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Distribution of Multiple Lymph Adenopathy (MLA) among HIV Positive and Negative Individuals in Onitsha and Their Relationship with Respect to Gender

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Abstract: Four hundred and seventy eight (478) individuals who exhibited some manifestations 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 immune-deficiency virus (HIV) infection and multiple lymph adenopathy (MLA) with respect to gender. There was no significant difference in occurrence of MLA in males for HIV positives and negatives. There were four females with more prevalence in HIV positives.

Key words: HIV • Multiple lymph adenopathy • Gender 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 immune-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 Moore et al., 2003 [2,4-7]. The study aimed at establishing or not, a relationship between multiple lymphy adenopathy among HIV positive and negative individuals in Onithsa with respect to gender. Lymphadenopathy or lymphadenitis refers to lymph nodes which are abnormal in size, number or consistency [8] and is often used as a synonym for swollen or enlarged lymph nodes. Common causes of lymphadenopathy are infection, autoimmune disease and malignancy [9].

Inflammation as a cause of lymph node enlargement is known as lymphadenitis [10]. In practice, the distinction between lymphadenopathy and lymphadenitis is rarely made. Inflammation of the lymphatic vessels is also known as lymphangitis [11]. 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) [12].

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 [13-16].

HIV infects vital cells in the human immune system such as helper T cells (specifically CD4⁺ T cells), macrophages and dendritic cells [17]. 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 [18]. 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 [19].

HIV is a member of the genus Lentivirus, part of the Retroviridae. Lentiviruses have family manv 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 [20]. 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 cofactors. 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 [18].

MATERIALS AND METHODS

Sample 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: Sample for HIV infection ELISA, Western blot analysis, CD4 Count were taken as described. All individuals under test were examined clinically for presence or absence of lymph nodes at the cervical, axial and analysis of the blood samples for HIV screen and Western blot confirmatory test were done by ELISA (Savyon Diagnostic, Ashdod Israel, Bio Rad Novopath Immuno Blot Paris France respectively. The four hundred and seventy eight individuals were physically examined by a consultant physician.

Pictures of the lymph enlargements were taken by means of a Camera. Records of Gender A statistical analysis of values of prevalences of MLA in HIV positive and negative individuals were carried out at P=0.05 with respect to Gender

There was no significant difference in occurrence of MLA in males for HIV positive and negative. There was significant difference in occurrence of MLA in females that were HIV positive.

RESULTS

HIV Positive: Females recorded higher prevalence rate 10.99% than males 10.26%

HIV Negative: Males recoded higher prevalence than females 2.53% and 1.32% respectively.

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

	HIV Positive			HIV Negative					
Factor									
	Total number of case divided		Total number	Total number of case divided	1	Total number	Total number of HIV		Sig. at
Gender	by total number of tested	%	of HIV Positive	by total number of tested	%	of HIV Negative	Positive and Negative	Chi square value	P=0.05
Male	2 236	3.39		4 236	1.69			1.374	Not sig.
Female	10 242	4.13		2 242	0.83			5.476	Sig.
Total	18		169	6		309	478		

Statistical compassion for MLA in HIV positive and negative individuals with respect to gender and testing at P=0.05 showed significant difference for MLA in HIV positive and negative with more MLA in HIV positive for both sexes. When MLA in males and females that were HIV positive and also HIV negative were compared statistically, there was no different in occurrence.

DISCUSSION

In HIV positive individuals, women were more at risk 4.13% prevalence, makes 3.39%. HIV negatives males were more at risk 1.69%. Females had 0.83% prevalence, in this study. For both HIV positive and HIV negative individual in the study, there was significant difference in MLA occurrence with move cases of MLA in HIV positive cases. However, when MLA males and females with HIV positive were compared there was no significance difference. Similarly, there was no significant difference in MLA occurrence in males and females with HIV negative.

REFERENCES

- Aiuta, F., J.T. Carotri, R.A. Coombs, M. Cooper, H. Dickler, S. Froland, H.G. Fundenbergh, J. Natving, Pred L. Homme, E. Rabelino, R.E. Ritts, D.S. Rowe, M. Seligman, Segel F.F. Sijern, J. Waw, W.D. Terry and J. wibran, 1975. Identification, enumeration and isolation of B and T Lymphocytes from human peripherial bood. International union of immunological societies report. Clin Immunol and Immunopathol., 3: 584-9.
- 2. Brusch, J.I. and I. Weinstin, 1988. Fever of unknown Origin. Med Clin. North AM., 72: 1247.
- 3. Centers for disease control, 1982. Persistant generalized lymph adenopathy among homosexual males. M.M WR., 31: 307-11.
- 4. Grierson, H. and D.T. Purtili, 1982. Estin Bar virus infections in males will the X-Linked lymphoprolifertive syndrome. Ann Inter Med., 106: 538-40.
- Metroka, C.E., 1985. Generalized lymph adenopathy in homosexuals Inem New York mason publishing coy USA Inc.
- Moore, S.N., J.W. Schneider and H.S. Schaaf, 2003. Diagnostic aspect of cervical lymph adenopathy in children in the developing world; a study of 1977 surgical specimens. Paediatric. Surgery Int., 19(4): 240-4.
- Rosembum, M.I., R.M. Levy and D.E. Bredesen, 1985. Primary central nervous system Lymphom in patients with AIDS. Ann. Neurol., 23 suppl., pp: 513.

- King, D., J. Ramachandra and D. Yeomanson, 2014. Lymphadenopathy in children: refer or reassure. Archives of disease in childhood. Education and practice edition, 99: 101-110.
- Fontanilla, J.M., A. Barnes and C.F. Von Reyn, 2011. Current diagnosis and management of peripheral tuberculous lymphadenitis". Clinical Infectious Diseases, 53(6): 555-562.
- Klotz, S.A., V. Ianas and S.P. Elliott, 2011. Cat-scratch Disease. American Family Physician, 83(2): 152-155.
- 11. Butler, T., 2009. Plague into the 21st century. Clinical infectious diseases, 49(5): 736-742.
- Zhang, C., S. Zhou, E. Groppelli, P. Pellegrino, I. Williams, P. Borrow, B.M. Chain and C. Jolly, 2015. "Hybrid Spreading Mechanisms and T Cell Activation Shape the Dynamics of HIV-1 Infection". PLOS Computational Biology, 11(4): 1004179.
- Jolly, C., K. Kashefi, M. Hollinshead and Q.J. Sattentau, 2004. "HIV-1 cell to cell transfer across an Env-induced, actin-dependent synapse". Journal of Experimental Medicine, 199(2): 283-293.
- Duncan, C.J., R.A. Russell and Q.J. Sattentau, 2013. High multiplicity HIV-1 cell-to-cell transmission from macrophages to CD4+ T cells limits antiretroviral efficacy". AIDS., 27(14): 2201-2206.
- Sewald, X., D.G. Gonzalez, A.M. Haberman and W. Mothes, 2012. "*In vivo* imaging of virological synapses". Nature Communications, 3: 1320.
- Sigal, A., J.T. Kim, A.B. Balazs, E. Dekel, A. Mayo, R. Milo and D. Baltimore, 2011. "Cell-to-cell spread of HIV permits ongoing replication despite antiretroviral therapy". Nature, 477(7362): 95-98.
- Robertson, D.L., B.H. Hahn and P.M. Sharp, 1995. "Recombination in AIDS viruses". J. Mol Evol., 40(3): 249-59.
- Rambaut, A., D. Posada, K.A. Crandall and E.C. Holmes, January 2004. "The causes and consequences of HIV evolution". Nature Reviews Genetics, 5(52–61): 52-61.
- Perelson, A.S. and R.M. Ribeiro, October 2008. "Estimating drug efficacy and viral dynamic parameters: HIV and HCV". Stat Med., 27(23): 4647-57.
- Sodora, D.L., J.S. Allan, C. Apetrei, J.M. Brenchley, D.C. Douek, J.G. Else, J.D. Estes, B.H. Hahn, V.M. Hirsch, A. Kaur, F. Kirchhoff, M. Muller-Trutwin, I. Pandrea, J.E. Schmitz and G. Silvestri, 2009. "Toward an AIDS vaccine: lessons from natural simian immunodeficiency virus infections of African nonhuman primate hosts". Nature Medicine, 15(8): 861-865.