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Relationship Between Patients Showing Multiple Lymphadenopathy and HIV Infection in Onitsha Metropolis with Respect to Location

O. Chukwuezi Fabian

Department of Biological Sciences,
Faculty of Applied and Natural Sciences, Tansian University, Umunya

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 (HIV) and multiple lymph adenopathy (MLA) infection. Statistical comparison of lymph adenopathy in HIV positive and negative individuals with respect to location and testing at P=0.05 showed no significant difference in Onitsha North, South, East and West.

Key words: Onitsha · HIV · MLA and Location

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].

Inflammation as a cause of lymph node enlargement is known as lymphadenitis [1]. In practice, the distinction between lymphadenopathy and lymphadenitis is rarely made. Inflammation of the lymphatic vessels is also known as lymphangitis [2]. Infectious lymphadenitides affecting lymph nodes in the neck are often called scrofula.

Due to its peculiar high incidence, the presence of lymphadenopathy is a particularly important sign on the diagnosis of HIV or even, untreated later stages of the infection, AIDS.

Lymph node enlargement is recognized as a common sign of infectious, autoimmune, or malignant disease.

Less common infectious causes of lymphadenopathy may include bacterial infections such as cat scratch disease, tularemia, brucellosis and prevotella.

The human immunodeficiency virus (HIV) is a lentivirus (a subgroup of retrovirus) that causes HIV infection and acquired immunodeficiency syndrome (AIDS) [3].

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 [5 and 6].

HIV infects vital cells in the human immune system such as helper T cells (specifically $CD4^+$ T cells), macrophages and dendritic cells [6]. 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 infected $CD4^+$ T cells by CD8 cytotoxic lymphocytes that recognize infected cells [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

Corresponding Author: O. Chukwuezi Fabian, Department Of Biological Sciences,
Faculty of Applied and Natural Sciences, Tansian University, Umunya.

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].

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. 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].

The study aimed at establishing or not, a relationship between multiple lymphy adenopathy and HIV.

MATERIALS AND METHODS

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 Lab by references.

Sample Collection: Samples for HIV infection, ELISA Western blot analysis, CD4 Count were taken by venepuncture and the serum collected by centrifugation in sterile test tubes.

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 were carried out for HIV using Savyons Diagnostics Ashdod Israel and Bio Rad Novopath Immune Blot Paris France for HIV screening and confirmatory test respectively. 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 location were taken.

Statistical analysis of the values of prevalence of MLA in HIV positive and negative individuals were carried out at P=0.05 with respect to location.

RESULTS

HIV Positive: Onitsha East recorded the highest prevalence 4.72%, next was Onitsha North 4.59% followed by Onitsha West 3.26%, least was Onitsha South 2.69%.

Table 1: Distribution of Multiple Lymph Adenopathy (MLA) among Hiv Positive and Negative Individuals in Onitsha and Their Relationship with Respect to Location

Factor								
Location	Total no. of cases/total no. tested	% Prevalence	Total no. of HIV positive	Total no. of cases/total no. tested	%	Total no. of HIV Negative	Total no. of HIV pos. and neg	Chi. Sq. Value	Sig.at P=0.05
O.N	5/109	4.59		2/109	1.83			3.68	No Sig
O.S	4/150	2.67		3/150	1.33			0.0176	No Sig
O.E	6/127	4.72		1/127	0.78			4.17	No.Sig
O.W	3/92	3.26		0/92	0			1.55	No Sig
Total	18		169			309	478		

There was no significant difference between HIV positive and negative in MLA with regards to locations ON, OS and OW, except OE where there was significant difference in MLA prevalence in HIV positive and negative individuals.

Key: ON=Onitsha North, OS = Onitsha South, OE= Onitsha East and OW=Onitsha West.

HIV Negative: Onitsha North recorded the highest prevalence 1.83% followed by Onitsha South 1.33% next was Onitsha East 0.78%, Onitsha West recorded zero.

Statistical comparison of lymph adenopathy in HIV positive and negative individuals with respect to location and testing at P=0.05 showed no significant difference in Onitsha North, South, East and West.

DISCUSSION

Generally even distribution of multiple lymph adenopathy in those with symptoms and disease in Onitsha suggests that MLA with HIV was not location specific. Onitsha North 4.59%, Onitsha East 4.72% and Onitsha West 3.26%. There was no statistical significant difference in occurrence of MLA positive and negative individuals.

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