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RESEARCH ARTICLE

THE IMPACT OF PANCYTOPENIA ON THE MANIFESTATIONS OF CHRONIC HEART FAILURE IN PATIENTS INFECTED WITH THE HUMAN IMMUNODEFICIENCY VIRUS: A CROSS-SECTIONAL STUDY

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Abstract

Background: Human Immunodeficiency Virus (HIV) infection affects not only immune function but also bone marrow hematopoiesis, resulting in hematological abnormalities including anemia, thrombocytopenia, neutropenia, and pancytopenia. Chronic heart failure (CHF) also occurs more frequently, at a younger age, and with more rapid progression among people living with HIV than in individuals without HIV infection. However, the relationship between pancytopenia and the clinical severity of CHF in HIV-infected patients remains insufficiently characterized.
Objective: To assess the association between pancytopenia and the clinical, instrumental, and laboratory manifestations of chronic heart failure among patients infected with HIV.
Methods: This cross-sectional study included 240 patients with HIV infection, of whom 160 had CHF. Among patients with CHF, 63 had pancytopenia. The influence of pancytopenia on CHF manifestations was evaluated by comparing clinical, anamnestic, instrumental, and laboratory parameters between HIV-infected patients with CHF with and without pancytopenia.
Results: HIV-infected patients with CHF and concomitant pancytopenia demonstrated more severe heart failure, reflected by a higher functional class and more pronounced clinical manifestations. Pancytopenia was also associated with greater diastolic myocardial dysfunction and higher indices of aortic stiffness. Patients with pancytopenia had higher levels of N-terminal pro-B-type natriuretic peptide (NT-proBNP) and tissue inhibitor of metalloproteinase-1 (TIMP-1), together with lower CD4+ T-lymphocyte counts, compared with patients without pancytopenia.
Conclusion: Pancytopenia is associated with more severe clinical and structural-functional manifestations of chronic heart failure in people living with HIV. The coexistence of pancytopenia and CHF may reflect interconnected pathogenic processes involving HIV-associated immune dysfunction, impaired hematopoiesis, myocardial remodeling, and vascular stiffness.
Keywords: bone marrow, chronic heart failure, HIV, pancytopenia.

INTRODUCTION

In 2022, approximately 38 million individuals infected with HIV were officially documented globally. HIV infection leads to complex damage to organs and systems, progressing to the stage of acquired immune-deficiency syndrome, where signs of severe systemic multi-organ failure become evident. The impact of the viral process on cardiomyocytes and bone marrow cells starts nearly at the same time, showing different forms of cytopenia (anemia, thrombocytopenia, leukopenia, pancytopenia) and leading to diastolic and systolic chronic heart failure[1]. Grasping the pathophysiological mechanisms of pancy-topenia and CHF development in HIV infection indicates the shared nature of these processes, where the main damaging elements include the direct effects of the virus on cardiomyocytes and bone marrow cells, the role of the inflammatory response, and the influence of factors associated with advancing immunodeficiency. This study aimed to

assess how pancytopenia affects the progression of CHF in HIV-infected patients receiving treatment at a major hospital in a populous city. The findings enable us to conclude the connection between CHF and bone marrow dysfunction in patients with HIV. The purpose of the research was to examine the unique effects of pancytopenia on the symptoms of chronic heart failure in individuals infected with HIV.

MATERIALS AND METHODS

The research was carried out in a large hospital located in a Russian city with a population of one million and utilized an exploratory, one-phase, cross-sectional, screening approach. The research involved 240 patients infected with HIV who were admitted to the hospital over a span of four years. Solely the authors' private finances supported the research. The study's principles adhered to the Declaration of Helsinki, and all enrolled patients willingly participated by signing informed consent forms. The Ethics Committee of E.A. Wagner Perm State Medical University approved the study in 2021.The requirement for participation in the study was the existence of an HIV immunoblot and the patient's consent. Reasons for exclusion from the study included oncopathology, exacerbations of serious accompanying illnesses, abnormal patient behavior, valvular heart defects, pregnancy, and being under 18 years of age.The diagnosis of chronic heart failure was made based on the criteria set forth in the clinical recommendations on CHF issued by the Russian Society of Cardiology in 2020[2]. The diagnosis of pancytopenia was based on the simultaneous occurrence of anemia, thrombocytopenia, and leuko-penia. The indicator of anemia was a reduction in hemoglobin levels below 120 g/L for women and below 130 g/L for men. Thrombocytopenia was confirmed when the platelet count fell below 150 x 10^9 cells/l. Leukopenia was identified when the leukocyte count fell below 4x10^9 cells/L. If all three types of cytopenia were observed, the patient was assigned to the pancytopenia category[3]. The overall count of patients - participants in the study was 240 individuals. Out of them, the number of individuals with CCN totaled 160 individuals (66.6%). Pancytopenia was identified in 63 patients with CCN, whereas 97 patients did not exhibit pancytopenia.

Echocardiography was performed on a VIVID T8 (GE) device. As part of the examination, Tei index was additionally determined in all patients to clarify diastolic dysfunction as the ratio of the sum of isovolumetric contraction time and isovolumetric relaxation time to the ejection time.

N-terminal fragment of brain natriuretic peptide (NT-proBNP) in blood plasma was determined in all examined patients using Vector Best (Russia) reagent kits for enzyme immunoassay, CRP level in blood serum was determined using Vector Best (Russia) reagent kits by enzyme immunoassay method. The level of tissue metalloproteinase-1 (TIMP-1) in blood serum was determined by immunoenzyme assay kits of Sun-Long Biotech (China).

Statistical analysis

The results of the study were statistically processed using the programs "Statistica 13" (Russia) and IBM SPSS Statistics 26 (USA). The distribution of signs was specified using the Kolmogorov-Smirnov and Shapiro-Wilk methods. There were no normally distributed signs. Signs with distribution different from normal are presented as median, first and third quartiles [Me (Q1; Q3)]. Categorical features are presented as absolute value and percentage [n (%)]. The critical value of statistical significance was represented as $p<0.050$.

RESULTS

The influence of pancytopenia on the course of CHF was analyzed. Table 1 presents changes in clinical and anamnestic parameters in patients with CHF infected with HIV, depending on the presence of pancytopenia**.** The obtained data indicate that in patients with CHF, infected with HIV, on the background of pancytopenia higher functional class of CHF, higher number of points on the scale of assessment of clinical condition of CHF patient, lower number of meters on the test of six-minute walk (TSH).

Table 1: Peculiarities of the course of CHF on the background of pancytopenia in HIV-infected patients - clinical and anamnestic parameters (n=160).

Index	Pancytopenia, n=63	Absent pancytopenia, n=97	*p*
Age in years	37 (34; 41)	37 (32; 40)	0.343
Male gender, n (%)	41 (65)	53 (54.6)	0.190
FC CHF	2 (2; 3)	2 (2; 2)	0.001
Smoking, n (%)	47 (74.6)	65 (67)	0.305
Alcohol dependence n (%)	31 (49.2)	62 (63.9)	0.065
CHD, n (%)	18 (28.6)	20 (20.6)	0.248
PICS, n (%)	1 (1.58)	3 (3.1)	0.551
AF, n (%)	1 (1.58)	4 (4.1)	0.367
VRD, n (%)	29 (46.0)	34 (35.0)	0.164
ART, n (%)	12 (19.0)	15 (15.5)	0.554
6-min test, m	375 (200; 400)	400 (350; 475)	0.006
Clinical assessment scale, scores	5.5 (5.0; 8.0)	5.5 (4.0; 7.0)	< 0.001
SBP, mm Hg	130.0 (115.0; 146.0)	115.0 (107.0; 132.0)	<0.001
DBP, mm Hg	77.0 (65.0; 84.0)	70.0 (60.0; 79.0)	0.018

FC CHF - functional class of Chronic Heart failure; CHD - ischemic heart disease; PICS- postinfarction cardiosclerosis; AF- atrial fibrillation; VRD - ventricular rhythm disturbances; ART, antiretroviral therapy, SBP - systolic blood pressure; DBP - diastolic blood pressure.

Table 2: Features of CHF manifestations on the background of pancytopenia in HIV-infected patients - some indices of echocardiography and non-invasive angiography (n=160).

Index	Pancytopenia, n=63	Absent pancytopenia, n=97	*p*
LVEF %	57.0 (48.0; 65.0)	55.0 (46.0 65.0)	0.982
HLV, n (%)	33 (52.4)	51 (52.6)	0.980
Increased left atrial volume, n (%)	29 (46.0)	42 (43.3)	0.730
Diastolic dysfunction, n (%)	50 (79.3)	71 (73.2)	0.374
E/e´	8.0 (5.9; 20.0)	6.3 (5.1; 14.9)	0.042
Tei Index	0.6 (0.4; 0.7)	0.5 (0.3 ; 0.55)	<0.001
SBPao, mm Hg	110.0 (103.0; 142.0)	104.0 (99.0; 120.0)	0.010
AIXao mean	16.5 (2.1; 37.6)	11.3 (2.9; 26.7)	0.655
AIXbrmean	-41.8 (-70.3; 0.0)	-52.0 (-68.7; -21.5)	0.655
Ppao, mm Hg	44.0 (32.0; 53.0)	39.0 (30.0; 45.0)	0.016
Pwao, m/s	7.6 (7.0; 10.0)	7.3 (6.3; 8.3)	0.002

LVEF - left ventricular ejection fraction; LVH - left ventricular hypertrophy; E/e´ - ratio of peak peak early filling velocity of the left ventricle to early diastolic velocity of the annulus fibrosus; SBPao - aortic systolic blood pressure; AIXao mean - aortic augmentation index; AIXbrmean - brachial artery augmentation index; Ppao - aortic pulse pressure; Pwao, aortic pulse wave velocity.

There were no significant differences by sex, age, smoking, and alcohol intake. No differences were obtained on the history of cardiovascular diseases. At the same time, the values of office systolic and diastolic pressures were higher on the background of pancytopenia. The main echocardiographic differences and indices of noninvasive angiography in patients with CHF infected with HIV, depending on the presence of pancytopenia, are presented in Table 2. Thus, ultrasound examination revealed that against the background of pancytopenia there was a higher index of the ratio of peaks of maximum early filling velocity of the left ventricle to early diastolic velocity of the fibrous ring (E/e´), which is diagnostically significant for manifestations of diastolic dysfunction. No other significant changes were found in the study of echocardiography indices. Among the indicators of noninvasive arteriography higher systolic pressure in aorta, pulse pressure in aorta (Prao), pulse wave propagation velocity in aorta (Pwao) reflecting elastic vessel stiffness were revealed. Data on changes in some laboratory parameters are presented in Table 3. According to the results obtained, the content of N-terminal fragment of brain natriuretic peptide in plasma and TIMP-1 in serum were higher in the group of patients with pancytopenia. Inflammatory tests were more intensely expressed in the group of patients with pancytopenia there was an increase in the concentration of C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR). Against this background, lower values of activated partial thromboplastin time (APTV) were observed in pancytopenia. There were no differences in the number of patients taking APT between the groups. Hemoglobin, platelet, and leukocyte content differed significantly ($p<0.001$), as did the number of segmented neutrophils ($p<0.042$) in the group of patients with CCN and pancytopenia. At the same time, no significant differences were obtained in the level of neutrophils, eosinophils and lymphocytes, except for CD-4 T-lymphocytes, the content of which was significantly lower ($p=0.010$). Correlation analysis was performed between the content of hematologic indices that showed significant differences when comparing between groups of patients with CHF and HIV infection, depending on the presence of pancytopenia, and indicators responsible for the severity of CHF. A marked inverse relationship between hemoglobin level and plasma NT-proBNP value (r=-0.521, $p<0.001$), hemoglobin level and Tei index value (r=-0.408, $p<0.050$), weak inverse relationship between hemo-globin level and E/e' ratio (r=-0.235, $p<0.050$) were revealed according to Chaddock scale.

Table 3: Laboratory parameters of patients with CHF and HIV infection, depending on the presence of pancytopenia (n=160).

Index	Pancytopenia, n=63	Absent pancytopenia, n=97	*p*
NT-proBNP, pg/ml	929.7 (291.2; 2173.0)	450.0 (223.3; 937.9)	0.018
TIMP-1, pg/ml	31.1 (17.6; 45.1)	19.7 (4.0; 43.4)	0.049
ESR, mm/h	55.0 (29.0; 65.5)	31.0 (18.0; 50.0)	0.003
CRP, mg/l	22.5 (8.0; 53.0)	13.5 (4.6; 33.0)	0.043
APTT, s	31.0 (30.0; 32.0)	36.0 (34.0; 39.0)	0.005
PTT, s	17.0 (15.0; 18.0)	16.0 (14.0; 17.0)	0.530
CD-4-T-lymphocytes, cl/ml	180.0 (26.0; 270.0)	250.0 (90.0; 450.0)	0.010
Hemoglobin, g/l	85.0 (64.0; 100.0)	102.0 (80.0; 119.0)	<0.001
Leukocytes, cells $\cdot 10^9$/l	2.7 (2.0; 3.0)	8.8 (5.8; 9.4)	<0.001
Platelets, cells $\cdot 10^9$/l	100.0 (60.0; 135.0)	214.0 (133.0; 295.0)	<0.001
Lymphocytes,%	18.0 (16.0; 37.0)	15.0 (9.00; 22.0)	0.122
Band neutrophils, %	5.0 (4.0; 6.0)	4.0 (2.0; 6.0)	0.341
Segmented neutrophils,%	40.0 (18.0; 43.0)	69.0 (59.0; 175.0)	0.042
Eosinophils,%	2.5 (1.0; 3.3)	1.0 (1.0; 2.0)	0.530

NT-proBNP – N-terminal fragment of brain narriuretic propeptide; TIMP-1 – tissue inhibitor of metalloproteinases-1; ESR – erythrocyte sedimentation rate; CRP – C-reactive protein; APTT – activated partial thromboplastin time; PTT – prothrombin time.

A moderate association was obtained between platelet count and NT-proBNP concentration (r=-0.356, $p<0.050$), as well as a weak association with Tei index value (r=-0.148, $p<0.050$), and a weak negative association with BMI (r=-0.193, $p<0.050$). Peripheral blood leukocyte count has an inverse correlation of moderate strength with plasma NT-proBNP level (r=-0.383, $p<0.050$), weak inverse correlation with diastolic dysfunction indices E/e' (r=-0.230, $p<0.050$) and Tei index (r=-0.268, $p<0.050$), as well as with BMI (r=-0.169, $p<0.050$) and left ventricular ejection fraction (r=-0.171, $p<0.050$). The level of segmented neutrophils has an inverse weak relationship with left ventricular ejection fraction (r=-0.232, $p<0.050$), a direct relationship of weak strength with plasma NT-proBNP value (r=0.278, $p<0.050$), with Tei index (r=0.278, $p<0.050$).

DISCUSSION

Human immunodeficiency virus belongs to the family of retroviruses and causes systemic damage to the body by directly acting on cells expressing CD4-proteins of their membranes in response to viral aggression. Progressive immunodeficiency in HIV infection reflects the increasing deficiency of CD4-T-lymphocytes[4]. HIV infection is associated with hematologic changes such as anemia, thrombocytopenia, neutropenia, and pancytopenia, indicating that the HIV target organ, the bone marrow, is affected[5,6]. The pathogenesis of hematologic disorders in HIV infection is complex and multifactorial, including drug-induced disorders of hematopoiesis, bone marrow suppression due to infiltration of infectious agents or malignant cells, HIV-induced disorders of hematopoiesis, and a number of other factors[4]. It is known that HIV can directly infect subpopulations of progenitor cells, exerting a direct damaging effect as well as persisting in them for a long time[7]. HIV-infected bone marrow stem cells may serve as long-term reservoirs of HIV[8]. One of the possible mechanisms of pancytopenia development in HIV is direct damage to the bone marrow as a result of cytokine storm[9].

Anemia is the most common hematologic complication of HIV infection and has three key factors in its development - decreased erythrocyte production in the bone marrow, increased erythrocyte destruction and poor erythrocyte quality due to intense metabolic disorders and progression of immunosuppression, which, among other things, is expressed in a decreased number of CD-4 T-lymphocytes[1]. Initiation and continuation of antiretroviral therapy (ART) is associated with decreased viral load and inflammation on cardiomyocytes and bone marrow cells[4]. The presence of CHF increases the chances of anemia in patients with HIV infection 2.75 times, and the development of anemia is associated with a worsening course of CHF, the development of chronic kidney disease stage IIIA and higher, an increase in left atrial volume, the development of left ventricular diastolic dysfunction, and the threshold level of NT-pro BNP≥170 pg/ml is correlated with the development of anemia on the background of CHF in HIV-infected patients[10].

Thrombocytopenia is the second most common hematologic complication of HIV infection; the main cause of thrombocytopenia is insufficient platelet production and autoimmune reactions[11]. Against the background of thrombocytopenia, HIV-infected people are almost 2 times more likely to have CHF, accompanied by an increase in TIMP-1, and severe thrombocytopenia with platelet count ≤30·109/L cells/l increases the chances of having CHF with low ejection fraction by 10.8 times[12].

Neutropenia against the background of HIV infection is often found together with anemia and thrombocytopenia and very rarely separately[13]. Leuko-penia in HIV infection is associated with a decrease in the level of CD4 T-lymphocytes less than 200 cells/ml and with a viral load of more than 100000 copies/ml, and can also develop against the background of the accession of severe opportunistic infections, malignant tumors, some ART drugs, antibiotics, chemotherapy[14].

Tissue inhibitor of metalloproteinases-1 is a marker of both local and systemic fibrosis. Its level is known to be increased in the tissues of kelloid scars, and in serum - in aortic stenosis and left atrial dilatation on the background of atrial fibrillation, which is explained by its high sensitivity to increased fibrosis of cardiomyocytes and increased stiffness of valve fibrous rings[15]. In this study, TIMP-1 demonstrated its sensitivity to pancytopenia in HIV-infected patients with CHF, indirectly indicating possible activation of the fibrosis process in the bone marrow against the background of progression of CHF and pancytopenia.

Damage to the heart muscle in HIV infection leads to myocarditis, chamber dilatation with the formation of HIV-associated cardiomyopathy, CHF, early atherosclerosis of arteries, including coronary arteries, and pericarditis[16]. The pathogenesis of cardiovascular damage in HIV is similar to the pathogenesis of bone marrow damage.

Initially, there is a direct effect of viruses on cardiomyocytes and endothelium, which further leads to the development of apoptosis and inflammation in infected cells[17,18]. The second important pathophysiological component in the development of CHF, as well as in bone marrow injury, is an active inflammatory process accompanied by an increase in inflammatory markers - interoeukin-6, soluble tumor necrosis factor (α-1 and α-2), CRP, fibrinogen[19]. The third common pathogenetic mechanism for the development of CHF and bone marrow failure is the use of ART[5]. The list of common pathogenetic mechanisms is supplemented by the influence of opportunistic infection[2].

The commonality of pathophysiological mechanisms allows us to conclude that the pathological processes of CHF development and bone marrow damage in HIV-infected patients are related. Previously, we described the relationship between the influence of CHF on the development of anemia and thrombocytopenia, as well as the aggravation of the course of CHF in patients with anemia and thrombocytopenia against the background of HIV infection[5,7]. In the present work we have shown for the first time the effect of pancytopenia on the manifestations of CHF, namely, an increase in

the functional class of CHF, a decrease in the number of steps in TSH and scores in SHOCS, an increase in systolic and diastolic blood pressure, increase in the index of diastolic dysfunction severity E/e', increase in aortic stiffness indices, increase in the concentration of NT-proBNP, a marker of CHD severity, increase in the index of vascular wall fibrosis TIMP-1 and increase in inflammatory indices CRP and CRP.

CONCLUSIONS

The manifestations of CHF and pancytopenia as a marker of insufficiency of medullary hematopoiesis in patients with HIV infection are closely related by common pathogenetic mechanisms of virus action. It is shown that the aggravation of CHF in HIV-infected patients is observed against the background of pancytopenia. Clinical changes are manifested in the increase of SHOCS scores, decrease of six-minute walk test parameters and detection of more severe functional classes of CHF. Instrumental data confirm the increase in diastolic dysfunction indices, such as the ratio of peak early left ventricular filling velocity to early diastolic annulus fibrosis velocity (E/e'), as well as Tei index. The group of patients with pancytopenia also has higher values of office blood pressure, aortic pulse wave velocity, and pulse pressure.

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AUTHOR'S CONTRIBUTIONS

Obeagu EI: methodology, literature review, writing original draft. **Goryacheva OG:** conceptualization, study design, supervision, methodology. **Zhelobov VG:** Data collection, clinical assessment, data analysis, manuscript review. **Zubarev MA:** data collection, interpretation of findings. **Lokesh R:** literature review, data analysis, manuscript drafting. **Goryachev AS:** supervision, clinical interpretation, critical review. The final version of the manuscript was checked, reviewed and approved by all authors.

DATA AVAILABILITY

The related author can provide the empirical data supporting the study's conclusions upon request.

CONFLICTS OF INTEREST

The authors declare no conflict of interest

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