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Abnormal Lipids and Acquired Immune Deficiency Syndrome: Association of Serum Lipids with HIV Status

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Abstract

Context and Aim: Hematological abnormalities are amongst the most common complications of infection with HIV. There have been quite a few studies on the alterations in lipid profile, too, though the results have largely been inconclusive. The present study was carried-out to assess CD4 cell counts and lipid profile in the HIV infected and AIDS patients in the Indian population and correlates them with the sero-negative controls.

Materials and Methods: The present study was designed as a cross-sectional, hospital-based study to assess CD4 cell counts and lipid profile in the HIV infected and AIDS patients in the Indian population and correlates them with the sero-negative controls. Evaluation of lipid profile was done using Erba EM 360, an automated analyzer powered by a diffraction grating photometer while CD4 cell counts were evaluated using Partec Cyflow Counter.

Statistical Analysis: The data was analyzed using SPSS version 15.0 (SPSS Inc., Chicago, IL, USA). Comparison of the said parameters was done using Analysis of Variance (ANOVA) and post-hoc Games-Howell test. p-value of <0.05 was considered statistically significant.

Results: The levels of total cholesterol and low-density lipoproteins (LDLs) were significantly decreased while triglycerides and very low-density lipoproteins (VLDLs) were significantly increased in the HIV infected and AIDS patients when compared with the sero-negative controls.

Conclusion: Total cholesterol, LDLs, triglycerides and VLDLs were significantly altered in the HIV infected and AIDS patients when compared with the sero-negative controls.

Keywords: CD4 Lymphocyte Count; Dyslipidemias; HIV; Acquired Immunodeficiency Syndrome; Serum; Lipids

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Introduction

AIDS is an Acronym for Acquired Immune Deficiency Syndrome caused by a retrovirus known as human immunodeficiency virus (HIV) which breaks down the body's immune system leaving the patient vulnerable to a host of life threatening opportunistic infections, neurological disorders or, unusual malignancies [1]. The two known types of this virus include the HIV-1 and HIV-2 which belong to a family of primate lentiviruses [2,3]. According to estimates by World Health Organization (WHO) and The Joint United Nations Program on HIV/AIDS (UNAIDS), 35 million people were living with HIV globally at the end of the year 2013 [4]. The first AIDS case in India was detected in the year 1986 [4]. HIV is transmitted by both homosexual and heterosexual contact, by blood and blood products, by infected mothers to infants either via intra-partum or, peri-natal routes or, via breast milk and by occupational transmission [5].

India carries the third largest number of HIV infected and AIDS patients in the world after South Africa and Nigeria [6]. In India, the highest prevalence of HIV/AIDS cases has been observed in Nagaland followed by Mizoram, Manipur and Andhra Pradesh according to the latest national AIDS statistics by National AIDS Control Organization (NACO, HIV Sentinel Surveillance 2012-13) [7]. HIV infection causes depletion of Cluster of Differentiation-4 (CD4) cells in peripheral blood and lymphoid tissues causing Cluster of Differentiation-8 (CD8) cell dysfunction. The CD4+T lymphocytes are the primary target of HIV infection because of the affinity of the virus to the CD4+ cell surface marker. Quantification of CD4 helper lymphocytes is, thus, essential in the staging and monitoring of HIV infected and AIDS patients. With reduced CD4 cell counts in HIV infection, granulocytopenia and thrombocytopenia are seen. When the counts of granulocytes fall <500 per mm^3, in the presence of an attendant anatomical barrier

damage that follows the viral infection, invasion of the bloodstream by microorganisms is facilitated with resultant sepsis and death [1,3].

Hematological abnormalities are amongst the most common complications of infection with HIV [8,9]. There have been quite a few studies on the alterations in lipid profile, too, though the results have largely been inconclusive as well as the reasons behind such alterations, too, have remained debatable [1,9]. A plethora of studies have been conducted correlating CD4 cell counts and hematologic parameters with the lipid profile in the HIV infected and AIDS patients across different parts of the world, however, very few studies have been reported from India. The present study was carried-out with the same intent to assess CD4 cell counts and lipid profile in the HIV infected and AIDS patients in the Indian population and correlates them with the sero-negative controls.

Materials and Methods

The present study was designed as a cross-sectional, hospital-based study to assess CD4 cell counts and lipid profile in the HIV infected and AIDS patients and correlates them with the sero-negative controls. The study population included 1500 subjects reporting to the Outpatient Department divided into 3 groups including:

Control Group: Consisting of 500 individuals who were healthy controls without any systemic illness;

HIV Group: Consisting of 500 patients who were diagnosed as HIV infected; and

AIDS Group: Consisting of 500 patients diagnosed as AIDS patients, depending on their CD4 cell counts.

A written, informed consent was obtained from the patients forming the study sample to participate in the study. Also, the study was sent for approval to the Ethical Committee of the Institution and permission was obtained before the start of the study. The patients at the extremes of ages, pregnant women and those on chemotherapy were excluded from the study because of possible weakened immune status. The patients who did not agree to give consent and were not willing to participate in the study were, also, excluded from the study. A detailed history was taken for each patient followed by clinical examination performed as per the protocol of Universal Precautions with the help of diagnostic instruments under artificial illumination. The findings were recorded in a specialized proforma and then, all the patients were subjected to the phlebotomy procedure to assess their CD4 cell counts and the lipid profile.

Evaluation of CD4 cell counts: 50 µl of Ethylenediaminetetraacetic acid (EDTA) anti-coagulated blood was added to 10 µl of monoclonal antibody and after 15 minutes of incubation, 1ml of No Lyse dilution buffer was added and the sample tube was attached to the Partec Cyflow Counter (Figure 1) for an automated evaluation of CD4 cell counts in the collected samples.

Evaluation of lipid profile, total cholesterol, triglycerides, high-density lipoproteins (HDLs), low-density lipoproteins (LDLs) and very low-density lipoproteins (VLDLs): Evaluation of lipid profile was done using Erba EM 360 (Figure 2), an automated chemistry analyzer powered by a diffraction grating photometer.

Statistical Analysis

The data was analyzed using SPSS version 15.0 (SPSS Inc., Chicago, IL, USA). Comparison of the said parameters was done using Analysis of Variance (ANOVA) and post-hoc Games-Howell test. p-value of <0.05 was considered statistically significant.

Figure 1: Partec Cyflow cell counter for evaluation of CD4 cell counts.

Figure 2: Erba EM 360 for evaluation of lipid profile.

Results

The distribution of patients based on age and gender as well as the distribution of male and female patients based on age is shown in Table 1. In the present study, the mean CD4 cell count in the control group was found to be 1125.38 while in the HIV group, it was seen to be 501.35 and in AIDS group, 256.41 dropping down significantly with the p-value being <0.001 as the HIV infection progressed to full blown AIDS (Table 2 and Figure 3). Furthermore, on analyzing the lipid profile in patients, a mean cholesterol level of 219.49 was observed in the control group with a mean of 219.29 in the HIV and 200.18 in the AIDS groups. The results were found to be statistically significant in this case, too, with the p-value being <0.001 (Table 3). In case of triglycerides, too, the results came-out to be statistically significant with a mean triglycerides level of 158.23 in the control group as against a mean value of 140.88 in the HIV and 167.43 in the AIDS groups (Table 3). For low density lipoprotein (LDLs), a mean LDL level of 144.09 observed in the control group with a mean of 138.47 in the HIV and 119.28 in the AIDS groups, again, made the results significant with a p-value of <0.001 (Table 3). In case of very low density lipoprotein (VLDLs), a mean VLDL level of 32.55 was observed in the control group with a mean of 32.08 in the HIV and 37.27 in the AIDS groups wherein the results were, again, found to be significant with the p-value being <0.001 (Table 3). The p-value in case of high density lipoproteins (HDLs), though,

Table 1: Distribution of patients based on age groups and gender.

Age group	Control Group				HIV Group				AIDS Group			
(in years)	Male	%	Female	%	Male	%	Female	%	Male	%	Female	%
10-20	29	5.80%	11	2.20%	11	2.20%	20	4%	6	1.20%	10	2%
21-30	79	15.80%	48	9.60%	79	15.80%	114	22.80%	70	14%	80	16%
31-40	52	10.40%	47	9.40%	81	16.20%	80	16%	90	18%	91	18.20%
41-50	71	14.20%	55	11%	41	8.20%	38	7.60%	55	11%	47	9.40%
51-60	38	7.60%	35	7%	14	2.80%	7	1.40%	31	6.20%	7	1.40%
61-70	22	4.40%	13	2.60%	9	1.80%	6	1.20%	7	1.40%	6	1.20%

Table 2: Evaluation of CD4 cell counts in the three groups.

	Group						p-value	Post-hoc test
	Control Group		HIV Group		AIDS Group			
	Mean	SD	Mean	SD	Mean	SD		
CD4 cell counts	1125.38	154.73	501.35	140.2	256.41	67.05	<0.001	C>H>A

p-value <0.001 Statistically significant.

Table 3: Evaluation of lipid profile and their mean comparison between the groups.

	Group						p-value	Post-hoc test
	Control Group		HIV Group		AIDS Group			
	Mean	SD	Mean	SD	Mean	SD		
Total Cholesterol	219.49	37.46	219.29	43.01	200.18	39.36	<0.001	C,H>A
Triglycerides	158.23	49.2	140.88	67.79	167.43	75.4	<0.001	C>H; A>H
HDLs	46.57	22.54	45.05	17.84	45.69	14.7	0.497	-
LDLs	144.09	43.44	138.47	46.48	119.28	27.89	<0.001	C,H>A
VLDLs	32.55	8.62	32.08	10.3	37.27	11.09	<0.001	A>C,H

p-value <0.001 Statistically significant.

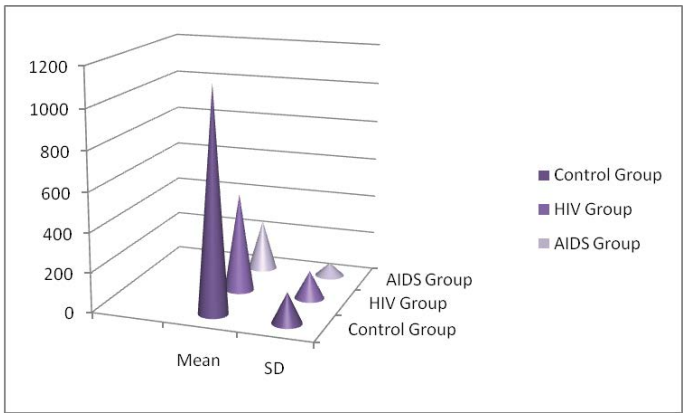

Figure 3: Mean comparison of CD4 cell counts between the groups.

was not found to be statistically significant (Table 3). To summarize, the levels of total cholesterol and low density lipoproteins (LDLs) were significantly decreased while triglycerides and very low density lipoproteins (VLDLs) significantly increased in the HIV infected and AIDS patients when compared with the sero-negative controls.

Discussion

Human immunodeficiency virus infection (HIV)/acquired immune deficiency syndrome (AIDS) are the most deadly diseases which cause devastation to the body by affecting the host's immune system. The pathogenesis of HIV infection is largely attributed to the decrease in the number of T cells (a specific type of lymphocytes) that bear the Cluster of Differentiation-4 + (CD4+) cell surface receptors. The immune status of an individual infected with HIV/AIDS can be assessed by measuring the absolute number (per mm^3) or, percentage of CD4+ cells and this is considered as the standard way to assess and characterize the severity of HIV-related immunodeficiency [8-10].

The mean CD4 cell count in the control group in the present study was found to be 1125.38 while 501.35 in the HIV group and 256.41 in AIDS group dropping down significantly with the p-value being <0.001 as the HIV infection progressed to full blown AIDS. The gradual decrease in the CD4 cell counts observed in the HIV infected and AIDS patients in the present study when compared to the controls were still higher than the mean values observed in the studies conducted by Pasupathi P, et al. (2008) [11] who recorded a mean CD4 cell count of 394 in the HIV infected and 191 in the AIDS groups and 375 in the HIV infected and 150 in the AIDS groups respectively in two different studies although the results obtained were found to be in accordance with the results of the study conducted by Tiwari BR, et al. (2008) [12] who recorded a mean value of 281 cells per mm^3 in the HIV patients [9]. The reason for the higher values obtained in the present study than as compared to most of the studies might be due to the difference in the classification of the patients into HIV infected and AIDS patients based on the CD4 cell counts. In the present study, the HIV infected and AIDS patients were categorized based on their CD4 cell counts with 10-350 and 350-500 cells per mm^3 of blood.

Tiwari BR, et al. (2008) [12] hypothesized that the fall in CD4 cell counts seen in HIV infection could be due to disruption of the cell membranes of the said cells brought-out by the budding of the infecting virus from the surface of the cells as well as the intra-cellular accumulation of the hetero-disperse RNAs and un-integrated DNAs with the progression of the disease process. Furthermore, it has, also, been proposed that an intra-cellular complexing of CD4 cells with the viral envelope products results in cell killing. Similarly, Tiwari BR, et al. (2008) [12] proposed untimely induction of a programmed cell death (apoptosis) as an additional mechanism for CD4 cell loss in HIV infection.

Hematological abnormalities are amongst the most common complications of infection with HIV [8,9]. There have been quite a

few studies on the alterations in lipid profile, too, though the results have largely been inconclusive. The present study, also, showed that the lipid profile was altered in the HIV infected and AIDS patients wherein alteration in the lipid profile occurred even during the early stages of HIV infection and more so, as the disease progressed.

The results of the present study showed that the levels of total cholesterol and low density lipoproteins (LDLs) were significantly decreased while triglycerides and very low density lipoproteins (VLDLs) significantly increased in the HIV infected and AIDS patients when compared with the sero-negative controls. Hypertrigyceridemia and a decrease in total cholesterol and HDL cholesterol occurring in advanced phases of HIV infection are considered as markers of a chronic inflammatory process as proposed by Grunfeld C (2010) [13] and Shor-Posner G, et al. (1993) [14]. However, it has, also, been proposed that highly active anti-retroviral therapy (HAART) leads to lipid changes with increases in both triglycerides and total cholesterol [15]. Infection can increase plasma triglycerides levels by decreasing the clearance of circulating lipoproteins, a process considered to be the result of reduced lipoprotein lipase (LPL) or, by stimulating hepatic lipid synthesis through increases in either hepatic fatty acid synthesis or, re-esterification of fatty acids derived from lipolysis [16].

Other factors that might contribute to dyslipidemia in HIV infection are altered cytokine profile, decreased lipid clearance and an increased hepatic synthesis of VLDLs. Cytokines such as tumor necrosis factor alpha (TNF-α) and interleukin-6 (IL-6) appear to promote lipid per-oxidation besides endothelial and platelet cell activation and the production of reactive oxygen species (ROS) [16]. An increase in serum triglycerides levels is observed in HIV infected patients as the disease progresses, particularly, in the presence of opportunistic infections possibly due to an increase in the levels of inflammatory cytokines (TNF- α, interleukins and interferon alpha [IFN- α]) and steroid hormones. The lower the CD4+ lymphocyte levels in peripheral blood are seen, the higher are the levels of triglycerides and the lower are the levels of total cholesterol and LDL cholesterol. In contrast, lower levels of LDL cholesterol are found in HIV infected patients regardless of their CD4+ T lymphocyte counts [16].

Different anti-retroviral drugs have been associated with abnormalities in the lipid profile in the HIV infected and AIDS patients. Various studies have shown an association between the use of protease inhibitors (PIs) and dyslipidemia. Young J, et al. (2005) [17] concluded that HDL cholesterol levels increase while triglycerides levels decrease with increasing exposure to non-nuclear reverse transcriptase inhibitors (NNRTIs)-based therapy. Similarly, triglycerides levels increase with increasing exposure to PI-based therapy. This might be one of the possible reasons for the patients in the present study to have increased triglycerides levels as the patients in the present study were on PIs.

Different studies carried-out in different countries have shown variations in their results on lipid profile in the HIV infected and AIDS patients. A study by Crook M (2007) [18] showed that HIV infection is normally associated with hypo-cholesterolemia, hyper-triglyceridemia and low plasma HDL cholesterol levels. Another study by Pynka ML, et al. (2004) [19] showed that there was no significant difference in total cholesterol and low density lipoprotein levels between HIV infected and healthy controls.

The results of the present study were in accordance with the results of the study conducted by Iffen TS, et al. (2010) [20] who concluded from their study an increase in the triglycerides and VLDL cholesterol in HIV infected patients compared to the controls. The probable reason given by Iffen TS, et al. (2010) [20] for the increase in triglycerides and VLDL cholesterol levels in their study was that increased tumor necrosis factor and other cytokines which occur during the said infection increase lypolysis and insulin resistance. Insulin regulates the uptake of glucose into the skeletal muscle tissue and other cells in the body. As insulin sensitivity decreases in HIV infected patient with reduction in CD4 cell counts, uptake of glucose into the skeletal muscle tissue and other cells is reduced leading to increased free fatty acids in the circulation and reduced storage of triglycerides in the adipose tissues. These free fatty acids return to the liver where they are sent back into circulation as triglycerides. Thus, significantly higher triglyceride levels are seen amongst HIV sero-positives compared to the sero-negative controls. VLDLs are composed predominantly of triglycerides. That is the reason for VLDL to be elevated when the levels of triglycerides are increased.

According to El-Sadir WM, et al. (2005) [21], patients with lower CD4 cell counts of below 200 cells per mm^3 of blood were associated with elevation in very low density lipoprotein (VLDL) cholesterol and triglycerides levels ($p < 0.05$). This observation was found to be in agreement with the findings from the present study. VLDL cholesterol carries fats around the body and elevation can increase the risk of heart disease. Grunfeld C (2010) [13] also, observed decreased total cholesterol levels in both HIV infected and AIDS patients.

The results of the present study were, also, found to be in accordance with the results of the study conducted by Pasupathi P, et al. (2010) [9] who observed decrease in serum levels of total cholesterol and low density lipoprotein cholesterol and increase in levels of triglycerides and very low density lipoprotein cholesterol in HIV infected and AIDS patients when compared to the controls as against the results obtained in the study conducted by Akpa MR, et al. (2006) [22] who found increased mean total cholesterol and LDL but decreased triglycerides and HDL levels in their study and Adewole OO, et al. (2010) [23] who observed increased total cholesterol, triglycerides and HDL levels in HIV positive patients when compared with HIV negative patients in their study. The probable reason for lack of association might be related to the close similarity in the CD4 cell counts as most patients were in the CD4 cell count range of 50-220 cells per mm^3 of blood.

Rogowska-Szadkowska D, et al. (1999) [24] and Ducobu J, et al. (2000) [25] determined the levels of plasma triglycerides, total cholesterol and HDL cholesterol levels in HIV infected patients by the level of immunological deficiency according to the CD4 cell counts and concluded that with an increase in the immunological deficiency and clinical development of HIV infection, lipid profile disorders indicated by an increase in triglycerides levels and decreased concentrations of HDL cholesterol levels intensified. The results of the present study were found to be consistent with the said studies which stated that HIV infection induced an early decrease of cholesterol and a late increase of triglycerides levels with a reduction of HDL levels. Ducobu J, et al. (2000) [25] and Crook MA, et al. (1999) [26], however, reported that patients with AIDS had increased levels of LDL cholesterol which contraindicated the results obtained in the present study. Shor-Posner G, et al. (1993) [14], also, reported similar findings in which they showed significantly low levels of total cholesterol, HDL and LDL in HIV infected patients.

Conclusion

From the findings of the present study, the present study concluded that total cholesterol, LDLs, triglycerides and VLDLs were significantly altered in the HIV infected and AIDS patients when compared with the

sero-negative controls. Further studies are, thus, mandated from across the country with correlation analyses to come to valid conclusions and manage this deadly, infectious disease process.

Limitations

The present study, also, suffered from certain limitations in the form of the duration of the disease process that was not considered in the present study while the present study did not take into consideration the pre-ART and ART patients, too, distinguishing between the patients as this was not a longitudinal study where a patient follow-up could have been done. Any co-existing disease processes which could have been significant in affecting the lipid profile in the patients could have, also, been considered and thus, pave way for future studies in this regard taking into consideration all these aspects that can be considered as the lacunae in the present study.

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