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Heart failure in human immunodeficiency virus-infected patients

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Aim. To determine the features of heart failure (HF) development in patients with human immunodeficiency virus (HIV) infection.

Material and methods. In a general hospital, 160 patients were examined during the year. All of them were divided into 2 groups: group 1 (n=100) — HIV-infected patients with specific clinical picture of HF; group 2 (n=60) — patients without HIV infection and with HF verified by echocardiography and concentration of N-terminal prohormone of brain natriuretic peptide (NT-proBNP).

Results. In comparison with group 2, HIV-infected patients had the following statistically significant differences: lower left ventricular ejection fraction (LVEF), lower prevalence and severity of left ventricle diastolic dysfunction, higher LV mass index (LVMI), and lower NT-proBNP. HIV-infected patients had statistically significant moderate inverse relationship of LVEF (r=-0,43; p=0,015), E/e' (r=-0,32; p=0,045), LVMI (r=-0,46; p=0,002) and strong relationship of NT-proBNP (r=-0,54; p<0,001) with CD4 T-lymphocyte count in 1 mm^3 in the presence of HF symptoms and signs and an increase in NT-proBNP over 125 pg/ml. In group 1, there was a significantly higher prevalence of smoking, chronic alcoholism, drug use, chronic hepatitis C and cirrhosis (especially manifested by hepatomegaly and splenomegaly in combination with ascites and hepatic cytolysis), chronic pancreatitis, pneumonia and inflammatory diseases accompanied by higher erythrocyte sedimentation rate and C-reactive protein concentration, and lower hemoglobin level. HIV-infected patients were statistically less likely to use all groups of drugs for HF treatment, with the exception of spironolactone, and more likely to use drugs for multimorbidity treatment.

Conclusion. The HF prevalence in hospitalized HIV-infected patients, estimated on the basis of symptoms and NT-proBNP increase >125 pg/ml, was 54%; on the basis of LVEF decrease <50% — 32%. The clinical picture of HIV-infected patients is characterized by various symptoms, including those typical for HF with normal NT-proBNP level, due to the high prevalence of comorbidities and concurrent medication.

Key words: heart failure, human immunodeficiency virus.

Relationships and Activities: not.

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Cardiac injury in HIV-infected patients is a common pathology in clinical practice. A number of researchers have identified a certain form of cardiac pathology in HIV-infected patients — HIV-associated cardiomyopathy. It is associated with the direct damaging effect of HIV infection on the myocardium, accompanied by apoptosis of cardiomyocytes and fibroblasts [1]. Cardiac injury in HIV infection has a multifactorial pathogenesis, characterized not only by a direct damaging effect, but also by the influence of a secondary infection, leading to the development of myo-, peri- and endocarditis, and by the use of antiretroviral therapy with cardiotoxicity [2, 3].

The patterns of the heart failure (HF) formation, its phenotypes, such as cardiac dysfunction in HIV-infected patients have been little studied and the effectiveness of conventional HF therapy has not been investigated. The choice of certain type of antiretroviral treatment in this category of patients has also not been determined. So, according to some reports, in 8% of HIV-infected people there is myocardial injury with severe dilatation of the cavities and a significant contractility decrease [2]. HF with systolic dysfunction of the left ventricle (LV) in HIV-infected patients occurs 10 years earlier than in patients without HIV infection [4]. With antiretroviral therapy, early diastolic dysfunction (DD) is formed due to the stimulation of myocardial fibrosis [3].

Thus, the presented study attempted to identify the features of HF formation in HIV-infected patients in order to further study the problem and select HF and antiretroviral therapy.

The aim of this study was to determine the features of HF formation in HIV-infected patients.

Material and methods

This study was performed in accordance with the Helsinki declaration and Good Clinical Practice standards. The local medical ethics committee approved this study. All participants gave written informed consent.

Table 1

Comparison of parameters reflecting the clinical course and severity of heart failure between groups (n=160)

Parameter	Group 1 (HF and HIV infection, n=100)	Group 2 (HF, n=60)	p
CSS, points	5,89 [3,22; 5,12]	6,33 [3,98; 6,38]	0,128
6MWT, m	437,2±126,9	372,4±41,2	<0,001
Mean HF FC	2,3 [1,4; 3,2]	2,5 [1,8; 3,5]	0,128
RHR, bpm	86,76±16,27	78,75±11,53	0,001
RHR >70 bpm, abs./%	76/76,0	36/0,60	0,050
LVEF, %	56,0±11,1	65,3±15,7	<0,001
LVEF >50%, abs./%	68/68,0	45/75,0	0,447
LVEF 40-49%, abs./%	26/26,0	10/16,7	0,242
LVEF <40%, abs./%	6/6,0	5/8,3	0,809
E/A	1,26 [1,0; 1,62]	1,11 [0,86; 1,68]	0,089
E/e´ mean	11,8 [4,5; 17,3]	15,0 [10,5; 19,3]	<0,001
E/e´ mean >14, abs./%	24/24,0	41/68,3	<0,001
LV IVRT, ms	94,0±35,7	92,7±18,9	0,682
LVDD, abs./%	40/40,0	41/68,3	0,002
Left atrial volume/BSA, ml/m^2	29,21 [24,11; 38,06]	25,12 [15,41; 34,03]	0,106
Left atrial volume/BSA >34 ml/m^2	36/36,0	11/20,0	0,051
LVMI, g/m^2	132,2 [96,5; 151,0]	109,2 [78,6; 118,5]	<0,001
LVMI >110 g/m^2 for men, >95 g/m^2 for women	88/88,0	28/46,7	<0,001
NT-proBNP, pg/ml	159,1 [49,9; 539,7]	234,6 [187,1; 558,6]	<0,001
NT-proBNP >125 pg/ml, abs./%	54/54,0	60/100,0	<0,001

Abbreviations: A — peak late filling velocity, BSA — body surface area, CSS — clinical state scale, E — peak early filling velocity, e' — velocity of early diastolic mitral annulus motion, FC — functional class, HIV — human immunodeficiency virus, HF — heart failure, IVRT — isovolumic relaxation time, LVEF — left ventricular ejection fraction, LVDD — left ventricular diastolic dysfunction, LVMI — left ventricular mass index, NT-proBNP — N-terminal pro-brain natriuretic peptide, RHR — resting heart rate, 6MWT — six-minute walk test.

Table 2

Anamnestic characteristics of groups (n=160)

Parameter	Group 1 (HF and HIV infection, n=100)	Group 2 (HF, n=60)	p
Age, years	36,0±6,3	54,0±8,6	<0,001
Gender, M/F, abs./%	63/37 (63/37)	16/44 (27/73)	<0,001/<0,001
Smoking, abs./%	67 /67,0	16/26,7	<0,001
Chronic alcoholism, abs./%	46/46,0	0/0	<0,001
History of narcotic use, abs./%	87/87,0	0/0	<0,001
HTN, abs./%	28/28,0	52/86,7	<0,001
CAD, abs.	2/2,0	21/35,0	<0,001
IM history, abs./%	1/1,0	9/15,0	<0,001
Type 2 diabetes, abs./%	8/8,0	10/16,7	0,156
AF, abs./%	2/2,0	7/11,7	0,027
Ventricular rhythm disturbances, abs./%	30/30,0	15/25,0	0,618
History of TIA, stroke, abs./%	4/4,0	5/8,3	0,426
History of CABG, PCI, abs./%	0/0	5/8,3	0,014
Chronic hepatitis C, abs./%	83/83,0	1/1,7	<0,001
Cirrhosis, abs./%	46/46,0	1/1,7	<0,001
Chronic pancreatitis, abs./%	31/31,0	4/6,7	0,001
History of infectious endocarditis, abs./%	4/4,0	0/0	0,297
Pneumonia, abs./%	18/18,0	3/5,0	0,035
Inflammatory diseases, abs./%	11/11,0	0/0	0,020
Thromboembolism (history, acute phase), abs./%	8/8,0	2/3,3	0,400
Osteoarthritis, abs./%	0/0	10/16,7	<0,001

Abbreviations: AF — atrial fibrillation, CABG — coronary artery bypass grafting, CAD — coronary artery disease, HIV — human immunodeficiency virus, HTN — hypertension, MI — myocardial infarction, PCI — percutaneous coronary intervention, TIA — transient ischemic attack.

In a general hospital, 160 patients were examined during the year. All of them were divided into 2 groups: group 1 (n=100) — HIV-infected patients with specific clinical picture of HF; group 2 (n=60) — patients without HIV infection and with HF verified by echocardiography and concentration of N-terminal prohormone of brain natriuretic peptide (NT-proBNP). There were following inclusion criteria: typical symptoms and specific signs of stable HF; verified HIV infection (group 1); typical symptoms and specific signs of stable heart failure, confirmed by echocardiography and NT-proBNP increase >125 pg/ml (group 2). There were following exclusion criteria: acute coronary syndrome <3 months ago; acute or decompensated HF; history of stroke or transient ischemic attack <3 months old; active cancer; dementia and mental illness preventing the patient from signing informed consent.

The assessment of the HF functional class (FC) was carried out using the clinical state scale (CSS) in V. Yu. Mareev's modification and the six-minute walk test (6 MWT).

Echocardiography was performed using VIVID T8 system (GE Healthcare, USA) according to the standard methodology recommended by the American Society of Echocardiography and European Association of Echocardiography. The LV ejection fraction (LVEF) was determined by the Simpson's method. Preserved LVEF was considered 50% or more, mid-range — 40-49%, reduced — <40%. Assessment of LV diastolic function was carried out with determination of transmitral flow velocity characteristics and visualization of mitral annulus motion.

The serum NT-proBNP levels were determined using the Vector Best reagent kit (Russia) by enzyme-linked immunosorbent assay on an Immulite 1000 analyzer (DPC, USA).

Statistical processing was performed using the Statistica 13.0. An analysis of the distribution type was carried out using the Shapiro-Wilk and Kolmogorov-Smirnov tests. Quantitative characters are presented as follows: mean values and standard deviations with normal distribution (M±SD); median, lower and upper quartiles with non-normal distribution (Me [LQ; UQ]). For qualitative characters, the absolute frequency of character manifestation and detection percentage (%) were estimated. For a statistical analysis of the normally distributed

Table 3

Clinical and laboratory characteristics of groups (n=160)

Parameter	Group 1 (HF and HIV infection, n=100)	Group 2 (HF, n=60)	p
BMI, kg/m^2	20,8±4,0	28,1±6,3	<0,001
BMI >30 kg/m^2, abs./%	10/10,0	21/33,3	<0,001
SBP, mmHg	128,4±19,5	124,9±14,5	0,159
DBP, mmHg	79,1±15,0	85,2±7,4	0,004
Ascites, abs./%	12/12,0	1/1,7	0,044
Hepatomegaly, abs./%	66/66,0	19/31,7	<0,001
Splenomegaly, abs./%	32/32,0	1/1,7	<0,001
Hemoglobin, g/L	118,4 [101,7; 138,4]	129 8 [113,9; 149,0]	0,005
Fasting plasma glucose, mmol/L	5,1 [4,3; 6,8]	5,4 [4,8; 8,3]	0,128
Total cholesterol, mmol/L	5,1 [3,6; 6,5]	5,7 [2,8; 7,1]	0,098
CD4-T lymphocyte count, cells/mm^3	150 [43; 300]	-	-
Serum sodium, mmol/L	141,8±6,5	139,4±8,4	0,074
Serum potassium, mmol/L	4,0 [3,6; 4,5]	4,2 [3,8; 4,6]	0,541
Total bilirubin, μmol/L	11,0 [10,0; 16,0]	14,5 [9,6; 18;1]	0,726
ALT, ME/L	31,0 [20,1; 60,4]	22,4 [18,5; 24,3]	0,018
AST, ME/L	44,5 [30,3; 75,0]	25,8 [19,6; 31,9]	0,008
Serum creatinine, μmol/L	86,4 [66,2; 107,1]	79,1 [55,4; 101,3]	0,084
GFR (CKD-EPI), ml/min/1,73 m^2	84,3±32,0	77,6±18,1	0,028
GFR (CKD-EPI) <60 ml/min/1,73 m^2, abs./%	18/18,0	8/13,3	0,580
ESR, mm/h	32,2 [25,3; 59,0]	18,6 [15,3; 34,8]	<0,001
C-reactive protein, mg/l	34,0 [12,1; 96,2]	4,8 [3,7; 9,8]	<0,001

Abbreviations: ALT — alanine aminotransferase, AST — aspartate aminotransferase, BMI — body mass index, DBP — diastolic blood pressure, ESR — erythrocyte sedimentation rate, GFR — glomerular filtration rate, HIV — human immunodeficiency virus, HF — heart failure, SBP — systolic blood pressure.

data obtained, the parametric methods were used — Student's t-test, for qualitative characters — chi-squared test. With non-normally distributed data, nonparametric statistics were used to compare quantitative and qualitative characters: the Wilcoxon T test and the Chi-squared test with Yates's correction, respectively. Differences were considered significant at p<0,05. A correlation analysis was carried out using Spearman's rank correlation coefficients.

Results

Table 1 presents the diagnostic criteria and characteristics of HF development in groups of subjects.

Given the comparability of the groups by severity of clinical manifestations, an objective assessment of the presence and incidence of HF in HIV-infected patients was performed based on NT-proBNP increase >125 pg/ml and amounted to 54%; on echocardiography LVEF decrease <50% — 32%; on LVDD — 40%; on combination of LVDD with an increase in the left atrial volume index >34 ml/m^2 and LV mass index (LVMI) >110 g/m^2 in men and >95 g/m^2 in women — 87%; on NT-proBNP increase >125 pg/ml and/or LVEF <50% and/or LVDD and changes in the cardiac structure — 98%.

HIV-infected patients had significantly lower LVEF, less severe LVDD, higher LVMI, and lower NT-proBNP levels. HIV-infected patients had significant moderate inverse relationship of LVEF (r=-0,43; p=0,015), E/e' (r=-0,32; p=0,045), LVMI (r=-0,46; p=0,002) and strong relationship of NT-proBNP (r=-0,54; p<0,001) with CD4 T-lymphocyte count in 1 mm^3 with HF symptoms and signs and an increase in NT-proBNP over 125 pg/ml..

Anamnestic characteristics of groups are presented in Table 2.

Clinical and laboratory characteristics of groups are presented in Table 3.

In group 1, there was a significantly higher prevalence of smoking, chronic alcoholism, drug use, chronic hepatitis C and cirrhosis (especially manifested by hepatomegaly and splenomegaly in combination with ascites and hepatic cytolysis), chronic pancreatitis, pneumonia and inflammatory diseases accompanied by higher erythrocyte sedimentation

Table 4

Structure of treatment of heart failure and comorbidities in groups (n=160)

Group of drugs (abs./%)	Group 1 (HF and HIV infection, n=100)	Group 2 (HF, n=60)	p
ACE inhibitors	25/25,0	36/60,0	<0,001
ARA	5/5,0	21/35,0	<0,001
Diuretics (loop and/or thiazide)	14/14,0	17/28,3	0,044
Beta blockers	10/10,0	44/73,3	<0,001
Spironolactone	25/25,0	6/10,0	0,025
Digoxin	0/ 0	3/5,0	0,014
Anticoagulants	2/2,0	3/5,0	0,558
Antiplatelet agents	0/0	36/60,0	<0,001
Statins	1/1,0	12/20,0	<0,001
Antianginal agents (calcium antagonists, nitrates, trimetazidine, ivabradine)	2/2,0	21/35,0	<0,001
Blood glucose-lowering drugs	2/2,0	3/5,0	0,558
Antibiotics	73/73,0	6/10,0	<0,001
NSAIDs	48/48,0	12/20,0	0,002
Fluconazole	11/11,0	0/0	0,020
Iron supplements	11/11,0	0/0	0,020
Proton pump inhibitors	23/23,0	5/8,3	0,032

Abbreviations: ACE inhibitors — angiotensin-converting enzyme inhibitors, ARA — angiotensin receptor antagonists, HIV — human immunodeficiency virus, HF — heart failure, NSAIDs — non-steroidal anti-inflammatory drugs.

rate and C-reactive protein concentration, and lower hemoglobin level. It was associated with symptom variety, including typical for HF, such as dyspnea, palpitation, weakness, fatigue, fluid retention, liver enlargement. HF patients without HIV infection were older, mostly women, more often had a history of hypertension, coronary artery disease, myocardial infarction, coronary artery bypass grafting or percutaneous coronary intervention, atrial fibrillation, and were more likely to use drugs for HF treatment. HIV-infected patients had a lower body mass index.

Table 4 presents the structure of treatment of HF and comorbidities in groups.

HIV-infected patients were significantly less likely to use all groups of drugs for HF treatment, with the exception of spironolactone, which is used for treatment of ascites in HIV-infected patients.

Patients with HIV infection significantly more often used drugs for multimorbidity treatment, such as antibiotics, non-steroidal anti-inflammatory drugs, fluconazole, iron supplements, proton-pump inhibitors.

Only 19% of HIV-infected patients received antiretroviral therapy.

The studies presented reflect problems associated with NT-proBNP levels. This study did not reveal As a result of the study, no early markers of myocardial damage were detected (galectin-3, sST2, micro-ribonucleic acid-27), which could confirm the presence of heart failure in patients with normal levels of NT-proBNP. The study included patients with acute inflammatory pathology (pneumonia, acute inflammatory diseases), which can be caused by unconfident accompanied myocarditis.

The limitations of the study are a small sample size of patients with HIV infection and significant heterogeneity of this group by NT-proBNP levels. The study did not assess early markers of myocardial injury (galectin-3, sST2, micro-ribonucleic acid-27), which could confirm the HF in patients with normal NT-proBNP levels. The study included patients with acute inflammatory pathology (pneumonia, acute inflammatory diseases), which could be the cause of unverified myocarditis, accompanied by transient HF.

Conclusion

The prevalence of HF in HIV-infected patients is unknown. In our study, the incidence of HF in HIV-infected patients, estimated based on the clinical symptoms and NT-proBNP increase >125 pg/ml, was 54%. In study by Alvi RM, et al., the HF incidence in HIV-infected patients was 16,8% [5]. A lower incidence rate of HF was probably due to the fact that 90% of the patients received antiretroviral therapy, 62% of them with a health-promoting effect. Therefore, this therapy suppressed immuno-medi-

ated damage to cardiomyocytes and fibroblasts. The authors found that an NT-proBNP increase in HIV-infected patients is associated with cocaine use, LVEF decrease, progression of HF and significant CD4 T-lymphocyte count decrease in 1 mm^3. We also obtained significant relationships between LVEF and NT-proBNP with the CD4 T-lymphocyte count in 1 mm^3.

The prevalence of LVEF changes in HIV-infected patients is debatable. In our study, only 6% of HIV-infected patients with HF had LVEF <40%, 26% — from 40 to 49%, 68% — >50% According to European data, HIV-infected people are more likely to have HF with reduced EF (40%), in 30% — HF with preserved EF, in 15% — HF with mid-range EF; in 15% the diagnosis of HF is uncertain [6]. The difference of above-mentioned data with our study can be explained by the overdiagnosing of HF in patients with normal NT-proBNP levels. Using earlier and more accurate biomarkers of myocardial injury, such as galectin-3 or sST2, the diagnosis of HF would be confirmed in every third patient with a normal level of myocardial stress [7]. There are studies that are consistent with our findings on LVEF in HIV-infected patients. Thus, in study by Chaudhary S, et al., cardiomegaly with LV contractile function reduction was recorded only in 8 of 73 HIV-infected patients; left heart structural changes were detected in 52,1%, while NT-proBNP increase was found only in 26,7% [8].

An analysis of the results of our study showed that there are certain difficulties in the diagnosis of HF in HIV-infected patients, which are associated with a high incidence of comorbidities and multimorbidities. This, on the one hand, leads to the symptoms and signs typical for HF that are not related to it, and on the other hand, affecting the clinical course of HF with both increasing and decreasing the concentration of natriuretic peptides [9]. According to study by Wagnew F, et al., anemia observed in 22,3% (95% CI 18,5-26,0%) of HIV-infected patients, clinically manifested by the dyspnea and tachycardia without established HF [10]. In study by Christensen S, et al., the incidence of hepatitis B (5,9% vs 03,%, p<0,001) and hepatitis C (8,8% vs 0,3%, p<0,001) in HIV-infected patients with hepatomegaly, and in case of cirrhosis, with ascites and splenomegaly, was significantly higher in comparison with patients without HIV infection [11].

Visceral adiposity, autoimmune hypothyroidism, a high frequency of taking mineralocorticoid receptor antagonists to suppress congestion in cirrhosis in HIV-infected patients can lead to a decrease in the natriuretic peptides levels, even with HF [12]. Taking certain antiviral drugs, such as synthetic low-molecular weight interferon inducer tilorone, which induces the formation of interferons (alpha, beta, gamma, lambda), suppress myocardial stress estimated by the NT-proBNP concentration [13].

The use of antiretroviral therapy in HIV-infected patients, especially in the high-dose regimen at the beginning of treatment, exacerbates myocardial dysfunction and, with prolonged use, leads to myocardial fibrosis and severe HF with high NT-proBNP levels with prolonged use [14].

Given the variety of clinical symptoms and high prevalence of multimorbidities in HIV-infected patients, Scherzer R, et al. divided patients depending on the level of biomarker increase into 3 clusters with a certain phenotype of myocardial injury [15]. Cluster 1 (n=143) was characterized by the lowest level of markers such as NT-proBNP, C-reactive protein, sST2 and others. In cluster 2, a predominant increase in sST2, NT-proBNP, and growth differentiation factor 15 (cardiac phenotype) was found. Cluster 3 (n=103) had higher levels of C-reactive protein, IL-6, and D-dimer (inflammatory phenotype). This approach of dividing HIV patients into clusters allowed the authors to show the versatility of pathogenetic mechanisms and forms of HF. Inflammatory phenotype was associated with increased risk of LVDD by 51% and a 3,3-fold higher 7-year mortality risk; cardiac phenotype was associated with increased risk of pulmonary hypertension by 67% and a 3,1-fold higher risk of all-cause mortality.

Thus, the study of HF development in HIV-infected people has demonstrated that many aspects of the prevalence, diagnosis, and therapy selection have not been investigated; current data are contradictory. Therefore, further research is necessary in order to improve the quality of life and prognosis of patients with HIV infection.

Conclusion

The HF prevalence in hospitalized HIV-infected patients, estimated on the basis of symptoms and NT-proBNP increase >125 pg/ml, was 54%; on the basis of LVEF decrease <50% — 32%. The clinical picture of HIV-infected patients is characterized by various symptoms, including those typical for HF with normal NT-proBNP level, due to the high prevalence of comorbidities, multimorbidities and concurrent medication. HIV-infected patients were significantly less likely to use all groups of drugs for HF treatment, with the exception of spironolactone, compared with subjects without HIV infection. Only 19% of HIV-infected patients received antiretroviral therapy.

Relationships and activities: not.

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