD28 that expressed on the T cell with their ligand (B7.1) and (B7.2) on antigen-presenting cell such as (mature DC, macrophages, and activated B cells).[22] CD80 and CD86 also can bind to another receptor molecule called CTLA4 (cytotoxic T lymphocyte antigen 4). Due to their great homology, CD28 and CTLA4 compete for the CD80 and CD86; due to the greater affinity of CTLA4 for these ligands than CD28 does, it can outcompete CD28 for proteins and block the activity of effector T cells. With regard to stimulating T cells, CD28 and CTLA4 have opposing actions; inhibitory signals are provided by CTLA4, and activating signals are delivered by CD28.[23] Since it is opportunistic and more prevalent in pregnant women, CMV identification is crucial to preventing transmission of the infection to the fetus. This study's objectives were to compare various immunological markers in pregnant CMV-infected women to a control group to determine the degree to which the participant's immune system was affected by this virus.

Material and Methods

Study design

In total, 180 samples from pregnant women in different trimesters of pregnancy aged between 18 and 40 years old (of whom 128 had CMV infection and 52 were in the control group) were obtained.

The study's timing and setting

The study was conducted on patients at Azadi Teaching Hospital from October 2022 to March 2023.

Sample collection

An amount of 5 mL of venous blood was taken from 180 participants; the specimen was separated into two designated tubes (a gel tube and an EDTA tube) afterward. After allowing one portion of the committed samples to coagulate at room temperature, it was centrifuged for 15 min at 4000 rpm. Once the blood was kept in an EDTA tube for later processing and the serum samples were extracted, aliquoted, and put into Eppendorf tubes, the proper patient code was correctly applied. Whole blood was collected to use in PCR to evaluate immunological markers, including CD28, CD80, and CD86.

Statistic assessment

The statistical analysis was performed using GraphPad Prism statistical software.

Ethical approval

The study was conducted in accordance with the ethical principles that have their origin in the Declaration of Helsinki. It was carried out with the patient's verbal and analytical approval before a sample was taken. The study protocol, the subject information, and the consent form were reviewed and approved by a local ethics committee according to document number 48206 (including the number and the date in November 1, 2022) to get this approval.

Result

This study involved the assessment of gene expression of CD28, CD80, and CD86 in CMV patients and the control group. Table 1 reveals the percentage of age in the patient group. Regarding age, divided into three categories: 20 years or below (8.59%), 21–30 years (42.97%), and 40 years or below (48.44%). Concerning the risk factor percentage of patients with diabetes (8.59%) and hypertension (3.13%).

Regarding hematological parameters, our data demonstrate that total white blood cell count was significantly elevated (P value < 0.0001) in the patient group with a mean (9.837) compared to the control group (8.063) when applying the Mann–Whitney t-test as depicted in Figure 1 and Table 2. As far as lymphocyte count is concerned, our research found that the mean level of lymphocytes in the patient group (49.23) is significantly higher than in the control group (39.16) with (P value < 0.0001) when utilizing Mann–Whitney t-test, as demonstrated in Figure 2 and Table 2.

The data displayed here reveals the assessment of gene expression of CD28, CD80, and CD86 for acute CMV infection. Regarding CD28 gene expression, this marker was significantly elevated (mean ± SD = 4.16 ± 3.748) in the CMV positive group compared to control (mean ± SD = 1.0 ± 0.3008) with P = 0.0079 using Mann–Whitney t-test, as depicted in Figure 3 and Table 3.

Table 1: Common characteristics of tested groups

Demographic variables	%
<20	8.59
Age 20 or above	42.97
30 or above	48.44
Diabetes	8.59
Hypertension	3.13

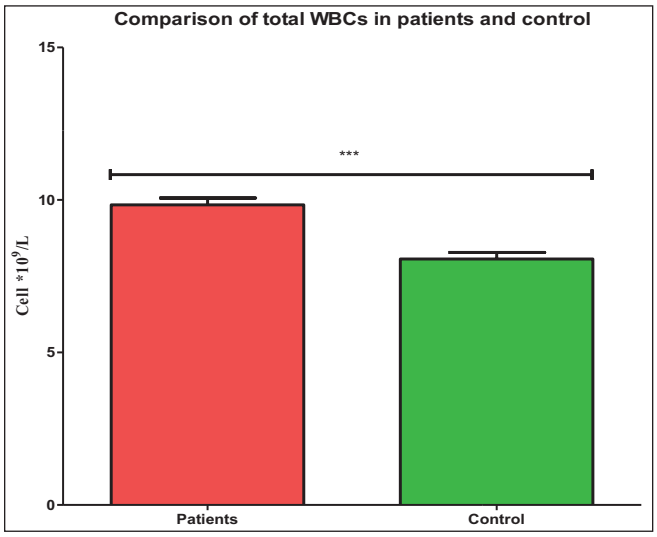

Figure 1: Comparison of total white blood cells in patients and control group

Table 2: Hematological parameter in patient groups

Hematological markers	Patients (mean)	Control (mean)	P value
Total WBC	9.837	8.063	0.0001
Lymphocyte count	49.23	39.16	0.0001

On the other hand, CD80 gene expression has shown a significant increment in the CMV positive group (mean ± SD = 2.08 ± 1.635) compared to non-CMV positive subjects (mean ± SD = 1.0 ± 0.2151), with P = 0.0079

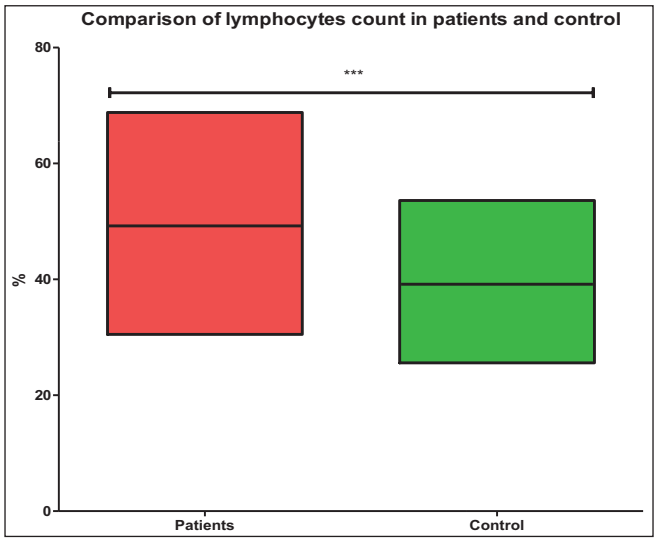

Figure 2: Lymphocyte count in the patient group compared to control group

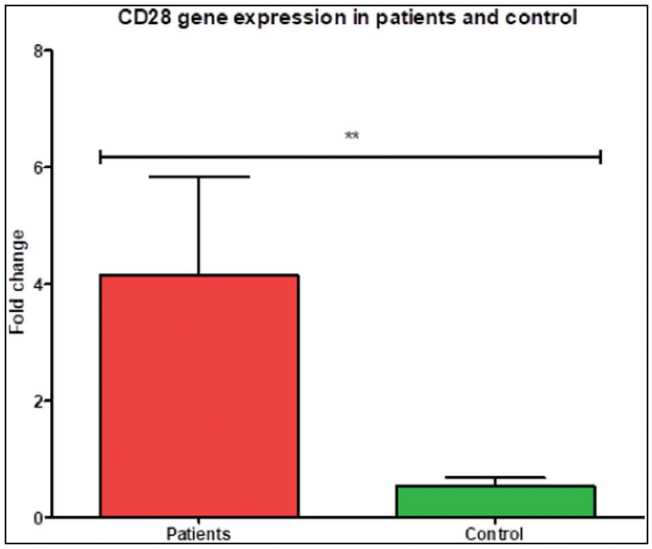

Figure 3: Using the Mann–Whitney t-test, CD28 gene expression was compared between the two groups, with a significant difference between them (P value of 0.0079)

Table 3: Gene expression of three immunological markers in tested group

Immunological marker	Patient	Control
	Mean ± SD	Mean ± SD
CD28	4.16 ± 3.748	1.0 ± 0.3008
CD80	2.08 ± 1.635	1.0 ± 0.2151
CD86	3.33 ± 2.19	1.0 ± 0.10

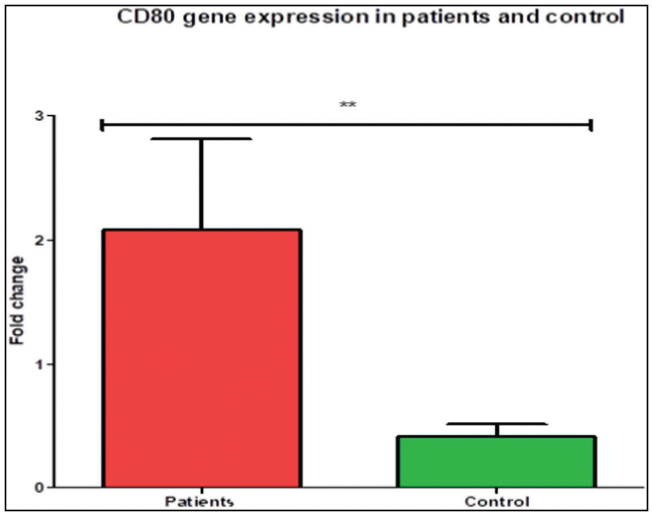

Figure 4: Using the Mann–Whitney *t*-test, CD80 gene expression was compared between the two groups, with a significant difference between them (*P* value of 0.0079)

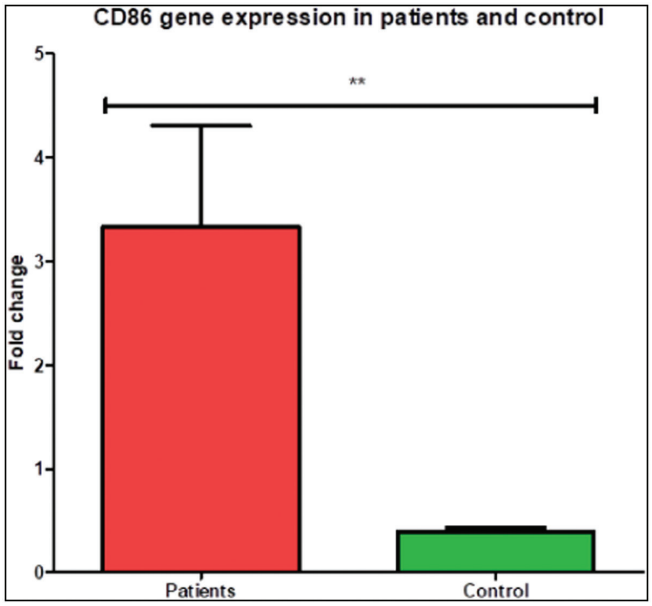

Figure 5: Using the Mann–Whitney *t*-test, CD86 gene expression was compared between the two groups, with a significant difference between them (*P* value of 0.0079)

when applying Mann–Whitney *t*-test, as displayed in Figure 4 and Table 3.

As far as CD86 gene expression, Figure 5 and Table 3 illustrate a significant increase among CMV-positive subjects (mean ± SD = 3.33 ± 2.19) compared to control individuals (mean ± SD = 1.0 ± 0.10).

Discussion

Infection with CMV is more common in pregnant women, and the risk increases with age. A higher rate of CMV infection was noted in the age group over 30 years.[24] Our findings are consistent with those of Lachmann *et al.*[3], who showed a higher frequency of CMV in females over 30 years of age. ADditionally, a different study found that the proportion of infected women over 35 was significantly higher.[25] The findings presented here, however, differ from those of Saleh *et al.*[26], who found an elevated ratio of infection in the age group under 30 years. The reason for this difference may be related to the fact that gynecologists only request CMV infection screening when a woman is pregnant; otherwise, the infection is not found until a condition related to CMV may coexist. ADditionally, due to higher chances of abortion, evaluation of the majority of females 35 and older may be necessary due to age influences on pregnancy outcomes.

A number of viral infections have been linked to an elevated risk of diabetes mellitus. The researchers found that CMV infection is connected with glucose organization or regulatory markers, indicating that CMV infection could be a risk factor for the advancement of diabetic disease.[27] Previous studies investigated the role of CMV as a risk factor for hypertension in relation to the possibility of hypertension in people who have CMV infection. This might be because of the immune reaction carried on by the CMV infection, which might then result in hypertension.[28]

Regarding total WBC count, the result of this study revealed that this parameter was higher in the patient group compared to the control group in research by Zhan *et al.*[29] on 257 CMV patients, they noted that the total WBC count was higher in the patient group when compared to the control group. While Ahmad *et al.*[30] noted that the total WBC count was lower in the immunocompromised patients than in the control group. The total WBC count in our data was significantly higher than the control, which may be related to the fact that chronic CMV infection can cause immune system chronic alerts that signal the proliferation of specific WBC clones, particularly lymphocytes, which act as the body's main defense against foreign substances. On top of this, NK cells and cytotoxic T lymphocytes (CD8) also play a role in this defense.[31] Additionally, the total WBC count in pregnant women increased, probably as a result of the physiological stress that comes with pregnancy.[32]

Regarding lymphocyte count, our data showed that a significant increment in lymphocyte count was noted in CMV-positive women, which agrees with other studies that reported a significant elevation of lymphocytes in CMV-positive participants compared to the non-CMV-positive group.[33-35] While our result conflicts with Chandra *et al.*,[36] who reported a decline in lymphocyte count in pregnant women infected with CMV. The possibility

behind such elevation in lymphocyte count could be due to the activation of viral proliferation, which takes place in certain tissues where the virus remains in hypnotic status. This could especially occur in pregnant women where the immune system is compromised.[37] This leads to increased signaling of interferon-gamma from infected cells to alert the key lymphocyte player to proliferate and be able to control viral replication.[38]

In order for T cells to become activated, APCs must provide two signals. The first signal is generated when receptors of T-cells recognize the antigen that MHC II has delivered. The second signal is formed by the interaction between the CD28 receptor on T cells and the costimulatory ligands CD80 and CD86, which are enhanced by APCs.[39]

Another receptor that binds B7 molecules is called CTLA-4 (cytotoxic T-lymphocyte antigen 4), also known as CD152. Contrary to CD28, CTLA-4 is not always expressed but is instead upregulated on T cells after activation.[40] In addition to being an immunoglobulin, CD80 is a ligand for CTLA4, which is found constitutively on many T cells, as well as on T cells' APCs and their receptors (DC, activated B cells, and macrophages, in particular). All APCs, including DC, macrophages, memory B cells, and others, express CD86, which transmits costimulatory signals that aid T cells in activating and surviving.[41] T lymphocytes are either activated or inhibited depending on how the costimulatory molecules CD80 and CD86 interact with their ligands CD28 and CTLA-4.[42] One prominent feature of the CMV is its capacity to stimulate strong T-cell responses. However, the number of CMV-specific T cells persists or increases over time.[43]

The release of cytokines is another feature activation of T cells, and signals from activation lead to the synthesis of several lymphokine genes, including IL-2, interferon-gamma, GM-CSF, and IL-3.[44] Cellular immunity significantly explains the maintenance of CMV infection control. Approximately within a month after the initial infection, CMV-specific CD4 and CD8 cells predominate the memory cell fraction and are adequate to limit viral replication. Since CMV infection persists for a longer period of time than other persistent human infections, to successfully slow down viral replication over time, a higher proportion of CMV-specific CD8 T cells is required.[45]

It has been shown that the T-cell response is essential for preventing both the onset of acute CMV disease and the reactivation of latent infection. Recent studies have shown that even after the original infection has resolved, seropositive people still have exceptionally high numbers of functional CMV-specific CD8 T cells.[46] One of the most obvious phenotypic aspects of CMV infection is a persistent increase in the proportion and quantity of CD8. CD8 lymphocytosis lasts for several months in immune-competent people while lasting considerably longer in immune-compromised people; variable amounts of cytotoxicity are present in several CD8 T cell subpopulations that express the CD28 T lymphocytes. The fact that CD8 lymphocytes increased in response to CMV and that these cells need costimulatory molecules to activate explains why CD28 increased in CMV-infected individuals as a costimulatory increase with CD8 T-cell increase.[47]

Conclusion

In contrast to patients with CMV and the control group, this study found that the patients group had higher gene expression of immunological proteins CD28, CD80, and CD86. This could be because CMV generates a robust immunological response that results in an increased level of lymphocytes.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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