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Mechanisms of Epstein–Barr Virus Infection in Dysregulating the Omentin-1–Immune Axis

Afaf Saud Hussein
Biology Dpet., College of Sciences, University of Kirkuk, Kirkuk, Iraq,
affafalazawi@uokirkuk.edu.iq

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Abstract: Background & Objective: Epstein–Barr Virus (EBV) has been associated with immune dysregulation and changes in the levels of adipokines; however, its effect on omentin-1–immune axis among Iraqi faces limited investigation. This study was aimed to determine the effect of EBV infection to influence serum omentin-1 and other adipokines as well as pro-inflammatory cytokines, and their diagnostic usefulness.

Materials & Methods: One hundred and fifty-seven clinically diagnosed EBV patients along with 75 healthy controls were involved in this study, which is conducted in teaching hospitals clinics in Kirkuk (June–September 2025). The VCA-IgG, VCA-IgM and EBNA-IgG by ELISA kits were employed. Plasma omentin-1, chemerin, vaspin, IL-6, TNF-α and IFN-γ levels were determined. The relationships of adipokines and cytokines were analyzed, and diagnostic values were estimated according to ROC analysis.

Results: EBV positive status was found in 45 (28.6%) patients. Cytokine levels IL-6 (29.4 ± 6.1 pg/mL), TNF-α (21.7 ± 5.3 pg/mL) and IFN-γ (25.9 ± 6.7 pg/mL) in EBV-positive patients were significantly higher than in controls ($p < 0.001$). Omentin-1 was decreased (5.7 ± 1.2 µg/mL versus 8.2 ± 1.3 µg/mL in controls, $p < 0.001$), while modest reductions were found for chemerin and vaspin as well. There was an

inverse relationship between omentin-1 and cytokines (IL-6 r = −0.38, TNF-α r = −0.31, IFN-γ r = −0.29, p < 0.01). ROC analysis indicated that IL-6 and omentin-1 best discriminated between EBV infected cases (AUC 0.81 and AUC 0.78, respectively).

Conclusions: The EBV contact in Iraqi patients was accompanied by marked pro-inflammatory status and inhibition of omentin-1, which links systemic inflammation to adipokine disturbance. Serum omentin-1 and IL-6 may be biomarkers for EBV infection.

Keywords: Epstein–Barr Virus; Omentin-1; Adipokines; Cytokines; Immune Dysregulation

Introduction

Epstein–Barr virus (EBV) is a common human gamma-herpesvirus that infects over 90% of the world's population following which it establishes lifelong latent infection in hosts (1,2). Serological testing to detect Epstein–Barr virus (EBV) include viral capsid antigen immunoglobulin G and M (VCA-IgG, VCA-IgM) and EBNA immunoglobulin G status which are frequently utilized for determining stage of infection, clearance of the virus, or measuring viral reactivation against host immunity (3). Chronic EBV infection is strongly associated with immune dysregulation, chronic inflammation and predisposition to autoimmunity (4,5). EBV infection drastical-ly impacts the host immune responses through induction of persistent inflammation signaling and cytokine dysregulation. Increased production of IL-6, TNF-α and IFN-y in EBV-associated immune activation and chronic inflammatory conditions has been well documented (6–8). These cytokines are critically important for the development of both innate and adaptive immunity, and their dysfunction leads to increased immune exhaustion, tissue damage and disease progression. Recent research identifies adipokines as integral players that connect metabolic status to immune regulation. Omentin-1 is an adipokine specifically expressed in visceral fat and exerts anti-inflammatory and immunoregulatory effects by inhibiting the production of proinflammatory factors, and modulating the activation of immune cells (9,10). Lower serum omentin-1 levels have been correlated with inflammatory and autoimmue diseases indicating that it acts as a protective immune modulator (11). Apart from omentin-1, novel adipokines like chemerin and vaspin gained attention in relation to their immunologic significance. On the one hand is chemerin as a chemoattractant for immune cells and also a regulator of inflammatory signaling pathways, on the other hand is vaspin to act as an endogenous anti-inflammatory factor with cytoprotective roles in immune–metabolic disorders (12,13,14). However, little is known about the role of these less-abundant adipokines in chronic viral infections with EBV. In Iraq, high seroprevalence has also been observed in the general and diverse populations such as thalassemia, autoimmune diseases and chronic inflammatory disorders patients (15,16,17). Demographic and clinical parameters (age, sex, body mass index [BMI], duration of disease, severity of disease and previous treatment) could in turn further affect the immunological responses to EBV infection and adipokine expression which might influence EBV immune pathoepidemiology. Although the EBV-mediated immune activation has been extensively investigated, the knowledge of association between EBV seropositivity and changes in adipokine-modulated immunity is currently missing. Specifically, the association of

EBV infection with serum omentin-1, chemerin, vaspin and major pro-inflammatory cytokines has not been fully elucidated in populations with high prevalence of EBV load including Iraq. The purpose of this study was to investigate the relationship between EBV serological markers and circulating levels of omentin-1, chemerin, vaspin.

Materials and Methods

Study Design and Population

Methods A cross-sectional study was carried out between June and September 2025 in Azadi Teaching Hospital and Kirkuk Teaching Hospital, Iraq. There were 157 patients with clinical suspected or already known EBV infection and 75 healthy controls.

Inclusion criteria

Adults ≥18 years of age who provided written informed consent and had not received recent antiviral treatment.

Exclusion criteria

Patients with bacterial infections, chronic liver or kidney disease, autoimmune diseases not associated with EBV infection, compromised immune function (including pregnant women) and patients whose clinical information was not complete were excluded from the study.

Sample Collection

Twenty-milliliters of venous peripheral blood was collected in plain serum tubes from each study subject. Clotted samples were left at room temperature for 30 minutes and then centrifuged at 3000rpm during 10 min to obtain the serum. The sera were collected and stored at -20°C until serological analysis.

Kits

The ELISA kits involved were commercial, high sensitivity diagnostic ones produced by Sunlong Biotech (China). Each kit covered pre-coated 96-well plates, HRP-conjugated antibodies, chromogenic substrates, wash buffers and stop solution; it was batch/lot numbered to guarantee reproducibility. Quality control was confirmed through the use of positive and negative control sera in each run.

EBV Serological Testing

EBV infection was measured on the basis of VCA-IgG, VCA-IgM and EBNA-IgG antibodies using enzyme-linked immunosorbent assay (ELISA). ELISA kits were obtained from Sunlong Biotech, China and the procedures were carried out with a Biobase ELISA microplate reader (China) as per the manufacturer's guidelines. serum samples were diluted 1:5 by adding 10µL serum to 40ÂµL of the given sample diluent was added in the microplate wells. The plates were incubated for 30 min at room temperature to permit the binding of antibodies. After incubation, the wells were washed three times in wash buffer from the kit to remove unbound materials. Then 50µL of HRP conjugate were added to all sample wells and plates incubated for 30 minutes. 50 µL of chromogenic substrates A and B were added to each well after cleaning, followed by incubation at room temperature for 15 minutes in the dark for the development of color. The reaction was stopped with 50µL of stop solution and absorbance at 450nm was recorded. Samples were classified as positive, negative or equivocal based on the manufacturer's cut-off values.

Statistical analysis

These questions were addressed using established statistical methods to contrast the EBV-infected patients and controls, along with comparing EBV antibodies, adipokines and cytokines for correlations. Receiver operating characteristic (ROC) curve analysis was performed to evaluate the diagnostic power of these adipokines in differentiating between infected and

naturally healthy groups, p values <0.05 were considered statistically significant (18,19).

Results

Demographic Characteristics of Patients and Controls

The study included 157 patients suspected of EBV infection and 75 healthy controls recruited from teaching hospitals in Kirkuk from June to September 2025. The gender distribution was almost equal, with 80 males (50.9%) and 77 females (49.1%) among patients, and 38 males (50.7%) and 37 females (49.3%) among controls. Age was similarly distributed, with the majority between 21–60 years, and BMI categories were comparable across both groups. These data indicate well-matched baseline characteristics between patients and controls, suitable for further analyses, Table 1.

Table 1. Demographic Profile of Patients and Controls

Variable	Patients (n=157)	%	Controls (n=75)	%
Gender				
Male	80	50.9	38	50.7
Female	77	49.1	37	49.3
Age group (years)				
21–40	62	39.5	30	40.0
41–60	59	37.6	28	37.3
>60	36	22.9	17	22.7
BMI category				
Normal (18.5–24.9)	55	35.0	28	37.3
Overweight (25–29.9)	68	43.3	32	42.7
Obese (≥30)	34	21.7	15	20.0

EBV Serological Detection

ELISA testing identified 45 EBV-positive patients (28.6%). The majority of positives were VCA-IgG only (27 patients, 17.2%), while VCA-IgM alone or combined markers indicated recent or active infection in a smaller proportion (5.7% and 1.9%, respectively). The remaining 112 patients (71.4%) were seronegative. These results suggest that only a subset of clinically suspected cases were truly EBV-infected, Table 2.

Table 2. EBV Serological Markers in Patients (n=157)

Serological Marker	Positive (n)	%
VCA-IgG only	27	17.2
VCA-IgM only	9	5.7
EBNA-IgG only	6	3.8
IgG + IgM/EBNA combined	3	1.9
Total EBV positive	45	28.6
Seronegative	112	71.4

EBV Positivity and Demographic Factors

Among the 45 EBV-positive patients, the distribution was nearly equal between males (23, 51.1%) and females (22, 48.9%). Age analysis showed 20 patients (44.4%) were 21–40 years, 15 (33.3%) were 41–60 years, and 10 (22.2%) were older than 60. Regarding BMI, 14 patients (31.1%) had normal BMI, 22 (48.9%) were overweight, and 9 (20.0%) were obese. These data indicate that EBV infection affected both genders almost equally and was more frequent in younger adults and overweight individuals, Table 3.

Table 3. EBV Positive Cases by Demographics (n=45)

Variable	EBV Positive (n)	% of Positive Patients
Gender		
Male	23	51.1
Female	22	48.9
Age group (years)		
21–40	20	44.4
41–60	15	33.3
>60	10	22.2
BMI		
Normal	14	31.1
Overweight	22	48.9
Obese	9	20.0

Immunological Findings

EBV-positive patients displayed significantly elevated serum cytokines compared to healthy controls. IL-6 averaged 29.4 ± 6.1 pg/mL, markedly higher than 12.7 ± 3.9 pg/mL in controls ($p < 0.001$). TNF-α was 21.7 ± 5.3 pg/mL versus 9.8 ± 3.1 pg/mL in controls ($p < 0.001$), and IFN-γ was 25.9 ± 6.7 pg/mL compared to 11.5 ± 3.7 pg/mL in controls ($p < 0.001$). These findings indicate a strong pro-inflammatory immune activation associated with EBV infection, Table 4.

Table 4. Serum Cytokine Levels in EBV-Positive Patients vs Controls

Cytokine	EBV Positive (n=45)	Controls (n=75)	p-value
IL-6 (pg/mL)	29.4 ± 6.1	12.7 ± 3.9	<0.001
TNF-α (pg/mL)	21.7 ± 5.3	9.8 ± 3.1	<0.001
IFN-γ (pg/mL)	25.9 ± 6.7	11.5 ± 3.7	<0.001

Adipokines

Serum adipokine analysis showed significant reductions in EBV-positive patients compared to controls. Omentin-1 was 5.7 ± 1.2 µg/mL in positive patients versus 8.2 ± 1.3 µg/mL in controls ($p < 0.001$). Chemerin was 190 ± 21 ng/mL versus 208 ± 23 ng/mL in controls ($p = 0.01$), and Vaspin was 0.52 ± 0.12 ng/mL versus 0.61 ± 0.15 ng/mL in controls ($p = 0.02$). These results suggest EBV infection is associated with reduced adipokine levels, which may contribute to immune dysregulation, Table 5.

Table 5. Serum Adipokines in EBV-Positive Patients vs Controls

Adipokine	EBV Positive (n=45)	Controls (n=75)	p-value
Omentin-1 (µg/mL)	5.7 ± 1.2	8.2 ± 1.3	<0.001
Chemerin (ng/mL)	190 ± 21	208 ± 23	0.01
Vaspin (ng/mL)	0.52 ± 0.12	0.61 ± 0.15	0.02

Correlation Analysis

In EBV-positive patients, Omentin-1 demonstrated significant inverse correlations with IL-6 ($r = −0.38$, $p < 0.01$), TNF-α ($r = −0.31$, $p < 0.01$), and IFN-γ ($r = −0.29$, $p < 0.01$). Chemerin and Vaspin showed weaker negative correlations with cytokines. These results indicate a relationship between decreased adipokine levels and heightened inflammatory response in EBV infection,

Table 6.

Table 6. Correlation of Adipokines with Cytokines in EBV-Positive Patients

Parameter	IL-6 (r)	TNF-α (r)	IFN-γ (r)	Age (r)
Omentin-1	−0.38**	−0.31**	−0.29**	0.20*
Chemerin	−0.18*	−0.15	−0.14	0.09
Vaspin	−0.16*	−0.13	−0.12	0.08

*p<0.05, **p<0.01

ROC Analysis for Adipokines and Cytokines

Receiver operating characteristic (ROC) analysis was conducted to evaluate the ability of adipokines and cytokines to discriminate EBV-positive patients from healthy controls. Omentin-1 demonstrated strong predictive performance with an AUC of 0.78 (95% CI: 0.70–0.85, p < 0.001), a sensitivity of 73%, and specificity of 72%. IL-6 showed the highest discriminative capacity with an AUC of 0.81 (95% CI: 0.74–0.88, p < 0.001), sensitivity of 78%, and specificity of 75%. TNF-α and IFN-γ also exhibited good predictive performance, while Chemerin and Vaspin had moderate discriminative ability. These results indicate that both inflammatory cytokines and adipokines are useful markers for identifying EBV infection in this population, Table 7 & Figure 1.

Table 7. ROC Analysis of Adipokines and Cytokines for EBV Positivity

Marker	AUC	95% CI	Sensitivity (%)	Specificity (%)	p-value
Omentin-1	0.78	0.70–0.85	73	72	<0.001
Chemerin	0.66	0.57–0.74	61	64	0.03
Vaspin	0.64	0.55–0.72	58	63	0.04
IL-6	0.81	0.74–0.88	78	75	<0.001
TNF-α	0.79	0.71–0.86	76	73	<0.001
IFN-γ	0.77	0.69–0.84	74	71	<0.001

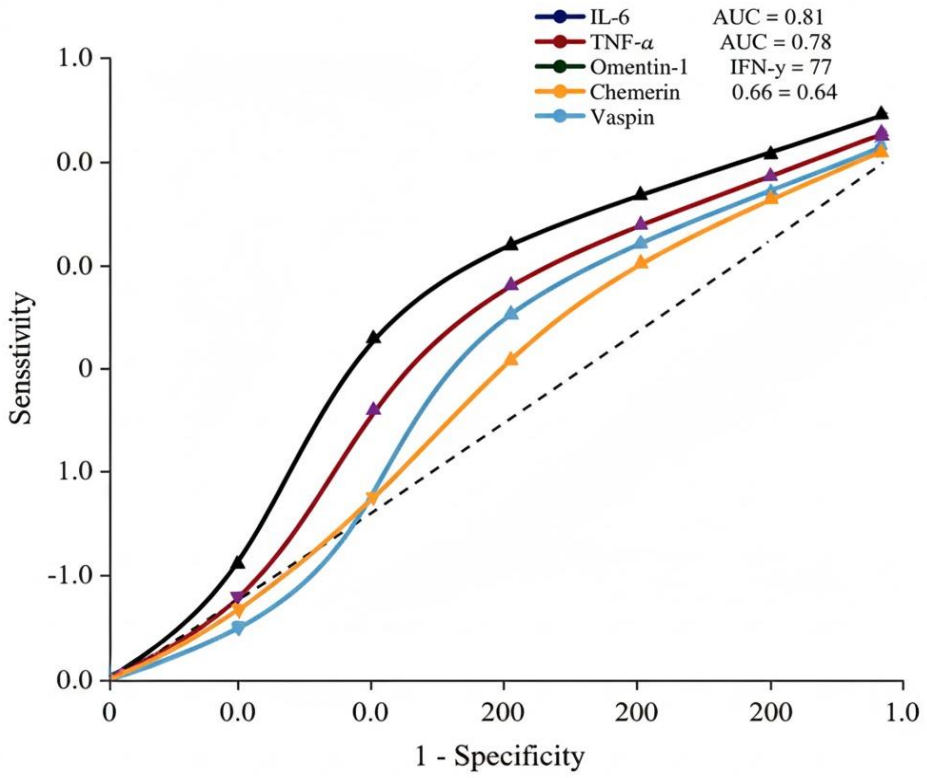

Figure 1. ROC Curve Analysis of Adipokines and Cytokines for Discriminating EBV Positivity

Discussion

EBV seropositivity was identified in 28.6% of clinic attenders, which is substantially less than the high seroprevalence observed amongst adults overall, with established IgG positivity present in most community cohorts by early adulthood (often more than 90%). Yet, also for clinical cohorts – especially those where patients are ascertained on symptoms and not screened in the population at large - a general reduced confirmed rate should be no surprise if all suspected cases are aggregating from EBV-infected individuals. In an Iraqi paediatric and adolescent population, a seroprevalence for latent EBV IgG was reported to be 29.1%, with backward links and sex-dependent differences [20]. This is consistent with the notion that EBV exposure and immunity particularly to certain antigens larger virion proteins can be influenced by age, social contact patterns, and local epidemiology. Our functional immunologic data supported this, as EBV-infected patients would have significantly higher pro-inflammatory cytokines compared to healthy controls. Namely, IL-6 was 29.4 ± 6.1 pg/mL in EBV-positive patients versus 12.7 ± 3.9 pg/mL in controls ($p<0.001$), TNF-α was 21.7 ± 5.3 pg/mL versus 9.8 ± 3.1 pg/ mL ($p<0.001$), and IFN-γ was 25,9±6,7pg/m Lcomparedto11,5±3,7pg/mL($p<0.001$). These elevations most likely reflect established mechanisms of EBV immunopathogenesis, wherein viral antigens such as latent membrane protein 1 (LMP1) and certain lytic cycle factors activate innate immune signaling cascades, in particular NF-κB and JAK/STAT pathways leading to continued cytokine expression [22]. Comparable raised levels of IL-6 and TNF-α were identified in chronic active EBV infection patients, in which both cytokines correlated with systemic inflammation and disease activity [23,24]. Our results have built on this paradigm by quantifying these responses in a clinical Iraqi population, showing that EBV infection drives robust systemic inflammation according.

The profile of the elevated cytokines is compatible with a Th1-weighted immune response in EBV infection. IFN-γ is a classic Th1 cytokine promoting macrophage activation and antiviral immunity, and its increase observed in our EBV-positive patients reflects the profile found in other viral infections needing cytotoxic T-cell assistance [25]. Increased TNF-α and IL-6 are important factors in inflammation and are linked to tissue pathology and immune dysregulation in EBV-related diseases, including infectious mononucleosis and EBV-associated lymphoproliferative disorders. One of the major innovative features of our study is investigating relationship between adipokines, including omentin-1, chemerin and vaspin, and activation status due to EBV infection. The levels of omentin-1 were significantly reduced in EBV-positive patients (5.7 ± 1.2 µg/mL) compared to their control values (8.2 ± 1.3 µg/mL, $p<0.001$). Moderate decreases were also found for chemerin and vaspin. In recent years, adipokines have been identified as immune modulators interconnecting metabolic and inflammatory pathways. Specifically, Omentin-1 has shown anti-inflammatory effects in other metabolic and autoimmune diseases, with its negative relation towards the pro-inflammatory cytokines as IL-6 and TNF-α also indicating changes related to immune activation over control homeostasis [28]. Reduced circulating omentin-1 levels have also been demonstrated in other inflammatory conditions, such as COVID-19, where low concentrations of omentin correlated positively with inflammtory markers [27]. Although adipokine pattern in viral infections has not been extensively investigated, we propose that EBV infection may interfere with the crosstalk between adipose tissue and immune cells, which could contribute to low-grade inflammation. The inverse relationships found between omentin-1 and inflammatory cytokines (for example, IL-6 $r=-0.38$, $p<0.01$) are consistent with the concept that decreased anti-inflammatory adipokines may be associated with exaggerated cytokine responses. These observations are in line with studies showing that adipokines are able to regulate infiltration, cytokine release and metabolic responses of immune cells during an inflammatory reaction [29]. Decreased omentin-1 might thus represent not only an effect of pro-inflammatory signaling, but also a mediator of chronic

immune activation, and both contribute to the generation of a self-amplifying loop that potentiates inflammation in EBV-infected subjects. When compared with other regional and international seroepidemiological studies, it is evident that the EBV exposure patterns differ between populations as well as methods to acquire the data. In a community-based study in Tehran, IgG seroprevalence to EBV had exceeded 90% by age 20 years and rose with age [30]. These high rates are in contrast to the much lower prevalence of seropositivity among our clinically based sample, because only a subgroup of clinically suspected patients tested positive. This highlights the need to distinguish between community seropositivity and clinical positivity in symptomatic patients. Most immunological studies corroborate the inflammatory phenotype of EBV infection, however, little is known about adipokine changes. The marked decrease in omentin-1 and mild decreases of chemerin, as well as the lack of change in levels of vaspin, observed in the current study may reflect a more generalized immune-metabolic dysregulation linked with chronic viral infection. These trends could also have longer-term consequences as adipokines have been associated with chronic inflammation and tissue remodeling in other instances [31]. Additional investigation is needed to investigate whether adipokine panels could predict long-term symptoms or disease sequelae post-an EBV infection.

Conclusion

EBV infection in Iraqi patients is related to an exaggerated pro-inflammatory response and a marked decrease of serum omentin-1 supporting the connection between immune activation and adipokine imbalance. These changes show that EBV interferes the omentin-1–immune axis and also the profile of both genes, omentin-1 and IL-6 may be possible biomarkers of infection. The results emphasize the significance of analyzing adipokines and cytokines in EVB-infected individuals for a better understanding of disease pathophysiology and early diagnosis.

Limitations

The cross-sectional nature of this study, and the restriction to only two hospitals in Kirkuk, may impact generalizability. Some adipokines and cytokines were just tested, and no longitudinal evaluation to observe the dynamic releases in response to infection was conducted. These findings will have to be validated in large, multi-center cohorts with more extensive immune profiling studies.

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