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ARTYKUŁ ORYGINALNY / *ORIGINAL ARTICLE*

The effects of CD4 nadirs on vessel stiffness in HIV patients undergoing antiretroviral therapy

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Abstract

Background: There are many factors associated with human immunodeficiency virus (HIV) patients having a greater risk of cardiovascular diseases (CVD). HIV damages vessel endothelium through chronic inflammation, which, combined with dyslipidaemia, arterial hypertension, and antiretroviral therapy leads to the progression of atherosclerotic changes.

Aim: Our goal was to determine if a CD4 nadir along with immunological, inflammatory, biochemical, and metabolic markers can be associated with higher vessel stiffness and therefore an increased risk of CVD in patients undergoing antiretroviral therapy for HIV.

Methods: Endothelial damage was evaluated in 20 patients (including four female) during successful antiretroviral therapy. We assessed the endothelial stiffness by recording the reactive hyperaemia of peripheral arteries using an Endo-PAT2000 (ITAMAR®) device. This device allowed us to measure the arterial tonometry and to determine the augmentation index for a pulse rate of 75/min (AI@75). We set the normal value for vessel stiffness at reactive hyperaemia index (RHI) > 1.67, as recommended by the manufacturer. Additionally, we recorded the length of antiretroviral therapy, number of CD4 lymphocytes, CD4 nadir, HIV viremia, and biochemical and immunologic results. Finally, we compared patients with normal and dysfunctional endothelium.

Results: The only parameter significantly differentiating between the group with and group without endothelium dysfunction was platelet count ($p = 0.012$).

Conclusions: We were not able to confirm the significance of a CD4 nadir in the progression of endothelial stiffness in HIV patients. However, platelet values could be an important complementary marker for assessing the risk for CVD amongst HIV patients undergoing antiretroviral treatment.

Key words: cardiovascular risk, tonometry, HIV

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INTRODUCTION

In 2015 UNAIDS (The Joint United Nations Programme on HIV and AIDS) estimated that 36.7 million people were infected with the human immunodeficiency virus (HIV), including 2.1 million new infections. While more than 17 million had access to antiretroviral therapy there were 1.2 million deaths amongst the HIV infected. In recent years the number of deaths caused by acquired immunodeficiency syndrome (AIDS) has decreased [1]. In Poland from 1985 to December 31st 2016 there have been 21,148 cases of HIV, 3441 cases of AIDS, and 1360 deaths recorded [2]. The most common cause of death in HIV infected patients are cardiovascular diseases (CVD). These are followed by liver and lung diseases and non AIDS defining cancer [3]. For the last 10 years CVDs are the leader among these statistics. Among risk factors of CVD we can distinguish modifiable and non-modifiable. Non-modifiable factors include genetics, cultural factors, and age. Sex is also an important non-modifiable factor as men are at a greater risk for CVD than women before menopause. However, after menopause the risk for women is similar to that for men.

The most significant modifiable risk factors are: smoking (including second hand), arterial hypertension, high cholesterol, obesity, sedentary lifestyle, diabetes, unhealthy diet, and

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alcohol abuse. Inappropriate blood lipid levels also increase the risk of stroke and heart disease. All of these factors favour an increase of inflammatory markers. The same increase is also observed in HIV patients during treatment with antiretroviral therapy [4]. The causes of these disorders are multifactorial and it is important to notice chronic inflammation caused by the HIV infection, endothelial dysfunction, blood clotting disorders, and antiretroviral drug toxicity.

The reduction of modifiable risk factors such as smoking cessation, weight reduction, controlling blood pressure, and blood sugars, as well as control electrocardiographs are crucial in the therapy of HIV patients [5].

Nitric oxide (NO) is one of the most active substances in vasodilatation throughout the entire circulatory system. Its action has a protective effect on the vessel walls mainly through the prevention of lipid oxygenation. It also decreases the effects of oxygen free radicals. NO is produced from L-arginine through NO synthase in endothelial cells; however, this pathway also occurs in platelets and cells of the central nervous system [6]. The Nef protein, which is part of HIV, induces apoptosis of endothelial cells through nicotinamide adenine dinucleotide phosphate (NADPH) oxidation. Apoptosis is directly responsible for the dysfunction of the endothelium, oxidative stress, and the increased risk of CVD, especially in patients with HIV [7]. The Tat protein also plays an important role in disturbing endothelial function through stimulating apoptosis. This protein takes part in one of the key stages of HIV replication, transcribing information from the human chromosome integrated provirus deoxyribonucleic acid (DNA) to the ribonucleic acid (RNA) template synthesising viral protein. A similar role is played by glycoprotein 120 (gp 120), which is a key molecule allowing for the attachment of HIV to T-lymphocytes and beginning the entire replication cycle [8].

The assessment of small vessel (microcirculation of peripheral arteries) tension (tonometry) before and during their reactive hyperaemia (reactive hyperaemia-pulse amplitude tonometry is one of the more commonly used non-invasive early detection methods for assessment of endothelial function [9]. Stressing the importance of this method, previous studies have shown that arterial stiffness is an independent risk factor of death in the course of CVD [10, 11]. Another method of evaluating the arterial tension is the augmentation index (AI@75) assessed for 75 heart beats per minute and pulse wave velocity (PWV).

Our goal was to determine the effects of the CD4 lymphocyte nadir, as well as other biochemical and immunological parameters, on cardiovascular risk in patients infected with HIV undergoing successful antiretroviral therapy.

METHODS

The study included adult patients with a confirmed HIV infection, successfully treated with antiretroviral drugs for a minimum of five years. Each of the enrolled patients was asked to sign an informed consent form for taking part in the study. The study proposal was accepted by the Warsaw University of Medical Sciences Bioethics Committee.

During the appointment day (AD) each patient underwent subjective and objective examination. Special care was taken to determine cardiovascular risk factors, especially smoking, obesity, arterial hypertension, and diabetes. A blood sample was taken in order to perform laboratory examination for CD4 lymphocytes, CD8 lymphocytes, platelet (PLT), mean platelets volume (MPV), white blood cells (WBC), red blood cells (RBC), total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), low-density lipoprotein (LDL), creatinine, bilirubin, alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase, gamma-glutamyltransferase (GGT), C-reactive protein (CRP), and D-dimers. The next step was to determine the reactive hyperaemia index (RHI) as well as the AI@75 based on the shape of the pulse wave using the Endo-PAT 2000. All tests were performed during the AD. Mean time between NADIR and AD in the study group was 5.4 years, ranging from 2 to 11 years.

The Endo-PAT 2000 device is used for non-invasive indirect evaluation of endothelial function and the stiffness of arteries covering microcirculation. We find the system particularly efficient. Contrary to Laser Doppler, this is an operator- and interpreter-independent method — the results are automatically analysed by the attached software. The examination is easy to perform and does not require costly diagnostic equipment, as required in methods such as NADPH fluorescence. All these features enable the Endo-PAT2000 system to deliver clear-cut results without great financial commitment.

We determined RHI > 1.67 as a norm for properly functioning endothelium. This was in accordance with the manufacturer's suggestions [12]. The AI@75 value is calculated based on the height of the two systolic pulse wave peaks. The AI was adjusted due to the significant effect of the pulse on the results. It was presented relative to the assessment of arterial stiffness at a rate of 75 bpm, commonly used in studies. Analysis was performed on all previous biochemical and immunological (NADIR) results, meaning from the day when the lowest ever CD4 T-cell values were recorded for the patient. We also analysed the duration and composition of the antiretroviral therapy for all patients. Next, we compared the results for two patient groups, one with endothelial dysfunction and one without, comparing the day the NADIR was recorded with the results from the AD.

Statistical analysis

The data was first analysed with the rho-Spearman correlation and further with the Wilcoxon and Mann-Whitney U test, comparing groups with or without endothelial dysfunction. Statistical significance was set at $p < 0.05$.

RESULTS

The study group included 20 subjects (including four female). The mean age of the study group was 43.3 years (from 25 to 74 years). We excluded from the study patients with coro-

nary disease, hypertension, diabetes, and impaired glucose toleration.

The small size of the group was compensated by the wide range of biochemical and immunological factors that we analysed in the study. Initially, we wanted the study group to include 40 patients. However, because of unsatisfying compliance we were forced to reduce its size to 20 patients.

All subjects enrolled in the study underwent successful antiretroviral therapy (VL HIV-RNA < 40 copies/mm^3). The mean duration of therapy was 6.9 years. Endothelial damage RHI ≤ 1.67 was confirmed for 6/20 (30%) of the patients.

Antiretroviral therapy contained Nucleoside and Nucleotide Reverse-Transcriptase Inhibitors (NRTIs and N[t] RTIs) in 18 patients, Non-Nucleoside Reverse-Transcriptase Inhibitors (NNRTI) in nine patients, and protease inhibitors (PI) and integrase inhibitors (INSTIs), respectively, in 13 and two patients. Significant rho-Spearman correlation level occurred between CD4, CD4%, CD8%, MPV, RBC, TC, LDL, bilirubin measured on NADIR, and AD. Correlation between CD8, PLT, WBC, TG, HDL, creatinine, ALT, AST, GGT, CRP, D-dimers measured on NADIR, and AD turned out to be insignificant. Table 1 presents correlation coefficients for the performed analyses.

In order to check if the NADIR and AD level of parameters were statistically different we performed the Wilcoxon test, as presented in Table 2. Test-Z showed statistically significant difference for parameters such as CD4, CD4%, CD8%, bilirubin, and CRP measured on NADIR and AD. For CD8, PLT, MPV, WBC, RBC, TC, TG, HDL, LDL, creatinine, ALT, AST, GGT, and D-dimers no significant difference occurred.

Next, we performed the Mann-Whitney U test in order to check the difference, with regard to the chosen parameters, between patients with a dysfunctional endothelium and those without dysfunction. PLT count was the only parameter significantly differentiating between groups with and without endothelial dysfunction. For other parameters such as CD4, CD4%, CD8, CD8%, PLT, MPV, WBC, RBC, TC, TG, HDL, LDL, creatinine, bilirubin, ALT, AST, GGT, CRP, and D-dimers no significant difference occurred. The results are presented in Table 3.

Table 1. Rho-Spearman correlation coefficients for variables from the NADIR group and AD analysed data

Variables NADIR/AD	Rho-Spearman	Significance level
CD4	0.835	< 0.001
CD4%	0.86	< 0.001
CD8	0.41	0.07
CD8%	0.78	< 0.001
PLT	0.43	0.057
MPV	0.6	0.006
WBC	0.25	0.291
RBC	0.63	0.003
TC	0.58	0.01
TG	0.31	0.203
HDL	0.46	0.062
LDL	0.54	0.031
Creatinine	0.29	0.218
Bilirubin	0.56	0.013
ALT	0.25	0.295
AST	0.20	0.394
GGT	0.29	0.235
CRP	0.001	0.997
D-dimers	0.4	0.286

ALT — alanine aminotransferase; AST — aspartate aminotransferase); CRP — C-reactive protein; GGT — gamma glutamyltransferase; HDL — high-density lipoprotein; LDL — low-density lipoprotein; MPV — mean platelets volume; PLT — platelet; RBC — red blood cells; TC — total cholesterol; TG — triglycerides; WBC — white blood cells

DISCUSSION

Amongst our patients we observed a high incidence of endothelial dysfunction and a higher cardiovascular risk in HIV infected patients. This is in accordance with other studies [13].

Ho et al. [14] confirmed that arterial stiffness and the CD4 NADIR are independent cardiovascular risk factors. Our results, despite significant statistical differences regarding the number of CD4 lymphocytes, CD4%, CD8%, bilirubin, CRP on the AD, and NADIR measurements, did not confirm the significance of these parameters in patients with and without endothelial dysfunction.

In a Brazilian study, Monteiro et al. [15] analysed the relationship between the PWV, HIV, antiretroviral therapy, HIV viraemia, CD4 NADIR, metabolic disorders and CVD. They compared the results of 261 HIV infected patients with a group of 82 healthy subjects. Amongst the HIV infected with a CD4 count < 200 cells/mm^3 the PWV was significantly higher ($p < 0.05$) than in patients with a count of CD4 lymphocytes ≥ 200 cells/mm^3. As in the study by Monteiro et al. [15], our mean number of CD4 lymphocytes recorded on the NADIR was below 200 cells/mm^3; however, we did not confirm significant differences in the number of CD4 lymphocytes in patients with and without dysfunctional endothelium. It is possible that the adopted RHI and AI@75 method of evaluating the vessel stiffness, being less sensitive than PWV, could have influenced the results of our analyses [15].

According to Chu et al. [16] higher values of MPV are associated with acute myocardial infarction, mortality post myocardial infarction, and a higher risk of restenosis after coronary angioplasty. The clinical significance of this marker is not entirely clear; however, in light of some studies MPV is correlated with arterial stiffness and related with other markers

Table 2. Wilcoxon test analysis for AD and NADIR results

Variable	Recording	Mean	Standard deviation	Z-test	Significance level
CD4	AD	368.90	279.68	**3.72**	**< 0.001**
	NADIR	164.65	156.67		
CD4%	AD	29.60	19.99	**3.42**	**0.001**
	NADIR	18.65	15.79		
CD8	AD	789.95	430.83	1.61	0.107
	NADIR	629.95	329.58		
CD8%	AD	74.15	16.73	**2.18**	**0.029**
	NADIR	67.30	19.51		
PLT	AD	215.00	60.83	0.34	0.737
	NADIR	205.40	72.73		
MPV	AD	8.93	1.05	0.04	0.968
	NADIR	9.04	1.58		
WBC	AD	6.02	2.37	1.67	0.095
	NADIR	4.90	2.16		
RBC	AD	4.07	0.68	0.32	0.747
	NADIR	4.11	0.55		
TC	AD	4.55	0.94	1.03	0.301
	NADIR	4.79	1.42		
TG	AD	1.69	0.88	1.18	0.237
	NADIR	2.06	1.59		
HDL	AD	1.24	0.38	0.28	0.778
	NADIR	1.23	0.47		
LDL	AD	2.58	0.88	0.53	0.594
	NADIR	2.60	1.06		
Creatinine	AD	75.56	14.88	1.44	0.149
	NADIR	80.16	18.87		
Bilirubin	AD	17.79	12.85	**2.11**	**0.035**
	NADIR	14.99	13.78		
ALT	AD	40.80	21.18	1.28	0.199
	NADIR	67.30	64.05		
AST	AD	77.75	124.21	1.49	0.136
	NADIR	34.30	16.95		
GGT	AD	44.32	25.99	1.25	0.210
	NADIR	71.47	66.08		
CRP	AD	6.16	9.90	**2.56**	**0.010**
	NADIR	20.28	39.91		
D-dimers	AD	726.18	684.01	0.73	0.463
	NADIR	1098.29	1224.64		

Abbreviations as in Table 1

as an important risk factor for CVD [16, 17]. We were able to determine a correlation between MPV values on the NADIR and on AD, but we were unable to confirm the statistical differences between these results, nor the significance for patients with and without endothelial dysfunction.

Platelets was the only factor statistically differentiating the higher and lower vessel stiffness groups in our study. Clotting disorders and the number and platelet activity are the subject of a number of studies and analyses. Endothelial dysfunction and platelet activity are confirmed risk factors for

Table 3. Mann-Whitney U test results

Variable	Recording	Dysfunction	Mean	Standard deviation	Z-test	Significance level
CD4	AD	Yes	385.83	358.64	0.16	0.904
		No	361.64	254.29		
	NADIR	Yes	144.67	150.83	0.21	0.841
		No	173.21	163.90		
CD4%	AD	Yes	30.50	21.09	0.41	0.718
		No	29.21	20.30		
	NADIR	Yes	19.17	16.58	0.04	0.968
		No	18.43	16.07		
CD8	AD	Yes	721.00	307.84	0.41	0.718
		No	819.50	481.35		
	NADIR	Yes	533.83	372.77	0.91	0.397
		No	671.14	314.99		
CD8%	AD	Yes	68.33	21.69	0.29	0.779
		No	66.86	19.35		
	NADIR	Yes	75.50	15.10	0.21	0.841
		No	73.57	17.89		
PLT	AD	Yes	165.83	44.22	2.47	0.012
		No	236.07	55.33		
	NADIR	Yes	163.83	52.42	1.90	0.062
		No	223.21	74.40		
MPV	AD	Yes	9.20	0.70	1.16	0.274
		No	8.81	1.18		
	NADIR	Yes	9.80	1.99	1.32	0.207
		No	8.71	1.33		
WBC	AD	Yes	5.67	2.89	0.21	0.841
		No	6.16	2.22		
	NADIR	Yes	4.72	1.70	0.21	0.841
		No	4.97	2.38		
RBC	AD	Yes	4.20	0.76	0.41	0.718
		No	4.02	0.67		
	NADIR	Yes	4.05	0.52	0.25	0.841
		No	4.14	0.58		
TC	AD	Yes	4.83	0.52	0.83	0.444
		No	4.45	1.05		
	NADIR	Yes	4.16	1.32	1.39	0.186
		No	5.02	1.43		
TG	AD	Yes	1.99	0.91	1.20	0.257
		No	1.58	0.87		
	NADIR	Yes	1.40	0.47	1.30	0.219
		No	2.30	1.80		
HDL	AD	Yes	1.30	0.39	0.42	0.721
		No	1.21	0.38		
	NADIR	Yes	1.13	0.46	0.37	0.754
		No	1.27	0.48		
LDL	AD	Yes	2.61	0.76	0.21	0.879
		No	2.63	0.96		
	NADIR	Yes	2.21	0.77	1.18	0.246
		No	2.72	1.16		
Creatinine	AD	Yes	73.00	20.39	0.66	0.547
		No	76.66	12.63		
	NADIR	Yes	83.50	19.72	0.66	0.547
		No	78.73	19.06		

→

Table 3. cont. Mann-Whitney U test results

Variable	Recording	Dysfunction	Mean	Standard deviation	Z-test	Significance level
Bilirubin	AD	Yes	16.95	14.54	0.09	0.966
		No	18.18	12.61		
	NADIR	Yes	15.17	16.08	0.13	0.898
		No	14.91	13.31		
ALT	AD	Yes	51.00	24.43	1.57	0.130
		No	36.43	18.91		
	NADIR	Yes	87.17	61.53	1.07	0.312
		No	58.79	65.42		
AST	AD	Yes	44.83	22.94	1.57	0.130
		No	29.79	12.01		
	NADIR	Yes	88.00	63.23	1.82	0.076
		No	73.36	144.71		
GGT	AD	Yes	53.17	17.10	1.93	0.058
		No	40.23	28.88		
	NADIR	Yes	53.50	21.30	0.58	0.602
		No	76.50	76.18		
CRP	AD	Yes	2.33	2.66	1.01	0.368
		No	7.92	11.55		
	NADIR	Yes	35.50	67.65	0.54	0.602
		No	12.95	17.05		
D-dimers	AD	Yes	639.69	325.15	0.24	0.905
		No	795.38	918.14		
	NADIR	Yes	1460.94	1515.06	1.79	0.082
		No	681.94	846.79		

Abbreviations as in Table 1

atherosclerosis and increase the risk of CVD. At the same time, antiplatelet therapy is one of the vital elements of prophylaxis and treating CVD in both HIV and non-infected patients [18].

Total cholesterol concentration and cholesterol and triglyceride sub-fraction are the main parameters in assessing patients at risk of CVD. These parameters have been confirmed in numerous studies. Asztalos et al. [19] analysed HIV viraemia, the number of CD4 lymphocytes, antiretroviral drugs, and the concentration of lipid sub-fraction and glucose in 176 patients. Those infected with HIV had a much lower level of large HDL particles and about three times higher level of small HDL particles, than the normal population. These parameters, however, were not significantly related to carotid intima media thickness (CIMT) assessed in that study. On the other hand, the relation of TG/HDL-C, insulin resistance, body mass index (BMI), and HIV viraemia are factors contributing to a higher CIMT. In the abovementioned study it was confirmed that the main lipid disorders observed in HIV patients are low HDL and a high level of TG caused by the blocking of cholesterol efflux from macrophages. Our results did not confirm this relation nor the significance of TC, HDL, LDL, and TG in patients with higher vessel stiffness [19].

We cannot rule out the effects of antiretroviral therapy on the results of our study. Many studies have shown that highly active antiretroviral therapy can cause serious metabolic complications regarding lipids. These are characterised by lipodystrophy, insulin resistance, central obesity, dyslipidaemia, increased risk for CVD, and increased risk for atherosclerosis. The addition of statins, fibrates, cholesterol absorption inhibitors, as well as diet and lifestyle changes to the treatment gives satisfying results [20].

Sevastianova et al. [21] presented a significant negative relation between the level of vessel stiffness and antiretroviral therapy. In their multivariate analysis, in which they performed multiple exposures to nucleoside reverse transcriptase inhibitors and protease inhibitors, they determined it to be an independent ($p < 0.05$) predictive factor of cardiovascular risk (assessed using the AI@75 method) related to higher mortality [21]. Other studies have confirmed the negative effect of protease inhibitors on endothelial functioning [22]. Lekakis et al. [23] showed an independent relationship between the duration of antiretroviral therapy, level of cholesterol, as well as blood pressure and increased arterial stiffness in HIV infected patients. Amongst our patients 90% underwent NRTI, 45% NNRTI, and PI were administered in 65% of the patients for about six years. Taking into account the high percentage of patients with endothelial dysfunction, our results may confirm the effect of antiretroviral therapy on vessel stiffness in HIV patients.

An inflammatory state is related to endothelial dysfunction in HIV patients both undergoing antiretroviral therapy

as well as those not treated. An increased concentration of high-sensitivity CRP as well as higher arterial pressure values was recorded in HIV patients in comparison to uninfected subjects. We stress the higher risk of atherosclerosis caused by a chronic inflammatory state in HIV infected patients [24–26].

In our study we determined a significantly higher concentration of CRP on the NADIR as opposed to the AD. This highlights the importance of antiretroviral therapy in limiting a chronic inflammatory state [27]. We were not able to find a significant difference of CRP in patients with and without endothelial dysfunction; nonetheless, it remains an important marker in determining the clinical state of HIV infected patients.

In a study by Hileman et al. [28] the relationship between total bilirubin concentration, interleukin 6, CRP, fibrinogen, D-dimers, oxidative stress, and endothelial function was analysed in patients successfully treated with antiretroviral therapy. In their results neither total bilirubin nor the administered atazanavir had any effect on improving endothelial function. An interesting result was presented by Li et al. [29]. Their analysis confirmed a negative correlation of bilirubin concentration with arterial stiffness [28, 29]. We were able to determine statistically significant differences of bilirubin concentration between results recorded on AD and on NADIR; however, no such significance was determined for groups with and without endothelial dysfunction.

Many studies have confirmed the importance of D-dimers in assessing vessel dysfunction, but our results did not support this. In their cross-sectional study, Hileman et al. [28] assessed endothelial function by measuring the brachialis artery dilatation induced by ischaemia (flow-mediated dilation [FMD]) as well as the concentration of biomarkers (D-dimers, inflammatory state markers, and other classic cardiovascular risk factors) in patients infected with HIV undergoing stable antiretroviral therapy (52% treated with PI) with a level of HIV-1 RNA < 400 copies/mm^3. The analysis included 98 subjects (88% male, mean age 47.5 years, CD4-578.5 cells/mm^3). The only independent factors related to FMD were D-dimer concentration and BMI, which confirmed the effect of D-dimers on endothelial dysfunction in patients successfully treated with antiretroviral therapy [30].

CONCLUSIONS

We did not confirm the importance of the CD4 NADIR and typically used markers for risk of CVD. Our results suggest the possible significance of PLT count in determining the risk of vessel stiffness in patients infected with HIV, who are successfully undergoing antiretroviral therapy.

Conflict of interest: *none declared*

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Czy nadir CD4 ma związek ze sztywnością naczyń u pacjentów zakażonych HIV leczonych antyretrowirusowo?

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Streszczenie

Wstęp: Obecnie wiadomo, że istnieje wiele czynników wpływających na zwiększone ryzyko wystąpienia chorób sercowo-naczyniowych u osób zakażonych ludzkim wirusem niedoboru odporności (HIV). HIV prowadzi do zniszczenia śródbłonka naczyń w mechanizmie indukcji przewlekłego procesu zapalnego, co razem z zaburzeniami lipidowymi, nadciśnieniem tętniczym i terapią antyretrowirusową prowadzi do progresji zmian miażdżycowych.

Cel: Celem niniejszej pracy było ustalenie, czy najniższy kiedykolwiek notowany poziom limfocytów CD4 w powiązaniu z markerami immunologicznymi, zapalnymi, biochemicznymi, a także metabolitycznymi wiąże się ze zwiększoną sztywnością naczyń, a zatem i z podwyższonym ryzykiem chorób sercowo-naczyniowych u pacjentów zakażonych HIV poddanych terapii lekami antyretrowirusowymi.

Metody: Funkcję śródbłonka oceniano u 20 pacjentów (w tym u 4 kobiet) skutecznie leczonych lekami antyretrowirusowymi. Sztywność naczyń oceniano za pomocą pomiaru reaktywnego przekrwienia tętnic obwodowych z użyciem urządzenia Endo-PAT2000 (ITAMAR®). Urządzenie to pozwoliło na ocenę sztywności naczyń obwodowych i ustalenie współczynnika wzmocnienia przy tętnie 75/min (AI@75). Jako normę przy pomiarze sztywności naczyń przyjęto współczynnik reaktywnego przekrwienia (RHI) > 1,67, zgodnie z zaleceniami producenta. Ponadto uwzględniono czas trwania terapii antyretrowirusowej, liczbę limfocytów CD4, najniższe kiedykolwiek stężenie limfocytów CD4, wiremię HIV oraz parametry biochemiczne i immunologiczne. Na koniec porównano wyniki u pacjentów z prawidłową i upośledzoną funkcją śródbłonka.

Wyniki: Jedynym parametrem istotnie różnicującym grupę z uszkodzeniem i bez dysfunkcji śródbłonka była liczba płytek krwi ($p = 0,012$).

Wnioski: Nie udało się potwierdzić znaczenia najniższego kiedykolwiek stężenia CD4 jako czynnika sprzyjającego wzrostowi sztywności naczyń u pacjentów z HIV. Jednak liczba płytek krwi może być ważnym dodatkowym markerem stosowanym w ocenie ryzyka sercowo-naczyniowego u osób zakażonych HIV poddanych terapii lekami antyretrowirusowymi.

Słowa kluczowe: ryzyko sercowo-naczyniowe, tonometria, HIV

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