

Full Length Research Paper

Prevalence of human immunodeficiency virus (HIV) infection among pregnant women in an ante-natal clinic in Port-Harcourt, Nigeria

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Women attending ante-natal clinic in Nigeria are routinely screened for HIV/AIDS. A retrospective study was conducted between 2000 and 2004 to investigate the prevalence of the human immunodeficiency virus (HIV) infection among pregnant women attending ante-natal clinic in Braithwalte Memorial Hospital (BMH), Port Harcourt, Nigeria. Data on positive cases of HIV infection within the duration were retrieved from the hospital's record department. A total of 10,032 pregnant women were screened for the possible occurrence of HIV 1 and HIV 2 within the period. The results shows that a total of 595 (5.93%) of the pregnant women tested positive to the HIV. The year 2001 had the highest prevalence of 138 (1.38%), while the year 2000 had the least prevalence of 89 (0.89%). Analysis of the age distribution of the infection among the studied pregnant women in the hospital showed that women in the age group of 41-45 had the highest prevalence rate (80%), followed by women in the age group of 31-35 with an occurrence rate of 20.83%. The least rate of occurrence was observed in the age group of 26-30 which showed only 3.14%.

Key words: HIV prevalence, pregnant women, antenatal clinic.

INTRODUCTION

The human immunodeficiency virus (HIV) was discovered in 1983 (two years after the disease AIDS was described) when Barre-sinoussi, Montagnier and colleagues at the Institute Pasteur, Paris, France, isolated the virus from the T cells of a patient with generalized lymphadenopathy and gave it the name Lymphadenopathy associated virus (LAV, now HIV1.). In the same year, Robert Gallo and colleagues, working at the National Cancer Institute (NCI), USA made a similar discovery while in their quest to find cancer-causing viruses. In 1986 a second closely related virus, termed HIV 2 was isolated from a patient from West Africa suffering from acquired immunodeficiency syndrome (AIDS) (Adler, 1992; Hardie, 1999; Noel, 2005). In just over two decades the virus has killed more than 20 million humans and

infected over 42 million people globally with the latest yearly infection rate of over 6 million (Mahender, 2006).

HIV is one of several complex retroviruses in the genus lentivirus. The virus contains three genes required for a replicating retrovirus-gag, pol and env. About three additional genes regulate viral expression and are important in disease pathogenesis *in vivo* (Brooks et al., 2002). These other gene products include the Tat, Rev, and Nef regulatory proteins that are translated from spliced mRNA species. The HIV structure consists of a lipoprotein surface studded by about 72 envelop knobs consisting of the glycoproteins gp120, the surface (SU) protein and gp41, the transmembrane (TM) protein. Inside this lipid bilayer is a matrix (MA) protein (p17). Below the matrix is the nucleoprotein made up of a capsid (CA) protein (p24, p25). Inside the cores are various cone-shaped nucleocapsid proteins (p9, p7), as well as polymerase enzyme containing reverse transcriptase (RT),

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p63), the protease (PR,p15) and the integrase (IN), p11 (Levy, 1993).

HIV is transmitted through blood products (including blood transfusions, intravenous drug abuse in sharing of needles, health care workers in needlestick injuries and mucocutaneous exposures), organ transplants, sexual intercourse (both homosexual and heterosexual exposures) and vertical transmission (10-40% of babies born of infected mother will be infected). Infection may occur *in utero*, during birth, post nately or through breast feeding. There is no evidence to suggest that HIV can be transmitted by insects, casual contact (saliva, kissing) or by sharing of eating and drinking utensils (Brooks et al., 2002). The virus enters the cell by fusing with the cell membrane. This event involves an interaction with a cellular receptor (the major one being CD4) followed by conformational changes in the viral envelope to permit virus-cell fusion. The latter may involve cleavage of the envelope gp120 by a cellular protease (Levy et al., 1997).

Clinical signs of acute (primary) HIV infection include: a red, slightly raised rash, lymphadenopathy, low or high-grade fever, night sweats, pains in the eyes, throats and muscles, and general malaise. Some infected individuals develop gastrointestinal and neurological symptoms (Levy et al., 1997). Following the clinical manifestations of the primary infection, individuals can be asymptomatic for up to 10 years. After the initial infection there is generally a slow but steady decrease in CD4+ cell numbers, a parameter usually used to predict the onset of symptoms. The reason for the CD4+ cell loss could be as a result of direct HIV destruction of the cell, cytokine injury and immunological anti-cellular responses. When the CD4+ cell count in the blood (normally 1000) drops below 200 to 300 cells, clinical disorders such as infections and malignancies that can lead to death often appear. These diseases define the condition called AIDS. It is important to recognize that the virus itself does not directly cause these diseases; they result from HIV-induced immune destruction (Dunn et al., 1992). Gynaecological problems in women positive for the virus include chronic vaginal candidiasis, vaginitis, colpitis genital folliculitis and dermatitis, herpes genitalis, cervical atypia, chronic pelvic infection and menstrual abnormalities (Adrian, 1992)

AIDS is not curable but it can be prevented and given the massive social and economic implications of HIV infection and AIDS-related deaths, this study was designed to provide empirical evidence of prevalence among apparently healthy persons as well as to create awareness as to the threat of HIV/AIDS and its possible consequences to maternal and infant health in the Nigeria environment in order that appropriate steps could be taken to prevent any nosocomial infection. It will also expose the need to provide adequate medication to pregnant women who have tested positive to HIV/AIDS infection.

MATERIALS AND METHODS

The study population

The sample population included a total of ten thousand and thirty two pregnant women who attended the ante-natal clinic of Braithwaite Memorial Hospital, Port-Harcourt, Nigeria from the period of January 2000 to December 2004. The purpose of the study was fully explained to them (and their health care providers) and their informed consent obtained prior to the study as recommended by World Health Organization (TDR, 2002)

Sampling procedure and HIV screening

All blood samples sent to the HIV unit of the hospital laboratory with completed and certified forms from clinicians were processed by standard operating procedure to check for HIV among the pregnant women. The ELISA (enzyme-linked immunosorbent assay) test was commonly employed to screen the pregnant women, while the western blot method was used to confirm all positive cases. In the ELISA test (using ELISCAN HIV 1/2 from M/S Ranbaxy Diagnostics, New Delhi), the patients serum was added to a microplate well to which HIV antigens derived from HIV grown in human T lymphocytes have been attached. This mixture was then incubated for about 3 h. All antibodies to HIV present in the serum, bound to the HIV antigens. The plate was then washed to remove all other components of the serum. Subsequently an antihuman globulin together with an enzyme capable of altering a colour substrate were added and allowed to bind to the bound HIV antibody. Finally, the colour substrate was added. A colour change occurred if HIV antibody was present in the patients' serum (Levy et al., 1997). The Western blot was the primary method used to confirm all positive cases. In the analysis (using Qualicode HIV 1/2 from Immunetics Inc. USA), individual proteins of the HIV were separated by electrophoresis through a gel material. The proteins were then transferred (blotted) from the gel onto a nitrocellulose filter. The patient's serum was added to the filter and any antibodies to HIV bind to the specific proteins. Enzyme-labelled antibodies to human immunoglobulin were then added. These bound to any anti-HIV antibodies that attached to the nitrocellulose filter after reacting with viral proteins. All un-bound antibodies and proteins were removed by rinsing the filter. Detection of the bound complex was revealed by adding a colourless substance that the antibody-bound enzyme converted to a colour product. The "3 band rule" was used to interpret the result of the western blot procedure. If no viral bands were detected, the result was considered negative. Results were interpreted as positive if bands appeared at the site of two or more of the following HIV antigens: p24, gp41 or gp120. The test was considered indeterminate if fewer than two of bands were present (Mckane and Kandel, 1996).

RESULT AND DISCUSSION

Table 1 presents the annual distribution of HIV among pregnant women attending Braithwaite memorial Hospital Port-Harcourt, Nigeria between January 2000 and recorded in year 2001 with 138 (1.38%), followed by December 2004. A total of 595 (5.93%) pregnant women tested positive to the virus. The highest cases were 2004 with 135 (1.36%). The least cases were recorded in year 2000 with 89 (0.89%). There was no significant difference ($P < 0.5$) in the prevalence of HIV between 2001 through year 2004. However a noticeable significant diff-

Table 1. Annual distribution of HIV among pregnant women attending Braithwaite Memorial Hospital, Port Harcourt, Nigeria.

Year	Jan	Feb	Mar	Apr	May	Jun	Jul	Aug	Sept	Oct	Nov	Dec	Total
2000	21	4	6	7	3	2	15	10	3	12	3	1	89
2001	8	15	13	25	5	1	9	7	16	2	23	14	138
2002	16	1	7	4	1	11	20	12	2	5	9	24	112
2003	5	4	8	1	22	12	2	17	16	6	12	16	121
2004	18	9	7	7	1	1	7	6	13	14	16	26	135
Total	68	33	44	44	32	32	53	52	52	39	63	81	595

Table 2. Age distribution of HIV prevalence among pregnant women in Braithwaite Memorial Hospital, Port-Harcourt, Nigeria.

Age interval	Total screened	HIV positive	HIV negative	% positive	
				Within grp	Within pop
15-20	519	50	469	9.63	0.50
21-25	3,762	180	3,582	4.78	1.75
26-30	3,820	120	3,700	3.14	1.20
31-35	480	100	380	20.83	1.00
36-40	1,441	137	1,304	9.51	1.37
41-45	10	8	2	80	0.08
Total	10,032	595	9,437		

erence ($P < 0.5$) occurred between the year 2000 and other years. A greater number (81) of the pregnant women were diagnosed of HIV in the months of December representing 0.81% and January (68) representing 0.68% while the least cases (33) were diagnosed in the months of February representing 0.33% and May with (32) representing 0.32% cases during the period of study.

Table 2 shows the age distribution of HIV among the pregnant women. A significantly higher number of the infected women fall in the age bracket of 41-45 years (80%) even though this age bracket had the least sample size of 10; while the least prevalence occurred in the age group of 26-30 (3.14%) in spite of the fact that the age group had the largest sample size.

While the prevalence rate was low in year 2000, there were significant increases in the prevalence as the year progressed. It is possible that in year 2000, enough public awareness had not yet been created about the threat of HIV/AIDS, so some of the women escaped proper screening. Similarly, there was high prevalence in the months of December and January. This may be associated with the seasons of Christmas and New Year when many women indulge in commercial sex business to provide life's necessities for their families. From Table 2, it will be observed that the age group of 26-30 had the least prevalence (3.14%) despite the high number screened. This is followed by 21-25 (4.78%). These age groups were made up, mostly of newly married women,

who may be presumed to still be in their "honeymoon" and therefore very faithful to their husbands. However the same may not be said of women aged 41-45 years where, despite the low number screened, the prevalence rate was high (80%) enough. This could be due to the fact that the women in this group are more exposed to the risk factors that predispose to the infection. The fact that the age group of 15-20 (teenagers) had a relatively low prevalence (9.63%) could mean that the Nigerian governments widely - publicized anti-AIDS campaign urging them to 'zip-up' till marriage is yielding positive results.

Infection with HIV represents something of an immunological paradox, in that HIV induces a strong antiviral immune response, whilst simultaneously and progressively disrupting the ability of the immune system to respond to new infections and antigens, ultimately leading to severe immune deficient of the cell-mediated immune system (Browning, 1996). HIV, unlike other viruses has a unique ability to escape detection by the immune system (McMichael, 1996). This is however made possible by the replication machinery of the virus which is so inaccurate that it generates new mutants for virtually every virion produced in an infected individual, thus, creating a myriad of new and unique viral particles every day. These mutant viruses keep continually damaging or killing the cells of the immune system (mainly CD4+ lymphocytes) and, thus progressively destroy the body's ability to fight opportunistic infections and certain

cancers resulting in AIDS and finally death in 7 to 10 years (Mahender, 2006).

Seropositive women or women with seropositive sexual partners are at increased risk of acquiring AIDS. If they become pregnant, their offsprings are also at high risk of acquiring AIDS (Brooks et al., 2002). In a recent study, Pietro et al. (2006) confirmed that occult hepatitis B virus (HBV) infection which we (Obi et al., 2006) found to be highly prevalent among pregnant women in Nigeria, could be reactivated by infection of HIV.

AIDS is neither water borne disease nor an air borne but a problem deeply rooted in peoples' life style. In an attempt to support the effort of our government and indeed other global organizations and governments in combating the menace of HIV/AIDS, we hereby recommend that since HIV cannot be detected ("AIDS no dey show for face") except one is screened for it; every one must be screened to establish their HIV status. Any one ignorant of his/her HIV status should be assumed positive until proved otherwise through HIV screening. Any sexual intercourse (outside of mutually monogamous HIV antibody- negative relationships) should be protected by a condom. All unmarried people should abstain totally from sexual intercourse. The sharing of needles and syringes must be avoided. All blood for transfusion purposes must be screened for the presence of the HIV 1 and HIV 2 antigens. Health care personnel must adhere strictly to infection-control guidelines. All women who have been potentially exposed should seek HIV antibody testing before becoming pregnant and if the test is positive should consider avoiding pregnancy. HIV infected mothers should avoid breast feeding to reduce the risk of trans-mission of the virus to their children. Adequate care, counseling and management of those already infected i.e. people living with HIV/AIDS (PLWHA). And finally, the government at all levels should create employment opportunities so that those driven into commercial sex business due to poverty could have an alternative and decent means of living.

REFERENCES

- Case files from the Bellevue Hospital centre at New York University.
- Adler M (1992). ABC of AIDS, Group B. 3rd edition. Fortune publishing company, USA. pp: 1-7, 40-44, 60-66.
- Adrian M ((1992). HIV and AIDS management by the primary care team. 1st edition. Oxford university press, London. pp: 18-20.
- Brooks GF, Butel JS, Morse SA (2002). Jawetz, Melnick and Adelbergs Medical Microbiology. 22nd edition. McGraw-Hill USA. pp: 516-529
- Browning M (1996). Antiviral immunity. www.virology.net
- Dunn DT, Newell ML, Aedes AE (1992). Risk of human immunodeficiency virus types 1 transmission through breast feeding. Lancet 240: 585-588.
- Hardie D (1999). Human reteroviruses. www.virology.net.
- Levy JA (1993). structure of the human immunodeficiency virus. Microbial. Rev. 57:183.
- Levy JA, Heinz FC, Owens RA (1997). Virology 3rd edition. W: B. Saunders company Philadelphia pp: 372-381, 404.
- Mahender S (2006). No vaccine against HIV yet- are we not perfectly equipped? Virol. J. 3: 60-75
- McKane L, Kandel J (1996). Microbiology: Essentials and Applications, 2nd edition. McGraw-Hill. Inc USA, pp 465-468.
- McMichael A (1996). How HIV fools the immune systems. www.virology.net.
- Noel H (2005). AIDS at 20: A look back, A look ahead. www.umm.edu
- Obi RK, Umeh SC, Okurede OH, Iroagba II (2006). Prevalence of hapathitis B virus infection among pregnant women in an antenatal clinic in Port-Harcourt, Nigeria. Afr. J. Clin Exp. Microbiol. 7(2): 78-82.
- Pietro F, Coppola N, Pisapia R, Carlo S, Marrocco C, Zaccariello A, Cesare N, Sagnelli C, De Stefano G, Ferraro T, De Stefano C, Sagnelli E (2006). Impact of occult hepatitis B virus infection in HIV patients naive for antiretroviral therapy. J. int. AIDS Soc. 20(9): 1253-1260
- TDR (2002). Work book for investigators. UNDP/World Bank/WHO Special Programme for Research and Training on Tropical Disease. (TDR/PRD/GCP/02). pp: 30
- WHO/UNAIDS (2004). Report on the global AIDS epidemic. <http://www.unaids.org>