Sero-detection of Epstein Barr Virus antibodies among blood donors in Khartoum state

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Abstract
The main aim of this study was to determine the seroprevalence of Epstein Barr Virus (EBV) (VCA) IgG antibodies using enzyme linked immunosorbent assay (ELISA), among blood donors attending Blood Bank Department at National Public Health Laboratory in Khartoum State, from January to February 2015 . A total of 90 subjects were included, 75 blood donors and 15 controls with age ranging from 18-63 years. The results revealed that the prevalence of EBV (VCA) IgG in the total samples were 83(92.2%) and among blood donors was 69(92%) while 14(93.3%) in control subjects. Possible risk factors were examined in this study including age, sex, marital status, major blood groups and previous blood transfusion. All these showed no significant effect ( P> 0.05 ) on EBV IgG antibodies among both blood donors and control group.

The results obtained showed that the prevalence of Epstein Barr Virus (VCA) IgG antibodies among both blood donors and control group increased with age, while it was 58(90.6%), 13(92.8%) in male, 11(100%), 1(100%) in female, 43(93.4%), 6(85%) in single, 26(89%), 8(100%) in married and 2(100%), 0(0%) in previously blood transfusion respectively.

The results showed that EBV is endemic in Sudan. Although primary EBV infection among normal immune competent individuals is not one of the clinically serious viral infections in Sudan, but the serious complications of diseases that occur in immune compromised individuals (transplant recipients and AIDS) can not be neglected. This study recommends the screening of blood for EBV antibodies among blood and organ transplant donors.

Key words: Epstein Barr Virus (EBV), Seroprevalence, Herpesviridae.
Introduction

Herpes simplex virus type 1 (HSV-1) and type 2 (HSV-2), varicella zoster virus (VZV), Epstein-Barr virus (EBV), and cytomegalovirus (CMV) are human herpesviruses (HHV) that are prevalent worldwide, their infections can lead to a variety of clinical conditions that range in severity from cold sores and genital ulcers (HSV), and chickenpox (VZV) to potentially sight-threatening (e.g. CMV uveitis and HSV keratitis) or even life-threatening diseases such as HSV encephalitis and EBV-associated malignancies (Schaftenaar et al; 2014).

Primary HHV infections, commonly acquired at young age, lead to a lifelong latent infection with intermittent reactivation resulting in periodic asymptomatic or recrudescent disease. The host immune system is pivotal to resolve lytic infections and to inhibit HHV reactivation from latency (Schaftenaar et al; 2014).

Epstein-Barr virus (EBV) belongs to the family Herpesviridae, subfamily Gammaherpesvirinae and Genus Lymphocryptovirus. which represent the most important human pathogens (Saravani et al; 2014).

The virion is Enveloped, icosahedral nucleocapsid symmetry, spherical to pleomorphic particle, 95-105 nm in diameter, and has linear, double-stranded DNA genome about 184 kbp in length. Between the capsid and the envelope is an amorphous layer of proteins termed the tegument (Sathiyamoorthy et al; 2014).

Primary Epstein-Barr virus (EBV) infection usually occurs within the first years of life. At an early age, the infection is usually asymptomatic, whereas, during adolescence and adulthood, it can present as acute infectious mononucleosis (IM). The infection is extremely common, and > 90% of adults are seropositive for EBV (Helminen et al; 2015).

The infection spread through salivary contact, and the mucosal epithelium of the oropharynx is considered to be the first site of infection.
and replication. From the oropharynx, the virus is transmitted to locally infiltrating B cells, where it persists for a person’s life (Helminen et al; 2015).

It is an oncogenic virus associated with a wide array of human tumors including epithelial cell tumors such as nasopharyngeal and gastric carcinomas, and lymphoid malignancies like Hodgkin and Burkitt lymphoma. Subsequent reactivations of the virus are asymptomatic and managed effectively by the immune system in healthy adults. However, immunocompromised patients suffer from severe opportunistic disorders, such as post-transplant lymphoproliferative disease (PTLD), oral hairy leukoplakia and HIV/AIDS related malignancies (Sathiyamoorthy et al; 2014).

**Historical background**

Epstein Barr Virus (EBV) was discovered as a result of pioneering work in the 1950s, by Denis Burkitt. Burkitt identified a previously unrecognized form of cancer which affected the jaws of young African children, and he made the crucial insight that the distribution of this common tumor (now known as Burkitt’s lymphoma) appeared to be influenced by climatic factors -- notably temperature elevation. Burkitt theorized that the tumor might be due to a mosquito-born virus, or arbovirus(Tortora et al; 2004).

This discovery led Michael Epstein, Yvonne Barr and Burt Achong to examine freshly excised tumor biopsies for the presence of a virus. In 1964, using electron microscopy, they found herpesvirus-like particles in a small number of the biopsied cells, and they subsequently established that this was in fact a new virus. Epstein-Barr virus was thus identified as the first candidate human tumor virus( Bauman; 2011).

A fourth human herpes virus is Epstein-Barr virus (EBV) which in the family Herpesviridae , subfamily Gammaherpesvirinae and Genus
Lymphocryptovirus. The official name for the species is *human herpesvirus 4*, or HHV-4 (Saravani *et al*; 2014).

**Virus replication**

The tissue tropism of EBV in vivo is mainly restricted to B lymphocytes and epithelial cells. Virus infection in B lymphocytes is mainly latent, whereas in epithelial cells, it is lytic, i.e., productive. EBV infection in B lymphocytes and epithelial cells is initiated by attachment of virions to the cell surface (Tugizov *et al*; 2013).

Viral membrane fusion is a requisite step for infection for all lipid bilayer encased viruses, such as the herpesviruses, and requires one or several virus-encoded glycoproteins that orchestrate the merging of viral and host membranes in a step-wise manner. This overall process leads to the release of viral capsid into the host cytoplasm initiating infection (Sathiyamoorthy *et al*; 2014).

The entry of EBV into B cells is complex and involves at least five different glycoproteins (EBV gp350/220, gH, gL, gp42 and gB). Of these five proteins, four (gH, gL, gB and gp42) are indispensable for membrane fusion with B cells and three (gH, gL and gB) are required for fusion with epithelial cells. gH, gL and gB form the core fusion machinery common to all herpesviruses (Sathiyamoorthy *et al*; 2014).

During B cell entry, EBV gp350/220 binds to complement receptor 2 (CR2/CD21) concentrating virus to the B cell surface, but this interaction does not activate membrane fusion or virus entry (Sathiyamoorthy *et al*; 2014).

The gp42 protein forms stable, high affinity complexes with the gHgL complex, and also binds to human leukocyte antigen (HLA) class II which acts as the triggering receptor for EBV entry into B cells (Sathiyamoorthy *et al*; 2014).
Transmission of infection

EBV replicates primarily in B-lymphocytes but also may replicate in the epithelial cells of the pharynx and parotid duct. The infection is spread primarily by saliva, and the incubation period is four to eight weeks (Ebello; 2004).

However, there are single reports of EBV detection in male and female genital secretions, suggesting the possibility of sexual transmission. A recent seroepidemiological study on university students lends support to this possibility by showing strong correlations of both EBV seropositivity and history of infectious mononucleosis (IM) with sexual intercourse and increasing numbers of sexual partners (Macsween and Crawford; 2003).

However, these data are not conclusive because they do not differentiate between direct transmission in genital secretions and spread by practices associated with sexual intercourse such as kissing (Macsween and Crawford; 2003).

Latent EBV infection of B lymphocytes in the blood of healthy donors affords another potential route of transmission. Infection has been documented after infusion of a large volume of fresh blood. Transmission from a transplanted organ can also occur with subsequent infection of a previously seronegative recipient and is a risk factor for post-transplant lymphoproliferative disease (PTLD) (Macsween and Crawford; 2003).

Pathology and pathogenicity

EBV associated diseases often arise from a failure of the host immune response to control the proliferation of latently infected cells. This is the opposite of what happens in other herpesviruses, where the problem is a failure to respond to lytic infection, and this correlates with the fact that latent infections by EBV predominate in vivo, with lytic-phase infection occurring in very few cells (Sugden; 1994).
EBV infection is usually asymptomatic in childhood, and about 90% of adults are positive for the virus. EBV is spread by saliva and the virus initially infects oropharyngeal epithelial cells, where it replicates efficiently. The virus then infects B cells, as they pass through the oropharynx and this results in the establishment of latent viral infection in B cells and persists lifelong. EBV infection of B cells is associated with the rapid proliferation and expansion of EBV+ B cells during primary viral infection (Sugden; 1994).

Normally, this EBV-driven proliferation of B cells is brought under control by cytotoxic T cells (CTLs). This commonly results in an infectious mononucleosis (IM), particularly in young adults. However, in certain individuals, the initial EBV-driven B cell proliferation is not adequately contained, and this may result in fatal IM, which occurs particularly in males with X-linked lymphoproliferative disorder (XLP) (Sugden; 1994).

During the persistent stage of EBV infection (i.e., following the resolution of the acute infection), the virus primarily infects long-lived memory B cells in the periphery. This is thought to allow the growth-promoting genes of the virus to be switched off to create a site of persistent infection in vivo, without causing disease or providing antigenic targets for the immunosurveillance (Sugden; 1994).

**Clinical significance**

As stated earlier, primary infection in infancy or childhood is usually asymptomatic, but as high as fifty percent of those infected later in life develop the disease infectious mononucleosis. Although B cells are the primary target of infection due to infection due to the presence of the EBV receptor molecule, EBV has more recently been found to be associated with a small number of T-cell malignancies as well (Strohl et al; 2001).
In patients who are immunodeficient or immunosuppressed, the lack of cell mediated immune control increases the likelihood of lymphoproliferative disorder of various kinds. Throughout life, healthy EBV carriers continue to have episodes of a symptomatic virus shedding (Strohl et al; 2001).

The source of this virus is presumably productively infected oropharyngeal cells that acquire the virus from latently infected B cells in which the lytic cycle has been activated (Strohl et al; 2001).

**Infectious mononucleosis**

The manifestations and severity of primary EBV infection vary greatly, but the typical IM syndrome appears after an incubation period of four to seven weeks, and includes pharyngitis, lymphadenopathy, increased levels of liver enzymes in the blood, fever. Headache and malaise often precede and accompany the disease, which may last several weeks. Complete recovery may take much longer (Strohl et al; 2001).

**EBV and malignancies**

Since the initial discovery of EBV in association with Burkitts lymphoma (BL), it has been shown to be associated with a number of other human neoplastic diseases (Strohl et al; 2001).

**Burkitts lymphoma (BL)**

BL was first described in 1985 as a rather unique malignancy of the jaw, found at unusually high frequency in children in regions of equatorial Africa. Distribution of Burkitts Lymphoma (see appendix-2).

Malaria and HIV infection are known risk factors for development of BL (Strohl et al; 2001).

Burkitts lymphoma is thought to result from an early EBV infection that produces a large pool of infected B lymphocytes. Such stimuli can lead to chromosomal translocation, which are pathognomonic for this lymphoma (Ahmad et al; 2010).
Nasopharyngeal Carcinoma (NPC)

It is one of the most common cancers in Southeast Asia, North Africa, and among the Eskimo population, but less common elsewhere (Strohl et al; 2001).

NPC differs from BL in that there is no characteristic chromosomal alteration, and the cells involved are epithelial in origin. The role of EBV is indicated by the fact that all cells of the tumor contain cytoplasmic viral DNA molecules (Strohl et al; 2001).

**EBV infections in immunocompromised and immunosuppressed patients**

In BL and NPC, EBV infection appears to be only one step in a multi-step, disease-causing process, and its specific role is still not well defined (Strohl et al; 2001).

In contrast, EBV alone appears to be sufficient for induction of B-cell lymphomas in immunocompromised patients, such as transplant recipients and individuals with AIDS, who cannot control the cell multiplication induced by the early protein (Strohl et al; 2001).

For example, many AIDS patients develop a B-cell malignancy of some type: BL of the sporadic type occurs with high frequency in the earlier stages of AIDS progression, where as non-BL-type lymphoplastic lymphomas are more characteristic in late stage AIDS patients (Strohl et al; 2001).

All of the HIV-associated BL cases contain the EBV genome. And AIDS patients infected with EBV may also exhibit nonmalignant, white-grey lesions on the tongue "hairy leukoplakia" (Strohl et al; 2001).

**Lymphoproliferative syndrome**

Patients with primary or secondary immunodeficiency are susceptible to EBV-induced Lymphoproliferative disease. For example, the incidence
of these lymphomas is 1% to 2% after renal transplantations and 5% to 9% after heart-lung transplantations (Ahmad et al; 2010).

The risk is greatest in patients experiencing primary EBV infection rather than reactivation. Most characteristic is persistent fever lymphadenopathy and hepatosplenomegaly(Ahmad et al; 2010).

**EBV and autoimmune diseases**

Epidemiological data suggest that EBV is associated with several autoimmune diseases, such as systemic lupus erythematosus (SLE), rheumatoid arthritis (RA) and multiple sclerosis (MS). However, it is not clear whether EBV plays a role in the pathogenesis of these diseases, and if so, by which mechanisms the virus may contribute. (Lossius et al; 2012).

A possible association between EBV seropositivity and autoimmune diseases was first observed by coincidence in 1968 in a Brazilian population. Since then, a multitude of studies have explored humoral immunity against EBV in SLE, RA and MS. North Americans of different ethnicities with SLE had an increased seroprevalence of EBV and studies in other populations have shown an increased frequency of antibodies against EBV early antigens (Lossius et al; 2012).

In one study, 99% of young SLE patients were seropositive for EBV compared to 70% of age-matched controls. Almost all adult MS patients are seropositive for EBV, compared to 90% of healthy adults. As for SLE, the differences in seroprevalence are more pronounced in lower age groups, where the general seroprevalence is lower. Further, it has been demonstrated that MS risk is very low in individuals not infected with EBV, but increases sharply after EBV infection (Lossius et al; 2012).

In a recent meta-analysis, previous EBV infection was actually found to be present in 100% of MS patients in studies using two independent methods of antibody detection. The authors claimed that findings of MS
patients without earlier EBV infection could be due to low sensitivity in the assays used for detection of antibodies (Lossius et al; 2012).

In SLE and MS, titers of antibodies against EBV antigens are elevated compared to healthy controls and for both diseases, this elevation seems to predate the first symptoms (Lossius et al; 2012).

**X-linked lymphoproliferative syndrome**

X-linked lymphoproliferative syndrome (XLPS or XLP) is a rare, familial, fatal form of IM that has been recognized for almost 30 years. Typically XLP affects young males who are clinically well before primary EBV infection, but when infected most rapidly succumb to fulminant IM (Macsween and Crawford; 2003).

**Host Response to EBV Infection**

Antibodies to early antigens diffuse (EA-D), methanol-resistant and restricted (EA-R), methanol-sensitive as well as EBNA-2 rise and fall with convalescence, while antibodies to EBNA-1 develop only after convalescence and persist at a low titer for life. Antibodies to the gp350 envelope protein, known as membrane antigen (MA), rise slowly during acute infection and persist after convalescence. The asymptomatic post-convalescent carrier state is characterized by persistent IgG antibody to VCA, MA, and EBNA-1 (Iwatsuki et al; 2004).

Virus reactivation occurring in immunocompromised virus carriers is marked by rising titers of IgG anti-VCA and anti-EA antibody, and accompanied by a rise in viral load in blood as detected by real-time quantitative EBV PCR. In addition to virus-specific antibodies, patients with EBV-associated infectious mononucleosis rapidly and transiently develop heterophile antibodies, low titer IgM antibodies of unknown primary specificity that agglutinate heterologous (sheep, horse, cow) red blood cells (Iwatsuki et al; 2004).
Heterophile antibodies are both sensitive and specific for EBV-associated infectious mononucleosis since they are not usually seen in infectious mononucleosis syndromes associated with other infections. The EBV-specific T cell response in acute primary infection (infectious mononucleosis) is dominated by CD8+ CTL with lytic antigen specificity, with a proportional increase in latent antigen specificity following recovery (Iwatsuki et al; 2004).

Lytic proteins targeted by EBV-specific CD8+ T cells are most often immediate early or early proteins rather than late proteins. The CD8+ T-cell response to latent antigens is largely targeted to EBNA-3 proteins (3A, 3B, 3C). The change in CD8+ T-cell antigen specificity likely reflects the biology of infection—acute primary infection initiated by a burst of lytic replication followed by immune suppression of lytic replication and establishment of a persistent pool of latent-infected B cells. In contrast to the CD8+ T-cell response, less is known about the CD4+ EBV-specific T-cell response during infectious mononucleosis (Iwatsuki et al; 2004).

**Laboratory identification**

The most commonly used diagnostic criterion is the presence of 50% lymphocytes with at least 10% atypical lymphocytes (large, irregular nuclei), in the blood smear of a patient with IM. The atypical lymphocytes resembled monocytes when they were first discovered, thus the term "mononucleosis" was coined. Infectious mononucleosis in peripheral smear, showing reactive lymphocytes (Murray et al; 2007) (see in appendix -2).

Detection of EBV DNA or RNA by hybridization, or of virus antigens using immunohistochemical techniques, can be done with cell homogenates or by in situ methods for visualization of individual infected cells (Strohl et al; 2001).
The "classic" test for infectious mononucleosis, the Paul-Bunnell-Davidsohn test, is based upon the fact that polyclonal stimulation of B cells by EBV infection results in a nonspecific elevation of hetrophile antibodies that specifically agglutinate horse and sheep red blood cells. These hetrophile antibodies are diagnostic for EBV-related IM, although they are not present in all cases of EBV IM (Strohl et al; 2001).

Materials and methods

This study was conducted in Blood Bank Department at National Public Health Laboratory and the experimental work was done in Research laboratory in Sudan University of Science and Technology, from January to February 2015. All blood donors attending the Blood Bank for donation of blood during the study period were considered eligible to be included in the study irrespective of age, sex and residence. Control subjects were randomly selected from apparently healthy individuals.

A total of 90 subjects were included, 75 blood donors and 15 controls to study the prevalence of EBV. The study is based on non-probability convenience sampling technique. Samples were taken from blood donors and control during the period of donation after their agreement to participate.

Data were collected according to personal structured interview. Aliquots of five ml of venous blood were collected by venous puncture after sterilizing the site of collection. The collected blood was drawn into plain containers, allowed to clot and then centrifuged at 3000 rpm for 5 minutes. Sera were separated into new sterile ependroff tubes preserved at 2°C – 8°C until used. Hemolytic or lipemic or icteric specimens were excluded.
The preserved sera for both blood donors and control group were then tested for Epestin Barr Virus IgG(VCA) antibodies using ELISA. The test kit contains micro titer strips with 8 break-off reagent wells coated with EBV-CA. In the first reaction step, diluted samples are incubated in the wells. In the case of positive samples, specific IgG antibodies will bind to the antigens. To detect the bound antibodies, a second incubation is carried out using an enzyme labelled anti-human IgG (enzyme conjugate) catalysing a colour reaction. A third incubation is carried out using chromogen/substrate solution. The reaction is then stopped by adding acid. The result of colours are read by micro ELISA reader and then the reading of samples is compared with the controls and calibrators (EUROIMMUM, Germany).

According to manufacturer guidelines (EUROIMMUM, Germany) the following steps were followed:

All reagents were brought to room temperature approximately 30 minutes before used.

Diluted washing buffer was prepared by adding 40 ml to 360 ml of distilled water in flask.

Samples were diluted by adding 10µl to 1 ml of samples diluent.

Aliquots of 100 µl of calibrators, controls and diluted sera were dispensed into appropriate wells, incubated for 30 min at room temperature (18°C to 25°C) and then washed three times using 300 µl diluted washing buffer.

100 µl of enzyme conjugate (Peroxidase-labeled anti-human IgG) were dispensed into all wells, incubated for 30 min at room temperature (18°C to 25°C) and then washed three times using 300 µl diluted washing buffer.
100µl of chromogen /substrate solution (TMB/H₂O₂) were dispensed into all wells, incubated for 15 min at room temperature. 100µl of stop solution (0.5 M sulphuric acid) were added to the wells containing chromogen/substrate solution. The colour intensity was read at a wavelength of 450 nm within 30 minutes of adding the stop solution. The test was considered valid if the range of:

- Calibrator 1 O.D. > 0.700
- Calibrator 2 O.D. > 0.140
- Positive Control Ratio 1.9 - 4.9
- Negative Control Ratio 0 – 0.7

Results were evaluated semiquantitatively by calculating a ratio of the extinction value of the control or patients sample over the extinction value of the calibrator 2. The calculation of the ratio were done according the following formula:

\[
\text{Ratio} = \frac{\text{Extinction value of the control or patients sample}}{\text{Extinction value of the calibrator 2}}
\]

EUROIMMUN recommends interpreting results as follows:

- Ratio < 0.8: negative
- Ratio ≥ 0.8 to < 1.1: borderline
- Ratio ≥ 1.1: positive

**Results**

**Detection of anti-EBV IgG among the blood donors and control group**

The results in table 4.1 demonstrated that 92% (69 out of 75) were anti-EBV IgG positive and only 8% (6) were EBV IgG negative, while the control subjects 93.3% (14 out of 15) were anti-EBV IgG positive and only 6.7% (1) was EBV IgG negative (Table 1).
Table 1 The seroprevalence of anti-EBV IgG positive cases among the blood donors and control group

<table>
<thead>
<tr>
<th>Study group</th>
<th>Anti-EBV IgG positive *</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. Examined</td>
<td>No. Positive</td>
</tr>
<tr>
<td>Blood donors</td>
<td>75</td>
<td>69</td>
</tr>
<tr>
<td>Control group</td>
<td>15</td>
<td>14</td>
</tr>
<tr>
<td>Total</td>
<td>90</td>
<td>83</td>
</tr>
</tbody>
</table>

*( P> 0.05 )

The effect of sex and marital status on EBV IgG prevalence

The results in table 4.2 revealed that there were no significant difference ( P> 0.05 ) between males or females and between single or married blood donors and control group on EBV IgG prevalence(table 2).

Table 2 The effect of sex and marital status on EBV IgG prevalence

<table>
<thead>
<tr>
<th>Target group</th>
<th>Anti-EBV IgG positive *</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Blood donors</td>
<td>Control group</td>
</tr>
<tr>
<td></td>
<td>No. Examined</td>
<td>No. Positive</td>
</tr>
<tr>
<td>Males</td>
<td>64</td>
<td>58</td>
</tr>
<tr>
<td>Females</td>
<td>11</td>
<td>11</td>
</tr>
<tr>
<td>Single</td>
<td>46</td>
<td>43</td>
</tr>
<tr>
<td>Married</td>
<td>29</td>
<td>26</td>
</tr>
</tbody>
</table>

*( P> 0.05 )
The effect of age on EBV IgG prevalence

The results in table 3 showed that the age of blood donors and control group had no significance effect (P > 0.05) on EBV IgG prevalence (in table 3).

Table 3 The effect of age on EBV IgG prevalence

<table>
<thead>
<tr>
<th>Age group (years)</th>
<th>Anti-EBV IgG positive *</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Blood donors</td>
</tr>
<tr>
<td></td>
<td>No. Examined</td>
</tr>
<tr>
<td>18-25</td>
<td>40</td>
</tr>
<tr>
<td>26-33</td>
<td>21</td>
</tr>
<tr>
<td>34-41</td>
<td>10</td>
</tr>
<tr>
<td>42-49</td>
<td>2</td>
</tr>
<tr>
<td>50-57</td>
<td>2</td>
</tr>
<tr>
<td>58-65</td>
<td>0</td>
</tr>
</tbody>
</table>

*(P > 0.05)

The effect of previous blood transfusion on EBV IgG prevalence

Previous blood transfusion showed no significant difference (P > 0.05) on blood donors and control group on EBV IgG prevalence (Table 4.4).

Table 4 effect of previous blood transfusion on EBV IgG prevalence

<table>
<thead>
<tr>
<th>Blood transfusion</th>
<th>Anti-EBV IgG positive *</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Blood donors</td>
</tr>
<tr>
<td></td>
<td>No. Examined</td>
</tr>
<tr>
<td>Yes</td>
<td>2</td>
</tr>
<tr>
<td>No</td>
<td>73</td>
</tr>
</tbody>
</table>

*(P > 0.05)
The effect of major blood groups (ABO and Rhesus) on EBV IgG prevalence

The results explained that the major blood groups of both blood donors and control group had no significance effect (\(P > 0.05\)) on EBV IgG prevalence, and the EBV IgG present irrespective of blood group (Table 5).

Table 5 The effect of major blood groups (ABO and Rhesus) on EBV IgG prevalence

<table>
<thead>
<tr>
<th>Target Group</th>
<th>Anti-EBV IgG positive *</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Blood donors</td>
</tr>
<tr>
<td></td>
<td>No. Examined</td>
</tr>
<tr>
<td>A</td>
<td>29</td>
</tr>
<tr>
<td>B</td>
<td>8</td>
</tr>
<tr>
<td>AB</td>
<td>2</td>
</tr>
<tr>
<td>O</td>
<td>36</td>
</tr>
<tr>
<td>Total</td>
<td>75</td>
</tr>
</tbody>
</table>

*(\(P > 0.05\))*
Discussion:

Primary EBV infection triggers a humoral immune response, an innate NK cell response, and a CTL response. Shortly after infection, rapid rise and fall of IgM anti-VCA (virus capsid antigen) antibody is soon followed by IgG anti-VCA antibody that persists at a low titer for life.

In this study, the enzyme-linked immunosorbent assay (ELISA) was used for the detection of EBV IgG antibodies in both voluntary blood donors and control subjects.

Seroprevalence of EBV IgG (VCA) was found to be almost equal in both target groups (92% blood donors, 93.3% control group).

Result is in agreement with those previously reported among blood donors by Sousa et al (2011) in Portugal (95%), Macsween and Crawford (2003) in UK (>90%), Helminen et al (2001) in Finland (90%), Saravani et al (2014) in Iran (>90%), Hurme and Helminen (1998) in Finland (95%) and by Lazda (2006) in USA which was (94%).

The results obtained in this study demonstrated that EBV IgG develops with progression of age and this agree with Lazda (2006). The study also showed that there was no significant difference (P > 0.05) between males or females, between single or married blood donors and control group on EBV IgG prevalence.

Although previous repeated blood transfusion were associated with increased risk of EBV transmission, this study did not show significant effect of this risk factor on EBV infection among both target populations.

In fact only 2 individual from blood donors received blood transfusions, a very low number that can not reflect the real effect of this important predisposing factor, since there is no previous study in the effect of the major blood groups on EBV infection and transmission. This study revealed no effect of the ABO and Rhesus blood groups of the two target groups on the EBV prevalence, indicating no direct association of this factor to EBV infection and transmission.
Conclusion:

Based on the result of this study it concluded that EBV is not only common, but highly endemic in Sudan. Although not significant, but some predisposing factors showed potential effects on EBV infection.

References


