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The interaction between cytokine profiles and Epstein-Barr virus-induced immune response

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The Epstein-Barr virus (EBV) infection is capable of triggering the complex of host immune response where the proportion of the pro- and anti-inflammatory cytokines is a key factor in the persistence of the virus, immune protection, and the progression of the disease. These are the interactions among cytokines which are essential in the pathogenesis of EBV and disorders that are affiliated with it. The paper will help to clarify the relations between the cytokine profiles and the immune reaction caused by Epstein-Barr virus infection and reveal the main cytokines that could determine the persistence of the virus, regulate the immune response, and determine the progression of the disease. The current case-control study was a study designed in Nasiriyah, Iraq, between February and September 2025 and consisting of 90 participants with known infection by the Epstein-Barr virus and 50 healthy participants of the same age group (between 18 to 50 years old). The exclusion criteria included pregnancy, chronic disease, autoimmune disease, malignancies and immunosuppressive drugs. Blood samples were collected by vein, serum was isolated and kept at –20 ºC. Pro- and anti-inflammatory cytokines (IL-6, TNF-a, IL-1b, IL-10, TGF-b), and anti-EBV antibodies (VCA IgM and IgG) were quantified by using enzyme-linked immunosorbent assays. Result: The researchers did not observe any significant differences between the groups regarding gender or age. The patients were much fatter than the controls (26.8 ± 4.2 vs. 24.5 ± 3.7 kg/m^2, P = 0.003) and it is possible that being fat is a risk factor of EBV. There was a significant rise in inflammatory cytokines (IL-6, TNF-a 1, and IL-10) and anti-inflammatory cytokines (IL-10 and TGF-b) in the patients, which shows that their immune systems were changing. Considerably more anti-EBV VCA IgM and IgG was found, and these were closely linked to molecules that cause inflammation. Conclusions: EBV infection elicits a robust immune reaction; it is manifested in proinflammatory (IL-6, TNF-3) and anti-inflammatory (IL-10, TGF-3) cytokines. This balance is an attempt by the host to balance viral proliferation and minimal tissue damage to facilitate successful antiviral protection.

Keywords: Epstein-Barr virus; cytokines; immune response; IL-6; TNF-α; TGF-β.

Introduction

The Epstein-Barr Virus (EBV) is a common human gammaherpesvirus that affects more than 90% of people in the world (Damania et al., 2022). Even though most people don't get infected as children, EBV can cause infectious mononucleosis in teens and young adults and is linked to a number of cancers, including Burkitt lymphoma, Hodgkin lymphoma, and nasopharyngeal carcinoma (Yu & Robertson, 2023). EBV-related disease development is closely linked to the immune system, specifically the balance between controlling viruses and immune pathology (Shechter et al., 2022).

Cytokines are soluble signaling proteins that regulate the proliferation, differentiation, and effector activities of immune cells. In EBV infection, the virus triggers innate and adaptive immune responses, leading to the release of a complex of cytokines, such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), interleukin-10 (IL-10), and transforming growth factor-beta (TGF-β) (Shechter et al., 2022; Malik et al., 2023). Activation of early viral recognition and cytotoxic T-lymphocytes, B-cells, and natural killer cells necessitates pro-inflammatory cytokines such as IL-6, TNF-α, and IL-1β (Rex et al., 2023; Abd-Alwahab et al., 2024). However, excessive production of such cytokines can result in systemic inflammation and tissue destruction, which is one of the causes of the clinical manifestations of acute EBV infection (Zhang, 2023).

On the other hand, IL-10 and TGF-β are regulatory cytokines that are very important for keeping the immune reaction in check so that it doesn't become too inflammatory. Instead, they help keep the immune system in balance (Naif et al., 2025). Namely, IL-10 suppresses antigen presentation and generation of pro-inflammatory cytokines, thereby limiting tissue damage. TGF-β has a wide-range of immunoregulatory functions, such as preventing T-cell proliferation and B-cell activity, which inhibit chronic inflammation and autoimmune responses. These pro and anti-inflammatory cytokines have a dynamic balance that is important in defining the outcome of the disease and determining the risk of developing EBV related complications (Rakityanskaya et al., 2024).

Researchers have said in the past that the severity of EBV symptoms was linked to high amounts of cytokines that cause inflammation. For instance, in people with infectious mononucleosis a link has been made to fever, swollen lymph nodes, and a bigger spleen when their blood IL-6 and TNF-alpha levels are high (Fevang et al., 2021). To make up for this, a higher amount of IL-10 and TGF-β is often seen as a way to protect against damage that would have come from the immune system being too active(Fevang et al., 2021; Ajeed et al., 2025). Although much data has been collected, the exact relationship between particular cytokine profiles and EBV-induced immune reactions has not yet been fully comprehended, especially regarding the impact that such interactions have on the production of antibodies and the longevity of viral persistence (Moppert et al., 2023).

EBV-induced cytokine milieu is not merely significant in discuss ion of the viral pathogenesis, but also has clinical consequences. Cytokine profiling would serve as an indicator of disease progression, disease severity and risk of malignancies associated with EBV, as a prognosis biomarker. Moreover, the immunologic response can be

possibly controlled by intervention targeting the specific cytokine pathways, and therefore reducing immunopathology and enhancing viral control (An et al., 2024).

The paper therefore is to discuss the relationship between the cytokine profiles and the EBV-mediated immune responses by comparing the pro-inflammatory and regulatory cytokines in the patients and healthy controls. These associations and the amount of anti-EBV antibodies are ways in which this study seeks to provide a comprehensive view of the immune dynamic that is brought about by the EBV infection in order to provide insights on the potential diagnostic and treatment options.

Materal and methods

The Human Ethics Committee of Al-Habbobi Teaching Hospital gave approval to the study. Every participant was made aware of the study and asked to give written consent to the study. Each patient was informed that his personal information woulds be kept in confidence and no other parties would have access to his data.

The study was a case-control case study, which was carried out in Nasiriyah City, Iraq, between February 2025 and September 2025, to examine the relationship between cytokine patterns and the immune reaction to Epstein-Barr virus (EBV). One hundred and forty individuals were recruited, comprising 90 patients who had confirmed EBV infection and 50 healthy age and gender-matched controls. The inclusion criteria were patients with EBV primary/acute infection who were aged 18 to 50 years and controls were healthy without a history of EBV infection or chronic illness. The eligibility criteria excluded pregnant women, patients with chronic liver or kidney disease, autoimmune disease, malignancy, or immunosuppressive/anti-inflammatory drug use. After going without food for a night, 5 mL of venous blood was collected from the participants. The samples were then clumped together and spun at 3000 rpm for 10 minutes to obtain the serum, which was then stored at –20 ºC until it could be analyzed (Arif et al., 2025) ELISA kits were used to measure pro-inflammatory (IL-6, TNF-α, IL-1β) and anti-inflammatory (IL-10, TGF-β) cytokines according to the instructions given by the manufacturer. The anti-EBV antibodies VCA IgM and IgG were also measured to confirm infection and check the humoral immune response.

We demonstrated the results in the form of mean standard deviation of continuous variables and proportions of the categorical variables. Unpaired t-tests were used to determine the differences between groups but Spearman rank -order correlation was used to determine associations. The level of significance of $P < 0.05$ was considered.

Results

The findings indicated that the patients and the healthy groups did not differ significantly in terms of their gender or age. They had means of 34.8 ± 10.5 years, and the means of the control group were 33.2 ± 9.8 years and the P = 0.386. The distribution of the men and women in the two groups was not greatly different. The body mass index (BMI), on the other hand, exhibited a significant difference; with a much higher value in the patients (26.8 ± 4.2 kg/m^2) than in the healthy controls (24.5 ± 3.7 kg/m^2) with a P = 0.003. This points to obesity and overweight being associated with the potential of contracting a virus. Regarding active smoking, more patients group showed a higher incidence (30.0%) compared to the control group (17.0%), but the difference was not statistically significant (P = 0.101).

Table 1
Comparison of age, gender, body mass index (BMI), and smoking status between the study groups

Variable	Controls (n = 50)	Patients (n = 90)	Test statistic	P-value
Age (years), mean ± SD	33.2 ± 9.8	34.8 ± 10.5	t = 0.87	0.386
Male, n (%)	28 (56.0%)	52 (57.8%)	$\chi^2 = 0.04$	0.841
Female, n (%)	22 (44.0%)	38 (42.2%)		
BMI, kg/m² (mean ± SD)	24.5 ± 3.7	26.8 ± 4.2	t = 3.05	0.003
Current smokers, n (%)	9 (18.0%)	27 (30.0%)	$\chi^2 = 2.68$	0.101

As indicated in Table 2, the levels of the inflammatory cytokines were significantly higher in the Epstein-Barr virus group (EBV) than in the control group. Mean IL-6 concentration was found to be 42.5 ± 12.3 pg/mL in the EBV group, compared to 14.8 ± 5.2 pg/mL in healthy controls. This was a very significant difference ($P < 0.001$). There was an important difference ($P < 0.001$) between the patients (38.6 ± 10.7) and the control group (12.5 ± 4.8) in the TNF-levels. The average IL-1ba in the patients was 18.3 ± 6.1 and the average in the healthy control was 6.4 ± 2.2. This indicates that the inflammation level in the infected patients was quite different from that of the healthy control patients. Apparently, such findings can be attributed to the enhanced immune response, which is associated with the presence of EBV and functions of such cytokines in the response.

Table 2
Comparison of IL-6, TNF-α, and IL-1β concentrations (pg/mL) between the study groups

Cytokine	Controls (n = 50) mean ± SD	Patients (n = 90) mean ± SD	Test statistic	P-value
IL-6	14.8 ± 5.2	42.5 ± 12.3	t = 14.2	<0.001
TNF-α	12.5 ± 4.8	38.6 ± 10.7	t = 15.1	<0.001
IL-1β	6.4 ± 2.2	18.3 ± 6.1	t = 14.0	<0.001

Table 3 revealed that the level of anti-inflammatory cytokines was significantly greater in individuals who had the Epstein-Barr virus (EBV) than in individuals who did not have the virus. The mean of the IL-10 of the cases was 15.8 ± 4.9 pg/mL and that in the healthy control was just 9.3 to 2.5 pg/mL. This was a statistically significant difference ($P < 0.001$). The EBV cases (8.7 ± 2.4) had also higher TGF-β than the control group (5.1 ± 1.6) and this is a statistically significant difference ($P < 0.001$). According to these findings, it appears that regulatory mechanisms are acting to prevent the excess response of inflammatory reaction to EBV infection. This implies that these cytokines maintain the integrity between an effectively functioning immune action and the control of excessive inflammation.

Table 3
Comparison of IL-10 and TGF-β concentrations (pg/mL) between the study groups

Cytokine	Controls (n = 50) mean ± SD	Patients (n = 90) mean ± SD	Test statistic	P-value
IL-10	9.3 ± 2.5	15.8 ± 4.9	t = 9.86	<0.001
TGF-β	5.1 ± 1.6	8.7 ± 2.4	t = 9.23	<0.001

The Table 4 results indicated that there was a high increase in the level of antibodies against the Epstein-Barr virus envelope in patients as opposed to the control group. The mean of the anti-EBV VCA IgM level in the patients was 78.5 ± 21.2 units/mL in comparison to the 12.4 ± 4.1 units/mL in the healthy controls with a high level of significance ($P < 0.001$) and the mean anti-EBV VCA IgG was 145.7 ± 35.4 in comparison to 98.6 ± 28.5 units/mL in the healthy control with a high level of significance ($P < 0.001$). These findings show that acute and long-standing immune reaction against EBV exists in the patients, it means that it is at the active stage of the infection and that immunological memory exists that is correlated to high level of IgG.

Table 4
Comparison of anti-EBV VCA IgM and IgG levels (U/mL) between the study groups

Marker	Controls (n = 50) mean ± SD	Patients (n = 90) mean ± SD	Test statistic	P-value
Anti-EBV VCA IgM, U/mL	12.4 ± 4.1	78.5 ± 21.2	t = 22.3	<0.001
Anti-EBV VCA IgG, U/mL	98.6 ± 28.5	145.7 ± 35.4	t = 7.64	<0.001

The results shown in Table 5 demonstrated that anti-EBV VCA ImgM levels and cytokines were significantly positive in patients who had Epstein-Bar virus infection. The results revealed that the antibodies had a strong correlation with IL-6 (0.564; P = 0.001), then TNF-alpha (0.502; P = 0.001) and IL-1-B (0.432; P = 0.001), which illustrates the role of these inflammatory cytokines in stimulating a vigorous immune response in the acute period of infection. Regarding the

anti-inflammatory regulatory cytokines, IL-10 (0.287, 0.007) and TGF-B (0.241, 0.021) were not significantly correlated, implying that regulatory mechanisms partially control an excessive response of the inflammation and provide an immune balance between active defence and control of tissue injury.

Table 5
Spearman's rank correlation coefficients (ρ) between anti-EBV VCA IgM and cytokines

Cytokine	Spearman's ρ	P-value
IL-6	0.564	<0.001
TNF-α	0.502	<0.001
IL-1β	0.432	<0.001
IL-10	0.287	0.007
TGF-β	0.241	0.021

Discussion

The given paper compared the immunological and serological patterns of the patients with Epstein-Barr Virus (EBV) infection and the healthy controls in terms of the levels of cytokines and anti-EBV antibodies. Sociodemographic analysis showed that there were no significant differences in age and gender between patients and controls, which showed that the groups were well-suited in these aspects (Cavalieri et al., 2022). Nevertheless, the patients demonstrated very high levels of the body mass index (BMI), and it might indicate a possible combination of metabolic status and resistance or immunity to EBV infection (Lin et al., 2023). Even though the prevalence of smoking was greater in the patients, this difference was not statistically significant, which means that the confounding influence of smoking on the indicators of immunological parameters that were measured was limited (Dickerson et al., 2023).

We looked at inflammatory cytokines and found that IL-6, TNF-001, and IL-100 levels were significantly higher in the EBV cases than in the controls (P = 0.001). Researchers have already found that high amounts of IL-6 and TNF-alpha are present during an acute EBV infection (Moppert et al., 2023). which supports the idea that these results are related to an active pro-inflammatory response. IL-6 has been shown to help B-cells grow and differentiate, which fits with the fact that the patients have high amounts of anti-EBV VCA IgM and IgG, which means the virus is still spreading and the humoral immune system is working. When TNF-alpha and IL-1 beta are present, they cause inflammation and activate immune effector cells. This suggests that the body has a strong innate immune reaction to stop the spread of viruses (Tan et al., 2025).

Surprisingly, the levels of anti-inflammatory cytokines IL-10 and TGF-b were also considerably high among patients, though to a lesser extent than those of the pro-inflammatory ones. This increase is probably a compensatory process to avoid overinduced tissue destruction as a result of the increased inflammatory reaction to preserve immunological homeostasis (Leng et al.,2023). Dual increase of pro- and anti-inflammatory cytokines such has been observed in EBV infections was viewed as a characteristic of the capacity of the virus to suppress the host immunity (Mohammadi et al., 2022).

Serological examination revealed that the presence of anti-EBV VCA needed to be performed at a significantly higher level in patients than in controls (P < 0.001), which confirmed active and recent viral infection. Acute-phase humoral response is evidenced by the increase in IgM whereas the presence of immunological memory and continuous immune surveillance is evidenced by the increase in IgG (Nishio et al., 2023). Correlation analysis of anti-EBV VCA IgM and cytokines indicated strong positive relationships with IL-6, TNF-a, and IL-1b and this implies that these inflammatory mediators play a direct role in the activation of B-cells and in the production of antibodies (Lupo et al., 2023). Conversely, the relationships with IL-10 and TGF-b were less strong but significant, which illustrates the modulative effect of the regulatory cytokines to avoid excessive activation of the immune system and reduce immunopathology (Kitamura et al., 2025).

These results can be highly correlated with the past reports that indicated the interaction between the EBV replication and the host cytokine profiles. To illustrate this, it was shown by Sighencea & Trifu (2025) that acute EBV infection was characterized by an increased level of IL-6 and TNF-alpha that were related to the level of viral load and symptoms. Equally, research by Bose et al. (2022) highlighted the duality of IL-10 in tuning the antiviral immunity and reducing the effects of inflammation. Nevertheless, other studies have found less significant or non-significant changes in regulatory cytokines during EBV infection, which may be caused by age differences in the patients, changes in disease stage, or time of sample collection, and the dynamism of cytokine responses (Bose et al., 2022).

The combined increase in inflammatory and regulatory cytokines is indicative of the EBV infection leading to a highly controlled immune network with pro-inflammatory mediators activating the process of viral containment, and anti-inflammatory cytokines responding to curtail collateral tissue damage. This interaction can also be one of the explanations of the large range of clinical manifestations observed in individuals infected by the virus with such symptoms as asymptomatic seroconversion and overt infectious mononucleosis (Gothe et al., 2022).

Conclusion

To sum up the current findings, it is important to note that the balance of cytokines plays a decisive part in the host reaction to EBV infection. Combined growth of pro- and anti-inflammatory mediators, and increase in specific antibodies are indicative of a well-coordinated immune reaction that balances the control of viral replication and prevents over immunopathology. They are still informative in the future treatment of cytokines modifying the disease EBV and can be applied in the context of the study of virus-host interaction in immunopathogenesis.

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