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Assessment of Some Haematological and Biochemical Parametrs in HIV Patients Before Receiving Treatment in Aba, Abia State, Nigeria.

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ABSTRACT

HIV is a multisysytemic disease that suppresses haematopoietic system.Assessment of haematological parameters and some biochemical parametres of HIV patients before treatment is necessary as a guide to the patients and the health care providers.The study was done in Aba, Abia State, Nigeria. Eighty (80) subjects were chosen for the study. Forty (40) subjects were HIV patients (25 females and 15 males) and forty (40) subjects were HIV non-infected persons (control) with age range of 22-53 years. The study showed significant change ($P<0.05$) in the mean values of TWBC, PCV, Hb, Neutrophil, Lymphocyte, MXD (Monocyte, Eosinophil and Basophil), Platelets, Creatinine, GOT and GPT and no significant change ($P>0.05$) in mean values of potassium ion.

Keywords: HIV, Neutrophil, TWBC, GOT, GPT and Lymphocyte.

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INTRODUCTION

HIV/AIDS is a gradual silent killer.The greatest problem with HIV/AIDS menance is that most people infected do not know and many are afraid to go for test to know their status thereby affecting many people. HIV/AIDS is a global pandemic [1]. As of 2012, approximately 35.3 million people have HIV worldwide with the number of new infections that year being about 2.3 million [2]. This is down from 3.1 million new infections in 2001 [2] of these approximately 16.8 million are women and 3.4 million are less than 15 years old (International Committee on Taxonomy, 2002) [2].

Sub-sahara Africa is the region mostly affected. In 2010, an estimated 68% (22.9) of all HIV cases and 66% of all deaths (1.2 million) occured in this region [3].This means that about 5% of the adult population is infected [3] and it is beleived to be the cause of 10% of all deaths in children [4].Here in contrast to other regions women compose nearly 60% of cases [3].South Africa has the largest poulation of people with HIV of any country in the world at 5.9 million [3].Life expectancy has fallen in the worst-affected countries due to HIV/AIDS; for example,in 2006 it was estimated that it had dropped from 65 to 35 years in Boswana(Levy,1993).Mother-to-child transmission,as of 2013, in Bostwana and South Africa has decreased to less than 5% with improvement in many other Africa nations due to improved access to antiretroviral therapy [2].South and South East Asia is the second most affected in 2010 this region contained an estimated 4 million cases or 12% of all people living with HIV resulting in approximately 250,000 deaths [3].Approximately 2.4 million of these cases are in India [3] .In 2008 in the United States approximately 1.2 million people were living with HIV, resulting in about 17,500 deaths.The US centers for Disease Control and Prevention estimated of their infection (CDC, 2011). In the United Kingdom as of 2009 there were approximately 86,500 cases which resulted in 516 deaths [7]. In Canada as of 2008 there were 65,000 cases causing 53 deaths. Between the first recognition of AIDS in 1981 and 2009 it has led to nearly 30 million death. Prevalence is lowest in middle East and North Africa at 0.1% or less, East Asia at 0.1% and Western and Central Europe at 0.2% [3].

Human Immunodeficiency virus (HIV) is a retrovirus that causes Acquired Immunodeficiency Syndrome (AIDS) (Weiss, 1993 and Douk *et al*., 2009), a condition in humans in which progressive failure of the immune system allows life-threatening opportunistic infections and cancers to thrive. Infection with HIV occurs by the transfer of blood, semen,vaginal fluids, HIV is present as both free virus particles and virus within infected immune cells.

HIV infects vital cells in the human immune system such as helper T cells (especially CD_4 T Cells), macrophages, and dendritic cells (Cunningham *et al*., 2010). HIV infection leads to low levels of CD_4 T Cells through a number of mechanisms including apoptosis of unifected bystander cells (Garg *et al*.,2012), direct viral killing of infected cells, and killing of infected CD_4 T Cells by CD_8 T Cells cytotoxic lymphocytes that recognise infected cells (Kumar, 2012).When CD_4 T Cells numbers decline below a critical level, cell-mediated immunity is lost and the body becomes progressively more susceptible to opportunistic infection.

HIV can infect a variety of immune cells such as CD_4 T Cells,macrophages,and microglial cells. HIV-1 entry to macrophages and CD_4 T Cells is mediated through interaction of the virion envelope glycoprotein(gp120) with the CD molecule on the target cells nd also with chemokine coreceptors(Chan et al,1997).Macrophage(M-tropic) strains of HIV-1, or non-syncitial-inducing strains(NSI) use the B- chemokine receptor CCR5 for entry and are, thus,able to replicate in macrophages and CD_4 T Cells (Coakley et al., 2005). Indeed,macrophages play a key role in several critical aspects of HIV infection.They appear to be the first cells infected by HIV and perhaps the source of HIV production when CD_4 T Cells become depleted in the patient. Macrophages and microglial cells are the cells infected by HIV in the central nervous system.

Sexual intercoures is the major mode of HIV transmission through the seminal fluid which is passed from a male to his sexual partner.The virions can then infect numerous cellular targets and disseminate into the whole organism.However,a selection process leads to a predominant transmission of R5 virus through thi pathway (Zhu *et al*.,1993,Wout et al.,1994 and Zhu et al.,1996).

Althouh numerous complications occur in HIV-infected patients(Anastos et al.,2012,Ajayi et al.,2009 and Coyle,1997).The most common haematological abnormalities are anaemia and neutropenia [11].Anaemia and neutropenia are generally caused by inadequate blood production because of bone marrow suppression by HIV infection mediated by abnormal cytokine expression and alteration of the bone marrow microenvironment [10,12].Anaemia in HIV-infected persons is associated with CD_4 T Cells depletion and progression to AIDS [14] and is one of the strongest predictors of HIV mortality and poor responses to antiretroviral therapy(ART)(Anastos et al.,2004).Neutropenia is frequently observed in advanced stages in HIV infection after development of AIDS,and has been associated with certain types of antiretroviral medications used to treat HIV infection.Thrombocytopenia is characterised by platelet count below 125,and also frequently occurs in HIV-infected patients()11-13.Haematological parameters mainly anaemia and leukopenia in HIV-infected ART- naive patients result in poor ART-treament outcome and otherwise strongly predict mortality [8,9,15].

Many HIV- positive people are unaware that they are infected with the virus(Kumaranaya and Watts,2001).For example, in 2001 less than 1% of the sexually active urban population in Africa had been tested and this proportion is even lower in rural population(Kumaranaya and Watts,2001).Furthermore,in 2011 only 0.5% of pregnant women attending urban health facilities were counselled,tested or receive their test result(Kumaranaya and Watts,2001).It is imperative to determine the baseline levels of some these haematological and biochemical parameters because the complications of HIV infection are multi-factorial affecting all aspect of the body system.This will gude the phsysicians in the choice of their presciption.

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MATERIAL AND METHODS

Study Area

Aba, Abia State Nigeria.

Subjects: 40 HIV infected patients (25 females and 15 males) before receiving treatment in Living Word Mission Hospital, Abayi, Aba, Abia State, Nigeria and 40 healthy individuals (25 females and 15 males) who visited the Laboratory of the hospital for other purposes were recruited for the study.

Sample and Methods

Venous blood samples were collected from the subjects into EDTA anticoagulated blood containers haematological analysis and others into plain containers for biochemical analysis.

STATISTICAL ANALYSIS

The data were analysed with t-test and statistical significance was set at $P<0.05$

Ethics: Oral consents were made to the subjects prior to sample collection.

RESULTS

Table 1:Mean Values of TWBC of The HIV Patients and HIV Non-Infected Subjects

Subjects	Mean	±SD	P-VALUE
HIV PATIENTS(40)	5.5	2.3	
HIV NON-INFECTED(40)	4.4	1.4	P<0.05

Table 2:Mean PCV Values of HIV Patients And HIV Non-Infected Subjects

Subjects	Mean	±SD	P-VALUE
HIV PATIENTS(40)	32.6	4.5	
HIV NON-INFECTED(40)	41.2	2.7	P<0.05

Table 3:Mean Haemoglobin Values of the HIV Patients and HIV Non-Infected Subjects

Subjects	Mean	±SD	P-VALUE
HIV PATIENTS(40)	9.7	3.7	
HIV NON-INFECTED(40)	14.6	1.3	P<0.05

Table 4:Mean Neutrophil Values of HIV Patients and HIV Non-Infected Subjects

Subjects	Mean	±SD	P-VALUE
HIV PATIENTS(40)	53.0	27.9	
HIV NON-INFECTED(40)	60.5	10.8	P<0.05

Table 5:Mean Lymphocyte Values of HIV Patients and HIV Non-Infected Subjects

Subjects	Mean	±SD	P-VALUE
HIV PATIENTS(40)	44.8	15.4	
HIV NON-INFECTED(40)	31.7	6.4	P<0.05

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Table 6:Mean Mxd Values of HIV Patients And HIV Non-Infected Subjects

Subjects	Mean	±SD	P-VALUE
HIV PATIENTS(40)	10.3	3.3	
HIV NON-INFECTED(40)	7.8	2.5	P<0.05

MXD=Monocytes, Eosinophils and Basophils

Table 7:Mean Platelet Values of HIV Patients and HIV Non-Infected Subjects

Subjects	Mean	±SD	P-VALUE
HIV PATIENTS(40)	174.3	28.5	
HIV NON-INFECTED(40)	230.1	10.2	P<0.05

Table 8: Mean Creatinine Values of HIV Patients and HIV Non-Infected Subjects

Subjects	Mean	±SD	P-VALUE
HIV PATIENTS(40)	136.5	10.2	
HIV NON-INFECTED(40)	93.2	7.4	P<0.05

Table 9: Mean Potasium Ion Values of HIV Patients and HIV Non-Infected Subjects

Subjects	Mean	±SD	P-VALUE
HIV PATIENTS(40)	4.1	0.5	
HIV NON-INFECTED(40)	4.0	0.7	P>0.05

Table 10:Mean Got Values of HIV Patients and HIV Non-Infected Subjects

Subjects	Mean	±SD	P-VALUE
HIV PATIENTS(40)	24.7	7.8	
HIV NON-INFECTED(40)	43.2	5.2	P<0.05

Table 11:Mean Gpt Values of HIV Patients and HIV Non-Infected Subjects

Subjects	Mean	±SD	P-VALUE
HIV PATIENTS(40)	18.4	9.1	
HIV NON-INFECTED(40)	25.1	6.4	P<0.05

DISCUSSION

Tables 1-4,7,10 and 11 showed significant decrease (P<0.05) in the mean values of TWBC, PCV, Hb, Neutrophil, Platelet, GOT and GPT and tables 6, 8 showed significant increase (P>0.05) in the mean value of potassium ion.The study is in line with Amegor *et al* (2009) where PCV value was reduced in HIV positive subjects showing mild anaemia.This confirmed generalised effects of HIV/AIDS on haematopoietic and blood cells (Amegor *et al*., 2009).This study is close to the values of PCV, Hb, and WBC of the positive patients before treatment. HIV is a systemic disorder caused by the HIV, and characterised by severe impairment and progressive damage of both cellular and humoral immune responses. Besides immunological complications of HIV disease [15], haematological abnormalities have been documented as strong independent predictors of morbidity and mortality in HIV –infected individuals [9]. The overall suppression on haematopoietic system is obvious as well as the kidney and liver.

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CONCLUSION

HIV has serious adverse supressive effect on the haematopoietic sysytem, liver and renal function, so these parameters should be noted immediately HIV patients visited hospital before treatment to help in better wholistic management of the patients.This baseline assessement should help to know if there is improvement in the treatment later on.

REFERENCES

[1] Cohen MS, Hellmann N, Levy JA, Decock K, and Lange J. The J Clin Inv 2008;118(4):1244-54.
[2] UNAIDS 2013. Reports.
[3] UNAIDS 2011.
[4] Mandell B, Doland. International Commitee on Taxonomy of Viruses, 2010.
[5] Levy JA. HIV Pathogenesis and Long-term Survival. AIDS 1993;7(11):1401-10.
[6] Centre for Disease Control and Prevention, 2011.
[7] Health Protection Agency, 2010.
[8] Munyazesa E, et al. BM J 2012;2(6).
[9] Anastos K, Shi Q, French A, et al. J Acquir Immune Def Syndr 2004;35:383-92.
[10] Obirikorang C, and Yeboah F A. J Biomed Sci 2009;16:102.
[11] Ajayi AO, Ajayi EA, and Fasakin KA. Ann Afr Med 2009;8:257-60.
[12] Aboulafia DM, and Mitsuyasu RT. Oncol Clin North Am 1991;15:195.
[13] Coyle TE. Med Clin North Am 1997;81:449-70.
[14] Mata-Marin JA, Gaytan-Martinez JE, Martinez-Martin RE. BMC Res Notes 2010; 3:230.
[15] Rudnicka D, and Schartwz O. Nat Immunol 2009;10:933-4.