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Relationship Between Patients Showing Multiple Lymphadenopathy and HIV Infectionin Onitsha Metropolis with Respect to Age

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Abstract: Four hundred and seventy eight (478) individuals who exhibited some manifestation of chronic and debilitating illness including persistent cough, skin cancer and dermatitis, multiple lymph adenitis, diarrhea and enteritis, genital sore, urethritis, vaginitis and weight loss were examined to establish relationships between human immuno-deficiency virus infection (HIV) infection and multiple lymph adenopathy (MLA) and HIV infection. Statistical comparison for MLA in HIV positive and negative individual with respect to Age groups and testing at $P \leq 0.05$ showed no significant difference in age group of 10-19 years, 20-29, 50-59 and 60-69 years. There were significant differences in Age group of 30-39 years and 40-49 years with more MLA in HIV positive individuals.

Key words: MLA · HIV · Age and Onitsha

INTRODUCTION

Multiple lymph adenopathy (MLA) was rare before HIV epidemic [1]. It occurred mainly in lymphomas, patients with congenital or acquired hypoglobulinaemia and Epstein Bar Virus infection [2]. Infection by HIV renders their victims immuno-incompetent resulting in proliferation of opportunistic infection that overwhelms the lymphatic system which is part of antibody generating mechanism leading to axial, cervical and inguinal enlargement [3-6]. The study aimed at establishing or not, a relationship between multiple lymphy adenopathy and HIV.

HIV is a member of the genus *Lentivirus*, part of the family *Retroviridae*. *Lentiviruses* have many morphologies and biological properties in common. Many species are infected by *lentiviruses*, which are characteristically responsible for long-duration illnesses with a long incubation period [4]. *Lentiviruses* are transmitted as single-stranded, positive-sense, enveloped RNA viruses. Upon entry into the target cell, the viral RNA genome is converted (reverse transcribed) into double-stranded DNA by a virally encoded reverse transcriptase that is transported along with the viral genome in the virus particle. The resulting viral DNA is then imported into the cell nucleus and integrated into the cellular DNA by a virally encoded integrase and host co-factors. Once integrated, the virus may become latent, allowing the virus and its host cell to avoid detection by the immune system [3]. Alternatively, the virus may be transcribed, producing new RNA genomes and viral proteins that are packaged and released from the cell as new virus particles that begin the replication cycle anew [6].

AIDS is a condition in humans in which progressive failure of the immune system allows life-threatening opportunistic infections and cancers to thrive. Without treatment, average survival time after infection with HIV is estimated to be 9 to 11 years, depending on the HIV subtype. Infection with HIV occurs by the transfer of blood, semen, vaginal fluid, pre-ejaculate, or breast milk. Within these bodily fluids, HIV is present as both free virus particles and virus within infected immune cells [1, 2].

HIV infects vital cells in the human immune system such as helper T cells (specifically $CD4^+$ T cells), macrophages and dendritic cells. HIV infection leads to low levels of $CD4^+$ T cells through a number of mechanisms, including apoptosis of uninfected bystander cells, direct viral killing of infected cells and killing of

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infected $CD4^+$ T cells by CD8 cytotoxic lymphocytes that recognize infected cells [3, 5]. When $CD4^+$ T cell numbers decline below a critical level, cell-mediated immunity is lost and the body becomes progressively more susceptible to opportunistic infections.

Two types of HIV have been characterized: HIV-1 and HIV-2. HIV-1 is the virus that was initially discovered and termed both LAV and HTLV-III. It is more virulent, more infective and is the cause of the majority of HIV infections globally. The lower infectivity of HIV-2 compared to HIV-1 implies that fewer of those exposed to HIV-2 will be infected per exposure. Because of its relatively poor capacity for transmission, HIV-2 is largely confined to West Africa [6].

MATERIALS AND METHOD

Sampled Population: Individuals under study were four hundred and seventy eight (478), some showed multiple lymph nodes at axial, cervical and inguinal regions, others showed only signs and symptoms of HIV including weight loss, diarrhea, persistent fever and malaise. They were referred patients from Government General Hospital and Private Hospitals and patients coming to FEZI Medical Laboratory by references.

Sample Collection: Samples for HIV infection, ELISA Western blot analysis, CD4 Count were taken as described. Samples for other relevant tests including Hb, ESR and WBC were collected as previously described.

All individuals under test were examined clinically for presence or absence of lymph nodes at the cervical, axial and inguinal regions supervised by a Consultant Physician, Dr. P. Obiegbu (Director of Health Anambra State).

Analysis of Samples: Analysis of blood samples viz CD4 Count and Western blot were done by the kits from, ELISA (Savyon Diagnostic, Ashdod, Israel), Bio Rad Novopath Immuno Blot, Paris France. The four hundred and seventy eight individuals were physically examined by a consultant physician.

Pictures of the lymph nodes enlargements were taken by means of a Camera. Records of age were taken.

A statistical analysis of values of prevalences of MLA in HIV positive and negative individuals were carried out at (P0.05) with respect to age.

RESULTS

HIV Positive: Age group 40-49 years recorded highest prevalence of 6.49% next was 30-39 years with 6.47%, next was 10-19 years 3.28%, next was 20-29 years 1.68%, 50-59 years and 60-69 years each recorded zero.

HIV Negative: Age group 30-39 years recorded highest prevalence 2.34% next was 20-29 years 1.68%, 40-49 years came third 0.93%. Age group 10-19 years, 50-59 years, 60-69 years scored zero each.

Statistical comparison for MLA in HIV positive and negative individuals with respect to age groups and testing at P=0.05 showed no significant difference in age group of 10-19 years, 20-29 years, 50-59 years and 60-69 years. There were significant differences in age group of 30-39 years and 40-49 years with more MLA in HIV positive individuals.

DISCUSSION

Age group 40-49 years recorded highest prevalence of 6.49% followed by age group 30-39 years 5.47%. There was no significant difference in occurrence of MLA in HIV positive and negative cases for age groups 10-19

Factor									
AGE GROUP	Total number of cases divided by total number of tested	% Prevalence	Total number of HIV positive	Total number of cases divided by total number of tested	% Prevalence	Total number of HIV Negative	Total number of HIV positive and negative Chi. Sq. Value		Significant at P≤0.05
10-19	$^2/_{61}$	3.28		$^0/_{61}$	0			1.034	Not Sig
20-29	$^2/_{119}$	1.68		$^2/_{119}$	1.68			0	Not Sig
30-39	$^7/_{128}$	5.47		$^3/_{128}$	2.34			4.86	Sig
40-49	$^7/_{108}$	6.49		$^1/_{108}$	0.93			4.67	Sig
50-59	$^0/_{53}$	0		$^0/_{53}$	0			0	Not Sig
60-69	$^0/_9$	0		$^0/_9$	0			0	Not Sig
Total			169			309	478		

years, 50-59 years, 60-69 years. However, this was not so for age groups 30-39 years and 40-49 years which showed significant difference in MLA occurrence between HIV positive and negative cases.

In HIV positive individuals, the prevalence of MLA generally increased with age from 1.68% in 20-29 years to 6.49% in 40-49 years group, but absent in 50 years and above. Whereas HIV negative individuals show no obvious pattern of MLA with respect to age.

Multiple lymph adenopathy seems to be more prevalent in the adolescent and middle ages.

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