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The relationship between viral load and miscarriages in pregnant women infected with CMV

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Abstract

A total of 204 blood samples were collected from pregnant women between November 2024 and March 2025, across various age groups and trimesters, to analyze the relationship between cytomegalovirus (CMV) viral load and the risk of miscarriage among pregnant women. The virus was diagnosed based on the measurement of IgM levels by ELISA, and IgG levels were measured to detect past infection among pregnant women. Viral load was measured using quantitative real-time polymerase chain reaction (qRT-PCR). Levels of TNF- α were also measured, as they are an inflammatory cytokine that can be associated with miscarriage.

The results were that the women aged between 18-25 years had the highest viral load and TNF- α levels during the first trimester of pregnancy. The miscarriage rate was also higher among the IgG negative women (48.4%) compared to the IgG positive women (15.9%), supporting an association of primary CMV infection with adverse pregnancy outcome. Levels of TNF- α were related to higher viral load and miscarriage.

These findings suggest that early detection of CMV viral load and immune response markers may play a pivotal part to play in the prevention of miscarriage as well as improved maternal and fetal health outcomes.

Keywords: Cytomegalovirus, pregnancy, miscarriage, IgG, TNF- α , qRT-PCR

Introduction

Cytomegalovirus is one of the largest and most widespread viruses that lie dormant. Most people become infected with cytomegalovirus early in life, and people with sufficient immunity spend many years with this virus ^[1].

This virus is a double-stranded DNA herpes virus characterized by the presence of a lipid envelope. It belongs to the Herpesviridae family and the Betaherpesvirinae subfamily ^[2].

It is called cytomegalovirus because it causes a characteristic enlargement of cells and is characterized by a slow proliferation cycle. It targets epithelial cells, kidneys, and salivary glands, i.e. it targets specific cells ^[3].

It affects more than 60% of adults worldwide, and the infection is usually asymptomatic. The virus is transmitted either through body fluids such as saliva, urine, and vaginal secretions. It may also be transmitted vertically from mother to fetus during recurrent infection or primary infection ^[4].

This virus is important because of its effect on pregnant women, causing miscarriages and dangerous complications for the fetus, such as spontaneous abortion, congenital malformations such as hearing loss and mental retardation, and it may also cause the death of the fetus. In addition, this virus causes complications for the mother, such as Placentitis and pregnancy disorders ^[5].

The rate of mother-to-fetal transmission of the virus may reach 30-40% in primary infections, with a significantly increased risk of miscarriage at high viral loads (>10,000 copies/ml) ^[6].

Despite extensive studies on CMV, the relationship between precise viral load levels and the risk of miscarriage remains unclear, especially in cases without clinical symptoms. Therefore, this study aims to analyze available data to determine whether high viral loads are directly associated with increased miscarriage rates. This may help develop early monitoring and treatment protocols for infected pregnant women.

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Materials and Methods

204 samples were collected from pregnant women at Al-Zahraa Teaching Hospital and Al-Furat Al-Awsat Hospital, as well as private gynecology clinics and private analysis laboratories, from November 2024 to March 2025. The samples were divided according to age (18 to 45 years) and trimesters. Cases infected with other infectious diseases, such as hepatitis B and C, and HIV, were also excluded, as were high-risk pregnancies not associated with CMV infection.

Sample Collection and Storage

5 ml of venous blood was drawn and collected in tubes containing anticoagulant (EDTA). The samples were immediately stored at 4 °C for transport to the laboratory. After separation, the (plasma) was stored at -80 °C.

Laboratory Analysis

The virus was identified by enzyme-linked immunosorbent assay (ELISA). Viral load was measured by quantitative RT-PCR. The WHO International Standard for CMV (NIBSC 09/162) allows for harmonization of quantitative nucleic acid tests (QNAT) between laboratories. A viral load above 10⁴ copies/ml, approximately equivalent to approximately 1.1 × 10⁴ IU/ml, is commonly viewed as clinically significant in pregnancy [25].

Cytokine levels were measured by enzyme-linked immunosorbent assay (ELISA).

Measured cytokines

Tumor necrosis factor-alpha (TNF-α)

Statistical Analysis

Software used: SPSS (version 28) and GraphPad Prism (version 9).

Tests

T-test or Mann-Whitney test for comparisons between two groups, Correlation analysis (Pearson/Spearman) to study relationships between variables, Multiple linear regression analysis to adjust for confounding variables.

Results and Discussion

204 samples were collected from pregnant women from Al-Zahraa Teaching Hospital, Al-Furat Al-Awsat Hospital, private conventions and private laboratories during the period from November 2024 to March 2025. 154 samples were found to be positive for CMV and 50 control samples. The samples were divided according to age into three groups (18-25 years), (26-35 years), and (36-45 years). The samples were also divided according to pregnancy duration into three groups: (first trimester), (second trimester), and (third trimester) as show in table 1:

Table 1: Sample Distribution of CMV in Pregnant Women

class	Subgroup	Positive of CMV (n=108)	Positive Miscarriages (IgG) OF MCV(n=46)	Control (n=50)
Age Groups	18-25 years	34 (31.4%)	18 (39%)	19 (38%)
	26-35 years	50 (46.3%)	15(32.6%)	21 (42%)
	36-45 years	24 (22.3%)	13(28.2%)	10 (20%)
Pregnancy Trimester	first trimester	31 (28.8%)	20(43.4%)	14 (28%)
	second trimester	45 (41.7%)	14 (30.4%)	20 (40%)
	third trimester	32 (29.6%)	12(26%)	16 (32%)

Comparing with the control group, infection with the virus was varied depending on the age group. The most infected age range was 26-35 years, with 50 women (46.3%) the most representative group. The least infected was aged 36-45 years, with 24 women (22.3%). The 18-25 years range had 34 women (31.4% of the positive sample). Pregnant women at the second trimester reported the most incidence of infection with 45 pregnant women (41.7%), compared to

the first and third trimester, and compared to the control group. This indicates that the CMV infection is most common among women of 26-35 years, the highest incidence of infection was at the second pregnancy trimester. control difference from patients is not significant due to the patient sample size compared to controls. 46% of women infected with the virus were found to have miscarriages.

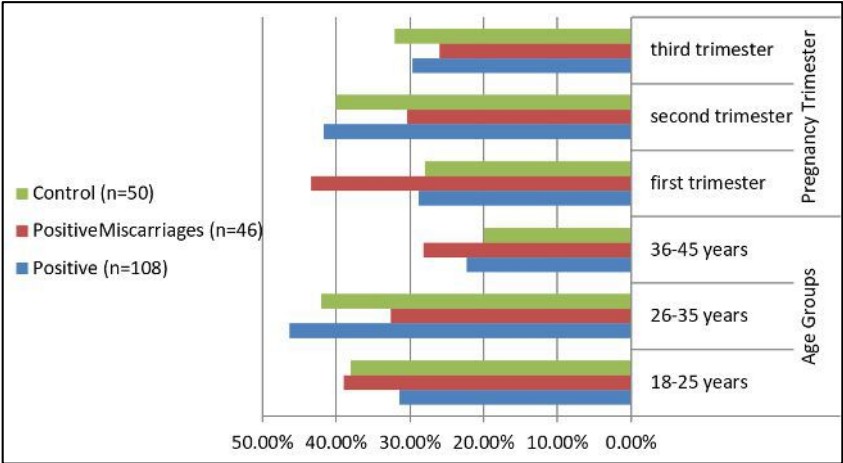


Fig 1: Sample Distribution of CMV in Pregnant Women

Cytomegalovirus (CMV) was diagnosed according to the level of IgM antibody, where all age groups were higher than the control group, which indicates an active infection. The age group (18-25, 26-35), and (36-45 years) has a significant difference ($p < 0.001$) with the control group (normal level < 12 pg/ml), which indicates an active or recent CMV infection. The study results also indicated different levels of age groups, where the (18-25) age group recorded the highest IgM (44.2 ± 11.1 pg/ml) followed by the (26-35) (37.7 ± 9.5 pg/ml), and was lowest in the (36-45) (31.4 ± 7.8 pg/ml) age group. This suggests a progressive reduction in IgM with advancing age. This may be due to

decreased primary exposure to the virus since some of the women already have immunity from a previous infection. It may also be the result of a compromised immune response with age, owing to the declining immunity of the body in its capacity to produce antibodies.

In those who miscarried in pregnancy, IgM values were strongly different from controls ($p < 0.001$), suggesting active CMV infection is associated with an increased risk for miscarriage. (18-25 years) had the highest level of IgM in miscarriage (65.3 ± 14.2 pg/ml), followed by 26-35 years old (50.1 ± 12.8 pg/ml) and 36-45 years old (46.9 ± 10.4 pg/ml).

Table 2: Detection of IgM level pg/ml of CMV in pregnant women according to age groups

Age Group (Years)	Cases (n)	Mean IgM (pg/ml) $\pm$ SD	p-value vs Controls	Miscarriages (n)	Mean IgM in Miscarriage Cases (pg/ml) $\pm$ SD	p-value vs Control
Controls	50	< 12 (Negative)	—	0	—	—
18-25	35	44.2 ± 11.1	< 0.001	18	65.3 ± 14.2	< 0.001
26-35	50	37.7 ± 9.5	< 0.001	15	50.1 ± 12.8	< 0.001
36-45	23	31.4 ± 7.8	< 0.001	13	46.9 ± 10.4	< 0.001
Total	108			46		

This would suggest that the immune response of young women may lead to an overactive inflammatory response, and this increases the risk of miscarriage. Young women (18-25 years) are more likely to have primary CMV infection (as they have not yet encountered the virus), resulting in a stronger immune response and higher levels of IgM production.

Older women (36-45 years) may be resistant in part to infection because they have been exposed before to the virus (where the response shifts from IgM to IgG), therefore their

lower levels of IgM.

The current research concurs with (Porobic-Jahic *et al.*, 2019)⁷, who confirmed that high IgM levels are associated with increased miscarriage risk. (Jones *et al.*, 2023)^[8] also pointed out that young women are more susceptible to CMV infection as they have never previously encountered the virus. Elevated IgM levels indicate a greater risk of miscarriage and are associated with serious complications in pregnancy. This is what (Ben Shoham *et al.*, 2023)^[9] noted and is consistent with what our study revealed.

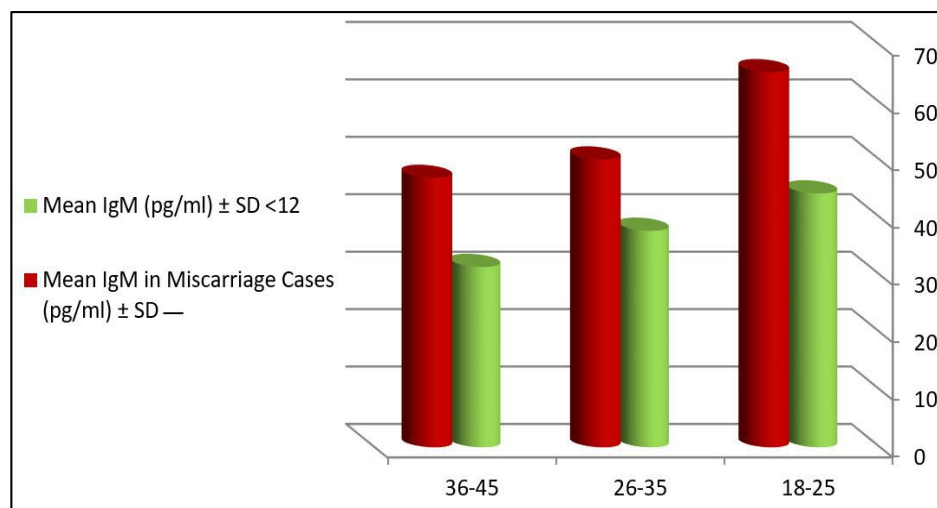


Fig 2: Detection of IgM level pg/ml of CMV in pregnant women according to age groups

IgM concentrations were also juxtaposed in terms of gestational age, and during all three trimesters (first, second, and third), significantly higher IgM concentrations ($p < 0.001$) were observed compared to controls, confirming the active CMV infection in pregnancy.

IgM levels varied by gestational trimester, the highest IgM levels (51.6 ± 15.3 pg/ml) being recorded in the first trimester, then the second trimester (42.1 ± 12.2 pg/ml), subsequently followed by the third trimester (38.8 ± 11.7 pg/ml). This, therefore, confirms a decrease in IgM levels as pregnancy progresses.

This may be due to a decrease in the severity of infection as

pregnancy progresses (possibly due to immune system adjustment).

Or, it could be a result of the effect of the mature placenta during the second and third trimester that can also serve as a partial barrier to viral transmission. The findings further show that the levels of IgM in miscarriages were significantly higher compared to controls ($p < 0.001$), lending support for the postulation that active CMV infection could raise the risk of miscarriage.

The maximum IgM levels in miscarriage were during the first trimester (74.5 ± 15.8 pg/ml), then the second (56.3 ± 13.1 pg/ml), and the third (50.2 ± 11.6 pg/ml).

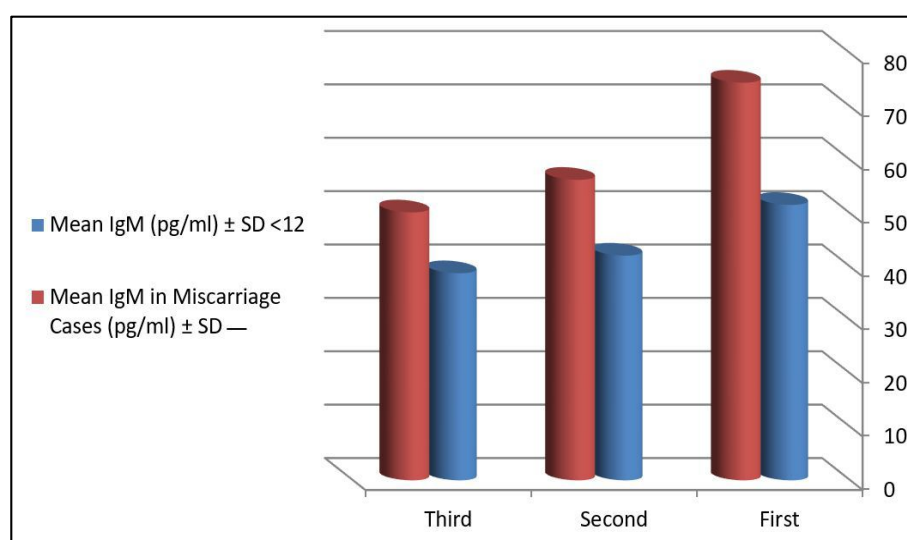
Table 3: Anti-CMV IgM Levels by Pregnancy Trimester Compared to Controls

Trimester	Cases (n)	Mean IgM (pg/ml)±SD	p-value vs Controls	Miscarriages (n)	Mean IgM in Miscarriage Cases (pg/ml)±SD	p-value vs Controls
Controls	50	<12 (Negative)	—	0	—	—
First	30	51.6±15.3	<0.001	20	74.5±15.8	<0.001
Second	45	42.1±12.2	<0.001	14	56.3±13.1	<0.001
Third	33	38.8±11.7	<0.001	12	50.2±11.6	<0.001
	108			46		

This may be due to the fact that the fetus is more susceptible to damage in the first trimester due to poor development of the placenta (which is not sufficiently protective against the virus). This may also be due to fetal cells being more sensitive to viral infection in early development.

The high level of IgM during the first trimester would also be attributed to the production of enormous amounts of IgM upon the first exposure of the body to the virus (new infection). Or else the infection may have been new, inducing high levels of IgM.

Our evidence agree with (Hemmersbach-Miller *et al.*, 2020) ^[10], who explained that concentrations of IgM rise highest during the first trimester of pregnancy, and this raises the risk of miscarriage. (Fowler *et al.*, 2022) ^[11] similarly explained that pregnant women are 2.3 times more likely to miscarry during the first trimester of pregnancy. (Chin *et al.*, 2022) ^[12], (Zehner *et al.*, 2023) ^[13], and (Ai *et al.*, 2022) ^[14] similarly discovered that initial infection during early pregnancy raises the risk of miscarriage.

**Fig 3:** Anti-CMV IgM Levels by Pregnancy Trimester Compared to Controls

Our outcome of the study revealed that the miscarriage rate in women who had IgG-negative antibodies was 48.4% (32 out of 66 cases), $p < 0.0001$ (very high significant

difference), while miscarriage rate in women who had IgG-positive antibodies was 15.9% (14 out of 88 cases), $p = 0.13$ (not statistically significant).

Table 4: Association Between Miscarriage Cases and CMV IgG Status

CMV IgG Status	Total Cases (n)	Miscarriage Cases (n)	Miscarriage (%)	p-value
IgG (Positive)	88	14	15.9%	0.13
IgG (Negative)	66	32	48.4%	<0.0001

This determines that there is a threefold increased risk of miscarriage in women with IgG-negative antibodies. This is evidence for a primary active CMV infection during pregnancy, an underlying immunity deficiency which exacerbates the infection, or an exaggerated immune response that leads to acute placental inflammation.

In the case of IgG-positive ones, the miscarriage rate was the same as for the normal one (15-20%).

Pre-existing immunity can also block transmission to the fetus and decrease symptom severity in the case of recurrence. For IgG-negative antibodies, it could be through direct injury of placental tissue by the virus, interference

with placental vascular development, or dysregulation of inflammatory cytokines. (TNF- α).

The results of our study are consistent with the study of (Andrievskaya *et al.*, 2015) ^[15], who indicated in his study that the absence of old IgG immunity leads to an excessive immune response and inflammation in the placenta, which leads to miscarriages, and this is what our study confirmed. It is also consistent with (Akunaeziri *et al.*, 2021) ^[16] whose results showed that 51% of women who experience miscarriage are those who did not have previous IgG immunity.

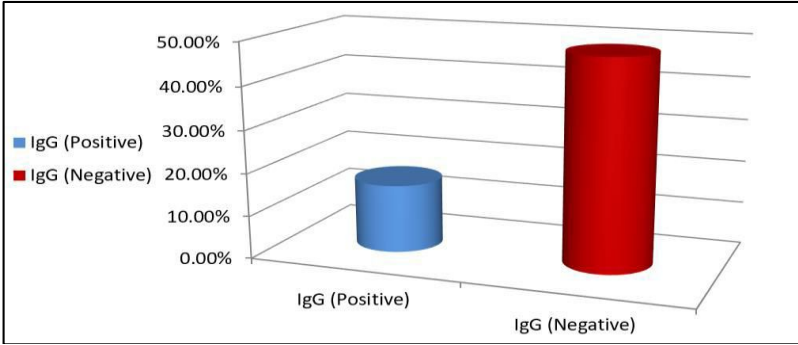


Fig 4: Association Between Miscarriage Cases and CMV IgG Status

TNF- α was measured as an inflammatory cytokine playing a critical role in the induction of uterine muscle contractions and subsequent abortions. It was compared with viral load by age. Viral load and TNF- α were markedly increased ($p<0.05$) in all age groups compared with controls. Our results show that viral load was stratified by age groups as (18-25 > 26-35 > 36-45 years), while TNF- α was: (18-25 > 26-35 > 36-45 years). By ANOVA analysis, we found that viral load ($p<0.001$) and TNF- α ($p<0.001$) were significantly higher in viral load and TNF- α in the miscarriage patients compared with the non-miscarriage groups.

The (18-25) had a 100% increase in viral load and 40% increase in TNF- α . The (26-35) a 92% increase in viral load and 40% increase in TNF- α . The (36-45) had an 85% increase in viral load and 54% increase in TNF- α . The results further showed a correlation between the severity of the infection and age and the highest infection rates among women belonging to the age group (18-25 years). A higher viral load and increased TNF- α levels may be the consequence of an overactive innate response because the immune system is immature and mounts a strong inflammatory response, or upon primary exposure to the virus when not previously immune.

Table 5: CMV Viral Load and TNF- α Levels in CMV Patient according to Age groups

Group	(n=108)	CMV Viral Load (IU/ml)	TNF- α (pg/ml)	p-value vs Control	Miscarriage Subgroup (n=46)	Viral Load in Miscarriage (IU/ml)	TNF- α in Miscarriage (pg/ml)
Control	50	<50	84 $\pm$ 11	-	-	-	-
18-25 years	35	3.4 $\times$ 10 ⁵ $\pm$ 9.6 $\times$ 10 ⁵	212 $\pm$ 48	<0.001	12	6.8 $\times$ 10 ⁵ $\pm$ 3.1 $\times$ 10 ⁶	297 $\pm$ 59
26-35 years	50	2.7 $\times$ 10 ⁴ $\pm$ 6.0 $\times$ 10 ⁴	166 $\pm$ 39	<0.001	18	3.4 $\times$ 10 ⁵ $\pm$ 2.3 $\times$ 10 ⁶	233 $\pm$ 48
36-45 years	23	1.5 $\times$ 10 ³ $\pm$ 3.4 $\times$ 10 ³	122 $\pm$ 33	0.013	16	9.5 $\times$ 10 ⁴ $\pm$ 5.7 $\times$ 10 ⁵	188 $\pm$ 46

Older women (36-45 years of age) showed lower viral loads,TNF- α may be due to the acquired immunity resulting from past infection with the virus, even if there is no IgG +, or simply due to a decreasing immune capacity with age. The association of miscarriages and viral load is a result of the fact that the virus infects placental cells responsible for supplying nutrients to the fetus, resulting in fetal hypoperfusion and placental damage. Vertical transfer of the virus from mother to child also causes infection of fetal tissue, especially during the first trimester pregnancy. TNF- α is also implicated in inducing uterine contractions, which causes inflammatory mechanisms that lead to premature contractions leading to miscarriage.

Our results are consistent with (Heydarifard *et al.*, 2022) ^[17], which reported a high correlation between high CMV viral load and TNF- α levels and the occurrence of miscarriage, which is entirely consistent with our results. Additionally, (Pontes *et al.*, 2024) ^[18] is in agreement with our study that viral load five times increases miscarriage risk and high TNF- α levels associate with placental lesions. (Al-Kinani *et al.*, 2022) ^[19] indicated a high concentration of TNF- α among women experiencing miscarriage, confirming the cytokine's role in miscarriage, as did our study. In a study in Iraq at the University of Babylon, (Huda *et al.*, 2024) ^[20] indicated that high levels of TNF- α accompanied by IL-10/IL-17 imbalance have direct association with recurrent miscarriage, as demonstrated in our study.

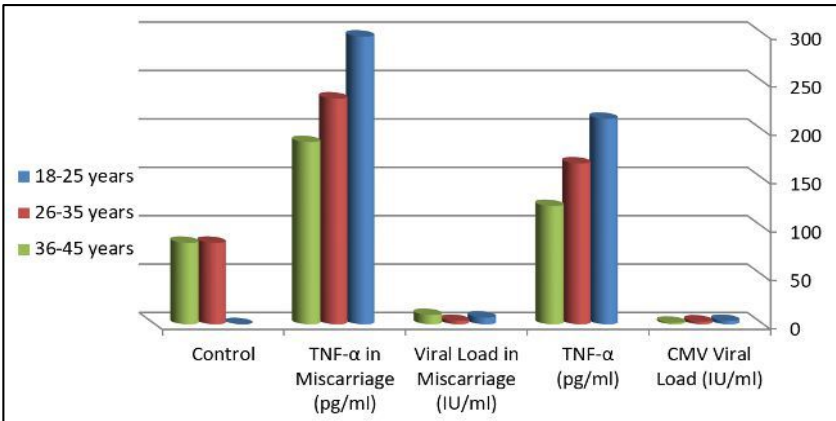


Fig 5: CMV Viral Load and TNF- α Levels in CMV Patient according to Age groups
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When viral load levels were compared with tumor necrosis factor-alpha (TNF- α) in pregnant women by gestational age and miscarriage status, viral load and TNF- α levels were higher in the CMV-infected pregnancy groups compared to the control group.

First-trimester viral load was more variable on average (4.5×10^5 IU/ml) with a large standard deviation ($\pm 1.6 \times 10^6$), indicating high between-case variability (some cases were extremely high). Second-trimester viral load was much lower (2.8×10^4 IU/ml) with a lower standard deviation

($\pm 5.5 \times 10^4$). It was lower in the third trimester (7.0×10^2 IU/ml), with a reduction in viral activity as pregnancy progresses.

TNF- α levels were higher in the first trimester (225 ± 53 pg/ml), possibly as a result of a robust immune response to the viral infection. Progressive decline throughout the second and third trimesters of pregnancy (182 ± 42 and 136 ± 35 pg/ml, respectively) was seen, perhaps indicating a better immune control of the virus.

Table 6: CMV Viral Load and TNF- α in CMV Patient according to Trimester

Trimester	Total Cases (n=108)	Mean Viral Load (IU/ml)	Mean TNF- α (pg/ml)	Miscarriage Cases (n=46)	Viral Load in Miscarriage (IU/ml)	TNF- α in Miscarriage (pg/ml)	p-value (Miscarriage vs Non)
Control	50	<50	84 ± 11	—	—	—	—
1st Trimester	30	$4.5 \times 10^5 \pm 1.6 \times 10^6$	225 ± 53	22	$6.2 \times 10^5 \pm 3.3 \times 10^6$	311 ± 66	<0.001
2nd Trimester	45	$2.8 \times 10^4 \pm 5.5 \times 10^4$	182 ± 42	16	$1.9 \times 10^5 \pm 8.7 \times 10^5$	246 ± 54	0.003
3rd Trimester	33	$7.0 \times 10^2 \pm 2.2 \times 10^3$	136 ± 35	8	$4.4 \times 10^4 \pm 2.5 \times 10^5$	191 ± 46	0.021

Both viral load and TNF- α were higher in miscarriage cases, particularly in the first trimester (6.2×10^5 IU/ml) compared to the overall average (4.5×10^5) (311 ± 66 pg/ml) compared to non-miscarriage cases. The difference was statistically significant (p-value < 0.001), indicating that high viral load and inflammation (TNF- α) are strongly associated with miscarriage.

In the second trimester, viral load was higher (1.9×10^5 IU/ml) compared to the mean (2.8×10^4). TNF- α was higher (246 ± 54 pg/ml) with a statistically significant difference (p-value = 0.003), confirming the role of infection and inflammation in miscarriage even in the second trimester. The third trimester also showed a higher viral load (4.4×10^4 IU/ml) compared to the mean (7.0×10^2), and higher TNF- α (191 ± 46 pg/ml) with a p-value of 0.021, which was statistically significant. The effect was not as pronounced as in the first and second trimesters, which may be due to the maturity of the immune system of the pregnant woman. This

means that viral load is closely linked with inflammatory immune response, thus leading to a high possibility of miscarriage. CMV induces the release of TNF- α , which is an inflammatory cytokine leading to poor vascular formation due to inhibition of placental growth. It also stimulates prostaglandins, which cause uterine contractions, and leads to damage of fetal tissues due to an overactive immune response.

The final answer is, All of these are capable of inducing miscarriage, especially during the first trimester, as the fetus is more susceptible to viral and inflammatory effects during this time.

In the first trimester, the mother's immune system is less tolerant of pregnancy, and therefore the inflammatory response (e.g., TNF- α) is stronger. Even in the third trimester, immunological tolerance may develop, and the effect of CMV and TNF- α on the pregnancy could be weaker.

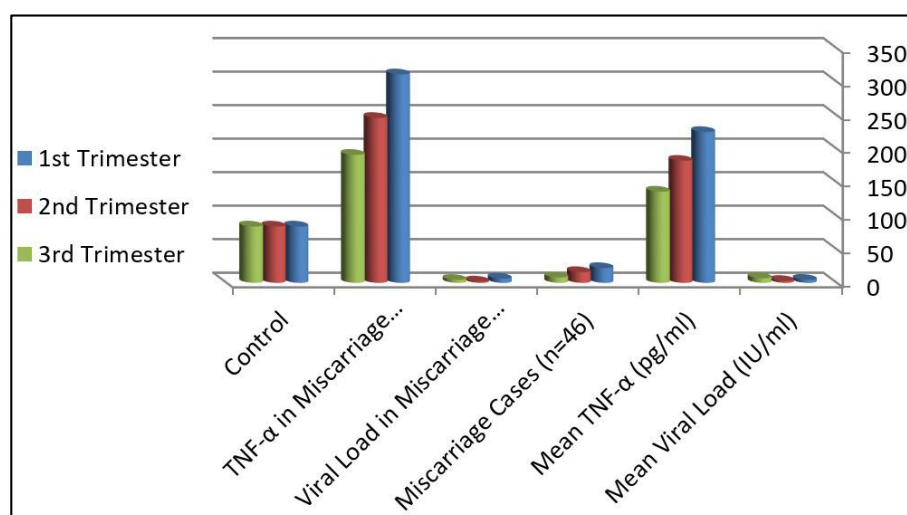


Fig 6: CMV Viral Load and TNF- α in CMV Patient according to Trimester

The results of the current research are consistent with (Griffiths *et al.*, 2016) [21], who showed that CMV infection, particularly in the first trimester, is associated with a significantly increased risk of miscarriage, partly due to an overwhelming viral load and inflammatory response (which includes TNF- α).

They are also agree with (Davies *et al.*, 2022) [23], who showed that an exaggerated TNF- α -driven inflammatory response can disrupt the immune homeostasis necessary for pregnancy maintenance. However, our study adds further evidence that this relationship attenuates as pregnancy progresses, supporting the theory that immune timing of

infection plays a significant role. These findings necessitate further studies examining the role of interventions against TNF- α in pregnant women with active CMV infection. (Dobrer *et al.*, 2024) ^[24-27] referred to the key role of the inflammatory response in viral infection-mediated pregnancy complications. Our findings showed that miscarriage was associated with the extremely high levels of TNF- α , particularly in the first trimester ($p < 0.001$). This trend is consistent with the mechanism proposed by the Dobrer study, which explains how viral infection can cause an excessive immune response that is harmful to pregnancy. However, our study adds a further dimension by specifically linking CMV viral load and TNF- α concentrations in miscarriages, strengthening our current understanding of mechanisms of infection-related pregnancy loss. Our findings are consistent with the (Salomè *et al.*, 2025) ^[25] in implicating both CMV viral load and the inflammatory response with which it is associated in pregnancy outcome. In our study, miscarriages were found to be associated not only with high viral loads but also with high levels of TNF- α , suggesting that the deleterious effects of CMV may be via the inflammatory response.

Conclusion

Infection with Cytomegalovirus (CMV) during pregnancy, especially in those who do not have prior immunity (IgG negative), is associated with a very high risk of miscarriage. Viral load and TNF- α levels are found to be significant predictors of unfavorable outcomes, especially in the first trimester of pregnancy. Screening for CMV and monitoring of inflammatory markers in pregnancy have been highlighted by this study as routine to identify high-risk cases early. The establishment of preventive measures and perhaps antiviral or anti-inflammatory treatment can reduce the incidence of CMV-related miscarriage.

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