



Original Research Article

Distribution of Herpes Simplex virus Type 1 IgG antibodies in Kaduna metropolis

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ABSTRACT

This study was to determine the prevalence rate of herpes simplex virus type-1 in Kaduna Metropolis. Herpes simplex virus type 1 causes vesicular lesions of the oral mucosa can also cause clinical diseases including the genitalia, liver, lung, eye, and central nervous system which may be severe, particularly in the setting of immunosuppression. Forty (40) to 85% of all Africans carry the antibodies to HSV-1. Between 10 to 20% of that population suffer recurring outbreaks. There is very little public awareness of Herpes in Kaduna State, Nigeria. Three hundred and eighty four (384) samples were collected and analyzed using commercial ELISA kit obtained from institute virion/serion GmbH Germany. The analysis was carried out according to the manufacturer's instruction, and the result read at 450nm and 630nm using Sigma Diagnostic EIA multi-well reader II. Of the 384 samples tested 381 (99.2%) were anti- HSV-1 positive. The male were 226 with 99.1 % and female 158 with 99.3 anti- HSV-1 seropositive. In this study, it can be seen that HSV-1 was found to be 99.2% meaning that HSV-1 is highly endemic in Kaduna State, Nigeria.

Keywords

Herpes Simplex virus Type 1; prevalence; ELISA; Cold Sores; Herpetic gingivostomatitis.

Introduction

Herpes Simplex Viruses are enveloped, double stranded DNA viruses whose genome encodes approximately 80 genes. (McGeoch *et al.*, 2006). There are at least 25 viruses in the family Herpesviridae. Eight or more herpes virus types are known to infect man frequently (Richard, 2008). Herpes viruses are a leading cause of human viral diseases, second only to influenza and cold viruses. They are

capable of causing overt disease or remaining silent for many years only to be reactivated (Richard, 2008).

Herpes Simplex virus Type 1 (HSV-1) is responsible for "cold sores" which are known as fever blisters (Nellissery *et al.*, 2007). Infection with HSV1 is almost universal. This is known because, many infections are sub-clinical, and virtually

100% of adults have antibodies in their serum and most individuals become infected in the first few years of life (Diana, 2007). Herpes Simplex Virus Type 1 (HSV -1) is usually acquired through direct contact with infected lesions or body fluids, typically saliva (David, 2012). The prevalence of HSV -1 infection increases progressively from childhood. Primary HSV-1 infections in children are either asymptomatic or following an incubation period of about one week gives rise to mucocutaneous vesicular eruptions (Vittone *et al.*, 2006;Gonzalez and Sanjuan,2013). Herpetic gingivostomatitis typically affects the tongue, lips, gingival, buccal mucosa and the hard and soft palate. Most primary oro-facial HSV infections are caused by HSV-1 (Vittone *et al.*, 2006). Following the initial episode, the virus moves away from the nerve endings up into portions of the nervous system close to the lips. The HSV -1 virus remains in the body for the remainder of the person's life. What causes approximately one-third of those initially infected to suffer from recurrent cold sores is unknown. However, for those individuals who do suffer from recurrent cold sores, certain triggers such as fever condition will initiate the development of a cold. Sore (Bedadala *et al.*, 2007).

Oral-facial herpes simplex virus type-1 (HSV-1) infection is a common, worldwide problem. Research has shown that between 50 to 80% of all Americans and 40 to 85% of all Africans carry the antibodies to HSV-1. Between 10 to 20% of that population suffer recurring outbreaks. Even though a person may visibly show no signs of recurrence, it is possible for a person to pass the HSV-1 virus. A staggering number of people, even those who have been infected for years, are unaware that these oral/facial

outbreaks are a form of Herpes, and are therefore highly contagious. There is very little public awareness, education, or discussion of Herpes. Most people are infected with the virus by the time they are 10 years old, the incidence of infection steadily increases with age, reaching 80 to 90 percent among adults 50 years of age and older (Sternberg *et al.*, 2006).

Studies on the prevalence of HSV-1 antibodies in children show variations for children at age of 4 to 5 years, the prevalence of 20% was reported in England (Kangro *et al.*, 1994), 25% in Sweden (Tunbck *et al.*, 2003), 30% in Germany, around 35% in Estonia (Uuskula *et al.*, 2004), 50% in Hong Kong (Kangro *et al.*, 1994). In the following years, there was an increase in all studies to between 37% (Sweden) and 75% (Estonia).

Antiviral treatment very early in the course of the disease may decrease the length of recurrences. However, there is no satisfactory treatment for HSV-1 infection; as long as the virus remains in some cells in a latent form, antiviral drugs cannot rid the body of infection. The development of agents capable of preventing HSV -1 production of micro RNAs is an area of great scientific interest. Such agents would cause the virus to become active, rendering it susceptible to existing antiviral agents that could then cure infection (Encyclopedia Britannica, 2007).

Geographic and socioeconomic differences affect the frequency of HSV infections, so the acquisition patterns of these infections vary greatly in different areas and populations (Kangro *et al.*, 1994). Continuous changes in societies might therefore affect these acquisition

patterns, and frequent studies would therefore be useful for monitoring purposes (Kangro *et al.*, 1994). Obviously, improving the etiological diagnosis of these infections might avoid unnecessary therapy and would allow for preventive isolation of infected patients (Gulen *et al.*, 2007)

Materials and Methods

Herpes Simplex Viruses are enveloped, double stranded DNA viruses whose genome encodes approximately 80 genes. (McGeoch *et al.*, 2006). There are at least 25 viruses in the family Herpesviridae. Eight or more herpes virus types are known to infect man frequently (Richard, 2008). Herpes viruses are a leading cause of human viral diseases, second only to influenza and cold viruses. They are capable of causing overt disease or remaining silent for many years only to be reactivated (Richard, 2008).

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gingivostomatitis typically affects the tongue, lips, gingival, buccal mucosa and the hard and soft palate. Most primary orofacial HSV infections are caused by HSV-1 (Vittone *et al.*, 2006). Following the initial episode, the virus moves away from the nerve endings up into portions of the nervous system close to the lips. The HSV -1 virus remains in the body for the remainder of the person's life. What causes approximately one-third of those initially infected to suffer from recurrent cold sores is unknown. However, for those individuals who do suffer from recurrent cold sores, certain triggers such as fever condition will initiate the development of a cold. Sore (Bedadala *et al.*, 2007).

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Antiviral treatment very early in the course of the disease may decrease the length of recurrences. However, there is no satisfactory treatment for HSV-1 infection; as long as the virus remains in some cells in a latent form, antiviral drugs cannot rid the body of infection. The development of agents capable of preventing HSV -1 production of micro RNAs is an area of great scientific - interest. Such agents would cause the virus to become active, rendering it susceptible to existing antiviral agents that could then cure infection (Encyclopedia Britannica, 2007).

Geographic and socioeconomic differences affect the frequency of HSV infections, so the acquisition patterns of these infections vary greatly in different areas and populations (Kangro *et al.*, 1994). Continuous changes in societies might therefore affect these acquisition patterns, and frequent studies would therefore be useful for monitoring purposes (Kangro *et al.*, 1994). Obviously, improving the etiological diagnosis of this infections might avoid unnecessary therapy and would allow for preventive isolation of infected patients (Gulen *et al.*, 2007)

Result and Discussion

Of the total number of 384 samples tested, 381 (99.21%) were positive for herpes simplex virus type 1. Two hundred and twenty six (226) male were tested of and

224(99.1%) were observed to be seropositive. Of the 158 females tested 157(99.4%) were observed to be positive for anti-HSV-1 (Table 1). The age group 6 to 9, 13 to 15, 19 to 21 and 22 and above had the highest seroprevalence of 100% followed by 16 to 18 with 99.3% while the age group 10 to 12 had the least seroprevalence of 94% as shown in table 2.

A total of 384 blood samples were tested for the presence of herpes simplex virus type-1 antibodies and out of these number, 381 (99.21%) were seropositive. Diana (2007) had earlier reported that virtually 100% of adults have antibodies in their sera and most individual become infected in the first few years of life.

The distribution of herpes simplex virus type-1 IgG antibody according to gender shows that out of the total number of 226 blood samples analysed from males, 224 (99.1%) were observed to be seropositive and 158 females tested revealed that 157 (99.3%) were observed to be positive. The $X^2=20.064$, P value = 0.0002($P<0.05$) indicated that HSV-1 infection is not associated with gender. This may be because Herpes Simplex virus type 1 is worldwide spread and the virus is transmitted primarily by close contact, although it can survive for several hours on plastic and cloth, therefore, the saliva of people with no symptoms can be infectious and the greatest risk of infection is from contact with lesions or saliva from patient within a few days of disease onset.

The distribution of herpes simplex virus type-1 in various age group shows that age groups 16-18 had the highest seroprevalence of 36.72% followed by group 13-15 with the seroprevalence of 22.39% and age group 6-9 had the least

Table.1 Distribution of Herpes Simplex Virus Type – 1 IgG According To Gender

Sex	Number of subjects tested	Seropositive	% seropositivity
Male	226	224	99.11
Female	158	157	99.36
TOTAL	384	381	99.21

Chi square analysis indicated no association between occurrence of disease and sex ($X^2=0.0058$, $P = 0.810 > 0.05$)

Table.2 Distribution of Herpes Simplex Virus Type – 1 IgG Antibody According To Age

Age group (years)	Number of subjects tested	Seropositive	%Seropositive
6 – 9	29	29	100
10-12	33	31	94
13-15	86	86	100
16-18	142	141	99.3
19-21	59	59	100
22 and above	35	35	100
TOTAL	384	381	99.21

Chi square analysis indicated an association between the occurrence of disease and age group ($X^2=20.064$, $P \text{ value} = 0.0002 < 0.05$)

seroprevalence of 7.5%. The seroprevalence in the various age groups was observed to vary significantly and this agrees with the work of Vittone *et al*, (2006) which stated that the prevalence of HSV-1 infection increases progressively from childhood and also an antibody titre showed a decrease in titre with an increase in age. This may be due to the primary infections in this population occurring mostly during the first few years of life. Primary infection occurs early in life and is usually asymptomatic and this agrees with the work of Bedadala *et al.*, (2007) in their work titled “Early growth response gene-1 regulates HVS-1” stated that herpes simplex virus type-1 remain in the body for the remainder of the person’s life what causes approximately one-third of those initially infected to suffer from recurrent cold sores is unknown. However, for those individuals who do suffer from

cold sores, certain triggers will initiate the development of cold sores.

From this study, it can be seen that HSV-1 has a prevalence of 99.21%. Indicating that HSV-1 is endemic in Kaduna metropolis. It is therefore necessary to adopt measures to prevent and reduce the rate of Herpes Simplex Type 1 in Nigeria. There is the need to organize public health lectures through mobilization of Local Government Health workers and community effort, in order to enlighten the public on prevention of transmission, diagnosis and treatment of the disease.

References

- Bedadala, G.R., R.C.Pinnoji and Hsia, S.C. 2007. Early growth response gene 1 (Egr-1) regulates HSV-1 American. J.Virol.546 (55): 328-339

- David, R ., 2012. Herpes Simplex Virus". Integrative Med. Elsevier Health Sciences: 165.
- Diana, H., 2007. Virology Lectures to 3rd Year Medical Students. Department of Medical Microbiology, University of Cape Town.
- Encyclopaedia Britannica, 2007. Herpes Simplex Virus Type-1 & 2
- Gonzalez, M., and Sanjuan, N.A. 2013. Striated muscle involvement in experimental oral infection by herpes simplex virus type 1. *J.Oral. pathol.Med.* 42 (8): 577-647.
- Gulen, F.C., Z. Cicek, E. Kurugol, O. Demir, R. Ozdemir, R. Tanac, and Karatos, T .2007. Herpes Simplex Viruses Therapy, *J.Med.* 352: 222~226
- Kangro, H. O., 1994. Seroprevalence of antibodies to human herpes viruses in England and Hong Kong. *J. Med. Virol.* 43:91-96.
- McGeoch, O.J., F.J.Rixon, and Davison, A.J. 2006. Topics in herpes virus genomics and evolution. *Virus Res.* 117 (1): 90-104.
- Nellisery, J.K., R. Szczepaniak, C. Lamberti, and Weller, S.K. 2007. A putative leucine zipper within the HSV-1 UL6 protein is required for portal formation. *J.Virol.* 22: 74-78.
- Richard, H., 2008. Microbiology and Immunology On-Line. The Board of Trustees of the University of South Carolina, School of Medicine. Chapter 11.
- Sternberg, M.R., F. Xu, B.J. Kottiri, G.M. McQuillan, F.K. Lee, A.J. Nahmias, S.M. Berman, and Markowitz, L.E. 2006. Trends in herpes simplex virus type 1 and type 2 seroprevalence in the United State, *J. Med.Virol.*19: 293-310.
- Tunbck, P., 2003. Prevalence of herpes simplex virus antibodies in childhood and adolescence: a cross-sectional study. *J.Med. Virol.* 35: 498-502.
- Uuskula, A., M., F.M. Nygard-Kibur, P. Cowan, R.S. Mayaud and French, J. N. R. 2004. The burden of infection with herpes simplex virus type 1 and type 2: seroprevalence study in Estonia. *J. Virol.*36: 727-732.
- Vittone, V., E.Diefenbach, O. Triffett, M.W., Douglas, A.L. Cunningham and Diefenbach, R.J. 2006. Determination of interactions between tegument proteins of herpes simplex virus type 1. *J. Virol.* 79 (15): 9566-9571.