

Assessment of inflammatory parameters in HIV-positive individuals under antiretroviral therapy

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Abstract

Introduction: Despite a considerable reduction of human immunodeficiency virus (HIV) viral load in people living with HIV (PLWH) due to the implementation of antiretroviral therapies, chronic immune activation persists, leading to ongoing inflammatory processes. The objective of this study was to assess the changes in inflammatory parameters during the initiation and first year of treatment among PLWH on different antiretroviral agents.

Material and methods: The study enrolled a total of 149 newly HIV-diagnosed patients between 2013 and 2022, aged above 18 years. In order to evaluate inflammatory and metabolic markers, various parameters were assessed at the initiation of treatment, including neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), mean platelet volume (MPV), plateletcrit (PCT), CD4/CD8 ratio, and hemogram.

Results: A total of 149 patients were included in the study. The mean age of patients was 36.9 years, ranging from 16 to 65 years. Notably, platelets ($p < 0.01$), MPV ($p = 0.039$), PCT ($p < 0.01$), and PLR ($p = 0.003$) indicated statistically significant changes. However, systemic immune inflammatory index (SII), systemic immune response index (SIRI), and NLR did not show statistically significant differences.

Conclusions: This study found that there is insufficient evidence to support the hypothesis that readily calculable chronic inflammation markers, such as platelet count, MPV, and PCT, may serve as valuable tools for clinicians in resource-constrained settings when monitoring HIV-infected patients.

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Key words: platelet-to-lymphocyte ratio, plateletcrit, HIV/AIDS, inflammation.

Introduction

With the improvement of antiretroviral treatments (ARTs), human immunodeficiency virus (HIV)/acquired immune deficiency syndrome (AIDS) has transitioned from fatal illness to chronic and manageable condition. While it is established

that HIV/AIDS is a disease characterized by systemic inflammation, the underlying mechanisms driving this inflammation are not yet fully comprehended. Immune activation and inflammation associated with HIV persist as long-term and systemic processes [1]. Although ART effectively suppresses HIV replication, it does not completely eliminate it.

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Consequently, residual virus replication occurs at low levels over extended periods, leading to persistent micro-inflammation.

Elevated biomarkers of inflammation, such as interleukin 6 (IL-6), tumor necrosis factor (TNF), D-dimer, fibrinogen, and C-reactive protein (CRP), have shown strong predictive values for non-AIDS events as well as for all-cause mortality in HIV-positive individuals with controlled CD4 count and HIV plasma replication while on ART. These biomarkers [2, 3], along with strategies and medications aiming at addressing HIV-1-related immune activation and inflammation, remain the focus of ongoing research efforts [4].

Blood analysis serves as easy and readily accessible method for evaluating systemic inflammation. This can be accomplished through the use of commonly available markers, such as measurable CRP, albumin, neutrophil-to-lymphocyte ratio (NLR), and platelet-to-lymphocyte ratio (PLR) [5].

Chronic inflammation has a profound impact on various leukocyte sub-types, leading to structural, functional, and numerical alterations. These changes involve the activation of neutrophils and monocytes, defects in CD4 T-cells, increased apoptosis, and heightened activation of CD8 T-cells and B-cells [6].

In recent years, several systemic inflammation markers, including NLR, lymphocyte-to-monocyte ratio (LMR), PLR, plateletcrit (PCT), and mean platelet volume (MPV), have been identified as independent predictors of prognosis and mortality in various benign and malignant systemic diseases.

NLR is recognized as an indicator of the equilibrium between the innate immune system (neutrophils) and adaptive immune system (lymphocytes). It holds significant prognostic value for a range of conditions, such as cardiovascular disease, malignancies, systemic infections, and inflammatory disorders [7-10].

MPV refers to the average size of platelets; a higher MPV value suggests an increase in platelet diameter, which can serve as an indication of both platelet production rate and activation. It has been observed that MPV levels are elevated in several cardiovascular diseases, peripheral arterial disease, and cerebrovascular disease [11]. Conversely, certain inflammatory diseases have demonstrated lower MPV levels [12].

PCT is a hematologic marker that measures the percentage of blood volume occupied by platelets. It is derived from a complete blood count, and provides insight into platelets' mass and activity. PCT is calculated using the following formula: $PCT = (\text{platelet count} \times MPV) / 10,000$ [13].

PLR is a calculated value obtained by dividing the absolute number of platelets by the absolute number of lymphocytes; it has been suggested as a potential indicator for inflammation [14].

This study investigated the changes in inflammatory parameter levels among HIV/AIDS-infected individuals receiving ART, at the beginning and after one year of treatment. The primary objective was to explore the potential utility of two markers, such as NLR and PLR, as indicators of inflammation in HIV/AIDS-positive patients.

Material and methods

In this study, a total of 149 patients who were diagnosed with HIV from July 1, 2013 to October 30, 2022 and underwent 48-week treatment until October 30, 2022, were included. These patients were receiving care at the Infectious Diseases Outpatient Clinic of Mersin University Faculty of Medicine Hospital, and their medical records were obtained from Nucleus, the hospital electronic file system. Individuals were included based on specific inclusion and exclusion criteria. Inclusion criteria: 1) patients obtaining HIV diagnosis from the Infectious Diseases Department of Mersin University Faculty of Medicine Hospital; 2) individuals admitted to the Mersin University Infectious Diseases Clinic between July 1, 2013 and October 30, 2022; 3) aged over 18 years; 4) patients without hematologic or oncologic conditions, which could potentially affect their complete blood count results. Patients aged under 18 years were excluded from the study.

Parameters used were calculated by formulas from complete blood count:

- systemic immune-inflammation index (SII) = neutrophil count $\times$ platelet count/lymphocyte count;
- systemic inflammatory response index (SIRI) = neutrophil count $\times$ monocyte count/lymphocyte count;
- NLR = neutrophil count/lymphocyte count;
- PLR = platelet count/lymphocyte count;
- PCT = platelet count $\times$ mean platelet volume (MPV)/10,000.

In addition, hemoglobin, leukocyte, lymphocyte, monocyte, neutrophil, platelet count, CRP, and erythrocyte sedimentation rate (ESR) values, were assessed. Also, changes in CD4 count, CD8 count, and CD4/CD8 ratio and blood lipid level ratios (LDL/HDL) were documented.

Patients were grouped according to their ART regimen as follows:

- abacavir, lamivudine, dolutegravir (ABC/3TC/DTG);
- tenofovir alafenamide, emtricitabine, bictegravir (TAF/FTC/BIC);
- tenofovir alafenamide, emtricitabine, cobicistat, elvitegravir (TAF/FTC/c/EVG);
- tenofovir disoproxil fumarate, emtricitabine, cobicistat, elvitegravir (TDF/FTC/c/EVG);
- tenofovir disoproxil fumarate, emtricitabine, dolutegravir (TDF/FTC/DTG).

Data of patients' 48-week treatment, both at the beginning and during the treatment, were analyzed and compared, and then subjected to statistical evaluation. The study aimed to examine the changes in chronic inflammation parameters in patients undergoing different antiretroviral therapies as well as to determine whether these changes can be useful in monitoring individuals living with HIV during follow-up.

Statistical analysis

In the study, Shapiro-Wilk test was used to assess normal distribution of variables, with descriptive statistics including

mean $\pm$ standard deviation and minimum and maximum values. For categorical variables, number (n) and percentage (%) values were evaluated. To compare two dependent variables, a paired t -test was employed. When comparing more than two groups, analysis of variance (ANOVA) test was utilized. To assess whether there were significant changes in dependent variables over time, the repeated measures ANOVA method was employed. In cases where significant differences were found between the measurements over time in the groups, the least significant difference method (a type of multiple comparison method) was applied to determine different measurements. All statistical analyses were carried out using Statistica v.13.3.1 software. A p -value of less than 0.05 was considered statistically significant.

Results

A total of 149 patients participated in the study. Among these patients, 131 (87.9%) were males and 18 (12.1%) were females. The average age of participants was 36.9 years, ranging from 16 to 65 years. ART regimens used by the patients were as follow: 26 (17.4%) individuals used abacavir, lamivudine, dolutegravir (ABC/3TC/DTG), 32 (21.5%) used tenofovir alafenamide, emtricitabine, bictegravir (TAF/FTC/BIC), 34 (22%) used tenofovir alafenamide, emtricitabine, cobicistat, elvitegravir (TAF/FTC/c/EVG), 29 (19.5%) used tenofovir disoproxil fumarate, emtricitabine, cobicistat, elvitegravir (TDF/FTC/c/EVG), and 28 (18.8%) patients used tenofovir disoproxil fumarate, emtricitabine, dolutegravir (TDF/FTC/DTG).

Following 48 weeks of treatment, the study found that virologic success was observed in 147 out of 149 patients (98.7%). The alterations in inflammation parameters and hemogram values of the included patients during their treatment initiation and after 12 months are shown in Table 1.

Upon comparing the changes in inflammatory parameters and hemogram values for the start of patients' treatment and at 48 week of treatment, statistically significant changes were observed in CD4+ T cell count ($p < 0.001$), Hb ($p < 0.001$), WBC ($p < 0.01$), Neut ($p < 0.01$), Lymph ($p < 0.01$), PLT ($p < 0.01$), MPV ($p = 0.039$), PCT ($p < 0.01$) and PLR ($p = 0.003$). These values were found statistically significant (Table 1). The change in ESR did not reach statistical significance ($p = 0.06$).

Discussion

In the current study involving patients diagnosed at our hospital over the past 10 years and receiving various ART regimens, we observed a significant decrease in PLR value, alongside with an increase in PCT and MPV values.

Hematologic abnormalities frequently occur in individuals infected with HIV. In developing countries where resources may be limited, routine tests assessing inflammation values are significant due to their affordability and ease of accessibility.

Thrombocytopenia, a condition characterized by low platelet counts, has been observed in older individuals with HIV. This phenomenon is attributed to the activation of platelets, subsequently leading to vascular and endothelial inflammation as well as migration of leukocytes out of blood vessels. This process is mediated by the release of cytokines and chemokines from platelet storage [15].

In HIV-infected patients, thrombocytopenia can occur through two main mechanisms, i.e., increased clearance of activated platelets from bloodstream or decreased platelet production from infected megakaryocytes. A study conducted in Africa reported that the overall prevalence of thrombocytopenia among individuals living with HIV was 6.3% [16]. In Ambler *et al.* [17] study involving a larger sample size of 5,290 individuals living with HIV, the incidence of thrombocytopenia was found even higher (26%). Duguma *et al.* [18] investigated hematologic parameters in 308 cases, and observed that platelet counts significantly increased from an average of 242 ± 109.7 to 273 ± 117 after ART initiation ($p = 0.001$). Tilahun *et al.* [19] in their study reported that the mean platelet count of participants before starting ART was $246 \pm 101 \times 10^3/\mu\text{L}$. After 6 months of ART, the mean platelet count increased to $275 \pm 96 \times 10^3/\mu\text{L}$, and this difference was found statistically significant. In the present study, in line with the literature, the mean platelet count increased significantly from $224.72 \pm 71.94 \times 10^3/\mu\text{L}$ to $253.03 \pm 64.01 \times 10^3/\mu\text{L}$ after 48 weeks of ART.

Data from previous studies indicate that MPV may offer crucial insights into the prognosis of various inflammatory diseases [20]. Research has shown that low MPV values are indicative of ongoing high inflammation [21]. However, studies examining MPV values specifically in HIV-positive individuals are lacking. In a single study focused on women infected with HIV, it was observed that MPV rate was lower in this population [22]. Our study corroborates with these findings by demonstrating a significant increase in MPV scores following one year of ART. Overall, these results suggest that HIV-related inflammation is associated with a decrease in MPV level.

PCT value has been considered an indicator of inflammation in numerous diseases, with findings consistently demonstrating a positive association between PCT and acute inflammation. Moreover, in cases with chronic inflammatory diseases and cancer, the PCT index has been found to mirror platelet reactivity, which is influenced by MPV levels. Additionally, notable alterations in PCT levels were observed [23-26], with research indicating that the normal range for PCT values was within 0.22-0.24% [25, 27].

Several studies have reported a relationship between MPV and pro-inflammatory cytokine release from platelets in chronic conditions, further revealing a significant negative correlation between PCT levels and extent of inflammation [28, 29]. Moreover, in relation to specific viral infections, elevated PCT levels were observed in patients diagnosed with viral hepatitis A compared with non-hepatitis control group [30]. Conversely, a decrease in PCT levels occurring with the progression of fibrosis in chronic viral

Table 1. Changes in inflammatory parameters and hemogram values at starting and 48th week of treatment

Parameters	<i>n</i>	Mean ± SD	Min.-Max.	<i>P</i> -value
CD4				
Start	149	440.47 ± 263.47	8.42-1,264.00	< 0.001
48 th week	149	738.15 ± 345.21	0.39-1,948.00	
CD8				
Start	149	1,202.99 ± 702.16	40.00-5,857.00	0.170
48 th week	149	1,106.69 ± 663.59	296.00-7,092.47	
CD4/CD8				
Start	149	0.45 ± 0.35	0.005-1.653	< 0.001
48 th week	149	0.81 ± 0.46	0.14-2.53	
HIV RNA				
Start	149	1,254,652.141 ± 8,305,134.337	63.00-100,000,000.0	0.067
48 th week	149	17.15 ± 48.39	0.00-436.00	
CRP				
Start	34	2.85 ± 3.94	0.35-19.79	0.365
48 th week	34	2.25 ± 2.16	0.28-10.42	
ESR				
Start	34	15.38 ± 22.57	2.00-99.00	0.006
48 th week	34	6.88 ± 11.32	0.00-68.00	
LDL/HDL				
Start	73	2.68 ± 1.03	0.43-5.76	0.792
48 th week	73	2.71 ± 0.87	0.45-4.38	
HB				
Start	149	14.60 ± 1.64	9.10-17.70	< 0.001
48 th week	149	15.07 ± 1.42	10.10-17.60	
WBC				
Start	149	6.12 ± 2.03	2.77-13.34	< 0.001
48 th week	149	7.12 ± 1.91	3.56-13.37	
NEUT				
Start	149	3.39 ± 1.54	0.48-10.50	< 0.001
48 th week	149	4.04 ± 1.52	1.53-10.44	
LYMPH				
Start	149	2.04 ± 0.96	0.38-7.60	< 0.001
48 th week	149	2.29 ± 0.65	1.10-4.74	
MONO				
Start	149	0.51 ± 0.25	0.16-2.76	0.078
48 th week	149	0.62 ± 0.71	0.23-9.00	
PLT				
Start	149	224.72 ± 71.94	0.40-524.00	< 0.001
48 th week	149	253.03 ± 64.01	5.01-466.00	
MPV				
Start	149	10.26 ± 1.07	7.60-12.70	0.039
48 th week	149	10.39 ± 0.91	8.40-12.70	

Table 1. Cont.

Parameters	<i>n</i>	Mean ± SD	Min.-Max.	<i>P</i> -value
PCT				
Start	149	0.23 ± 0.07	0.00-0.57	< 0.001
48 th week	149	0.26 ± 0.06	0.005-0.550	
SII				
Start	149	443.26 ± 322.32	0.39-2,262.27	0.205
48 th week	149	477.96 ± 268.98	9.46-1,847.07	
SIRI				
Start	149	1.01 ± 0.88	0.11-6.68	0.185
48 th week	149	1.20 ± 1.61	0.21-18.94	
NLR				
Start	149	1.95 ± 1.30	0.24-9.54	0.437
48 th week	149	1.87 ± 0.88	0.70-6.32	
PLR				
Start	149	127.87 ± 59.96	0.15-349.18	0.003
48 th week	149	116.09 ± 36.08	3.29-233.63	
LMR				
Start	149	4.28 ± 2.04	0.67-16.17	0.860
48 th week	149	4.31 ± 1.38	0.19-7.97	

CRP – C-reactive protein, ESR – erythrocyte sedimentation rate, HB – hemoglobin, LDL/HDL – low density lipoprotein/high density lipoprotein, LYMPH – lymphocyte count, MONO – monocyte count, MPV – mean platelet volume, NEUT – neutrophil count, NLR – neutrophil-to-lymphocyte ratio, PCT – plateletcrit, PLR – platelet-to-lymphocyte ratio, PLT – platelet count, SII – systemic immune-inflammatory index, SIRI – systemic immune response index, WBC – total leukocyte count

hepatitis B patients was reported, reaching 0.12 in patients with cirrhosis [23]. In Şahin *et al.* [31] study monitoring PCT levels in patients living with HIV, PCT level was 0.1895 (ranging from 0.052 to 0.381) in the 24th month of treatment, while in the group not receiving treatment, PCT was 0.1765 (ranging from 0.03 to 0.385).

In the current study, we observed a significant increase in PCT level, from 0.23 to 0.26, after one year of ART. This suggests that PCT can serve as a readily accessible marker for monitoring inflammation during ART treatment. It is worth noting that the evaluation results of PCT in HIV and non-HIV chronic inflammatory diseases vary in the existing literature. Therefore, further research is needed to gain a more comprehensive understanding of this biomarker and its applicability in different contexts.

Conclusions

While numerous pathogenic mechanisms underlying HIV-associated immune activation and inflammation have been extensively studied, there is an unmet requirement to develop targeted therapeutic strategies for addressing these issues. It is important to recognize that conditions unrelated to AIDS yet associated with inflammation, may potentially accelerate disease progression. In light of this context, we suggest the utilization of readily available and easily calculable markers of chronic inflammation, such as platelet count, MPV, and PCT, which are currently recommended for

monitoring inflammation in chronic conditions. According to the results of the study, the afore-mentioned markers have potential applicability in monitoring inflammatory parameters in HIV-positive individuals.

Disclosures

1. Institutional review board statement: The study received approval from the Clinical Research Ethics Committee of Mersin University Faculty of Medicine, issued on October 26, 2022, under the reference number of 2022/702.
2. Assistance with the article: None.
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4. Conflicts of interest: None.

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