UDC 616.12-008.331.1-055.1:575.174
DOI: http://doi.org/10.31928/2664-4479-2025.4.713

Intracardiac hemodynamic parameters in male patients with essential hypertension carrying polymorphic variants of the cardiotrophin-1 gene

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The aim – improvement of diagnosing the intracardiac and systemic hemodynamic changes in male left ventricular hypertrophy (LVH) patients with underlying essential hypertension (EH) being carriers of polymorphic variants of the corresponding coding gene (rs8046707) using the cardiotrophin-1 (CT-1) biomarker.

Materials and methods. The study involved 70 male individuals without signs of cardiovascular pathology and LVH of other etiology, aged 48.81±0.78 years, and 50 male LVH patients with underlying EH without signs of chronic heart failure, aged 50.62±0.73 years. All study participants underwent a standard echocardiographic examination, blood serum CT-1 concentration enzyme-linked immunosorbent assay (ELISA), and CT-1 gene polymorphism (rs8046707) test of venous blood samples by polymerase chain reaction.

Results. The CT-1 blood serum concentration in GG-genotype individuals without signs of cardiovascular pathology (n=31) was found to be lower than that in carriers of GA + AA genotypes (n=39), being equaled to 55.77±2.53 pg/ml and 92.46±1.54 pg/ml, respectively (p<0.001). The CT-1 blood plasma concentration in LVH subjects was significantly higher than in the control group patients, equaling 188.22±7.95 pg/ml and 282.33±11.52 pg/ml in GG males (n=22) and in GA + AA genotype carriers (n=28), respectively (p<0.001). The Spearman rank correlation method was used to establish a correlation between blood plasma CT-1 concentration and echocardiography readings.

Conclusions. Stage II EH patients being carriers of CT-1 gene GA + AA genotypes demonstrated higher blood plasma CT-1 concentrations and correlation with echocardiography indicators. This fact suggests that changes in wall thickness and myocardial mass of EH II patients can be diagnosed using the above-mentioned marker as an additional ascertaining indicator.

Key words: left ventricular hypertrophy, essential hypertension, cardiotrophin-1.

Cardiovascular diseases are the number one cause of death worldwide. According to WHO estimates, 17.9 million people die annually of cardiovascular diseases, which accounts to 31 % of all lethal cases in the world [1–3]. According to the European Association of Cardiology, the worldwide EH prevalence among adults is about 30–45 %; the disease prevalence aggravates progressively with age and makes about 60 % among people over 60 years old [5]. Left ventricular (LV) hypertrophy is the earliest cardiac EH complication and is known to be a major risk factor for the development of a number of the disease complications. A buildup of LV wall tension, caused, inter alia, by EH-driven cardiac load, stimulates myocyte hypertrophy, collagen and fibroblast formation, and, therefore, a myocardial remodeling with a disproportionate

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Стаття надійшла до редакції 25 червня 2025 року

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Received on 25.06.2025

growth of fibrous tissue [1, 3–5]. Since LVH development is associated with degenerative changes in cardiac myocytes and abnormal accumulation of collagen in the interstitium, new biomarkers of such processes are urgently needed. For example, the research community has paid much attention to cardiotrophin-1 (CT-1), a member of the interleukin-6 (IL-6) cytokine superfamily, in recent years. The latter is considered one of the key regulators of cardiomyocyte hypertrophy and hyperplasia. CT-1 also influences the intensity of apoptosis and myocardium sensitivity to ischemia [6–8]. Therefore, CT-1 peptide, which is produced by cardiomyocytes and fibroblasts of the heart under conditions of biomechanical stress, can be considered a possible biomarker for early LVH diagnosis under the conditions of impossible instrumental tests and during screening of such pathological conditions even prior to the development of clinical changes.

MATERIALS AND METHODS

The study included 120 men from the Podillia region (Ukraine). The control group consisted of 70 men without clinical signs of cardiovascular disease at the time of the study. The main group included 50 men diagnosed with LVH secondary to EH.

At the time of screening, EH patient groups included LVH subjects without clinical signs of chronic heart failure. For example, the criteria for exclusion from the study included the confirmed secondary nature of arterial hypertension, endocrine diseases, blood system diseases, chronic obstructive pulmonary disease, significant impairment of kidney function (CKD – EPI glomerular filtration rate less than 60 ml/min), liver dysfunction, data from anamnesis and documents about a history of EH complications (myocardial infarction, acute cerebrovascular accident(s)), clinically significant symptoms (stable cardiac angina of II–IV functional classes) or unstable forms of coronary heart disease, anamnestic indications of coronary heart disease, the development of which preceded the EH onset. All patients were observed from December 2017 to July 2018 and hospitalized in the Vinnytsia Regional Specialized Clinical Dispensary of Radiation Protection of the Ministry of Health of Ukraine and the Military Medical Clinical Center of the Central Region of the Air Force of Ukraine.

The study protocol was approved by the local ethics committee. Each participant of the study gave the informed consent to participate in the study. All study subjects underwent the Doppler echocardiography examination under the standard protocol. Hypertrophy of the left ventricle was diagnosed according to echocardiography results, including left ventricular myocardial mass index (LVMI) reduced to a height$^{2.7}$, if the index was higher than 50 g/m$^{2.7}$. Left ventricular remodeling classification was based on the criteria recommended by the American Society of Echocardiography and the European Association of Cardiovascular Imaging, as well as on national Ukrainian clinical protocols for hypertension diagnosis and treatment (Ministry of Health of Ukraine, 2024). The ECG was performed according to the generally accepted method in 12 standard leads. Blood pressure was measured in accordance with the recommendations of WHO, ESC/ESH, ACC/AHA experts, and Guidelines for Management of Arterial Hypertension (2018–2024). Body mass index (BMI) was calculated using the Quetelet formula (BMI = body weight (kg) / height (m)^2) and obesity was classified according to WHO recommendations *(Table 1)*. All patients received standard antihypertensive therapy in accordance with the Unified Clinical Protocol of Medical Care for Arterial Hypertension approved by the Ministry of Health of Ukraine, as well as the clinical guidelines of the European Society of Hypertension (ESH) and the European Society of Cardiology (ESC).

Table 1

Clinical and anthropometric characteristics of the study participants

Parameter	Control Group Men (n=70)	Men with Grade II EH (n=50)
Age, years	48.81±0.78	50.62±0.73
Body weight, kg	78.16±1.32	81.12±1.51
BMI, kg/m^2	25.23±0.31	25.94±0.29
GFR, ml/min/1.73^2	110.60±2.47	104.80±2.59
SAP, mm Hg	120.9±1.0	160.00±1.67
DAP, mm Hg	78.86±0.90	97.80±0.90
Duration of EH, years	—	8.70±0.60

EH – essential hypertension; BMI – body mass index; GFR – glomerular filtration rate (estimated by CKD-EPI); SAP – systolic arterial pressure; DAP – diastolic arterial pressure.

Table 2

Indicators of the structure and function of the myocardium and systemic hemodynamics in Stage II EH male patients, carriers of polymorphic variants of the CT-1 gene (M±m)

Description	Control group males (n=70)		Stage II EH males (n=50)		R
	Genotype GG (n=31)	Genotypes GA+AA (n=39)	Genotype GG (n=22)	Genotypes GA+AA (n=28)	
	1	2	3	4	
PWT, cm	0.92±0.0 1	0.95±0.01	1.16±0.02	1.29±0.02	p_{3-1}*, p_{4-1}*, p_{3-2}*, p_{4-2}*, p_{4-3}*
SWT, cm	0.93±0.0 1	0.94±0.01	1.19±0.02	1.25±0.02	p_{3-1}*, p_{4-1}*, p_{3-2}* p_{4-2}*, p_{4-3}*
RWT, RU	0.40±0.01	0.41±0.01	0.480±0.008	0.500±0.007	p_{3-1}*, p_{4-1}*, p_{3-2}*, p_{4-2}*, p_{4-3}*
LVMI, g/m$^{2.7}$	36.76±2.75	38.68±2.43	62.0±0.9	64.25±0.70	p_{3-1}*, p_{4-1}*, p_{3-2}*, p_{4-2}*, p_{4-3}*
EF, %	64.68±1.16	63.21±1.11	61.25±1.55	58.53±0.97	p_{3-1}*, p_{4-1}*, p_{4-2}*
LA, cm	3.24±0.09	3.38±0.07	3.78±0.09	4.19±0.07	p_{3-1}*, p_{4-1}*, p_{4-2}*, p_{4-3}*
SAP, Hg mm	121.42±1.54	120.7±1.3	161.42±2.12	163.91±1.33	p_{3-1}*, p_{4-1}*, p_{3-2}*, p_{4-2}*
DAP, Hg mm	75.80±1.27	77.84±1.28	98.77±1.46	101.1±0.9	p_{3-1}*, p_{4-1}*, p_{3-2}*, p_{4-2}*
HR, bpm	67.00±1.22	69.03±1.25	76.31±1.91	77.88±1.48	p_{4-1}*, p_{4-2}*, p_{3-1}*, p_{3-2}*

Note: the difference is considered statistically reliable * if p<0.05; PWT – posterior wall thickness; SWT – septal wall thickness; RWT – relative wall thickness; LVMI – left ventricular mass index (g/m$^{2.7}$); EF – ejection fraction; LA – left atrial size; SAP – systolic arterial pressure; DAP – diastolic arterial pressure; HR – heart rate.

To establish the cardiotrophin-1 gene polymorphism (rs8046707), we took DNA samples from the peripheral blood using a set of reagents for isolating genomic DNA from whole blood by polymerase chain reaction. Genotyping of the CT-1 gene was carried out jointly with specialists of the Research Institute of Genetic and Immunological Pathology Studies and Pharmacogenetics of the Ukrainian Medical and Stomatological Academy (Poltava, Head – O. Shlykova). CT-1 blood serum concentration was measured using an enzyme – linked immunosorbent assay (ELISA) method.

All obtained data were subjected to mathematical processing on a personal computer using the standard statistical package Statistica 17.0. Microsoft Excel was used for initial preparation of tables and intermediate calculations. Values are reported as mean ± standard deviation or as a percentage. Appropriate statistical methods were used to assess differences between groups of patients: Student's t-test, Spearman's correlation, Mann – Whitney U-test. If normality was not confirmed, the Mann – Whitney U test was used. Differences were considered significant at p<0.05.

RESULTS

Previously, we published the frequency variant of gene CT-1 genotypes distribution (rs8046707) [10]. It was established that males without signs of cardiovascular pathology had the frequency of GG genotype of CT-1 gene (n=31) and a pool of GA + AA genotypes (n=39) equal to 44.29 and 55.71 %, respectively. The frequency distribution of CT-1 gene genotypes in LVH male patients with underlying EH for GG (n=22) and GA + AA genotype carriers (n=28) was 44.00 % and 56.00 %, respectively.

We found that structural and functional myocardium indicators in subjects of the control group were within the generally accepted normative readings for this age group [6]. The ECG data of Stage II EH patients suggested higher structural and functional myocardium indicators, such as end diastolic dimension (EDD), end systolic dimension (ESD), end diastolic volume index (EDV), end systolic volume index (ESV), cardiac index (CI), thickness of the back wall of the left ventricle (PWT), thickness of the interventricular septum (SWT), and LVMI, compared to that in the control group subjects (p<0.05) *(Table 2)*.

The analysis of the LV systolic function established that the value of the LV ejection fraction (EF) was statistically significantly higher in the subjects of the control group (p<0.001). EH II patients had statistically significantly higher readings of both systolic and diastolic blood pressure and heart rate compared to the control group subjects (p<0.001).

Statistical analysis of the LV geometric pattern distribution in Stage II EH male patients showed that

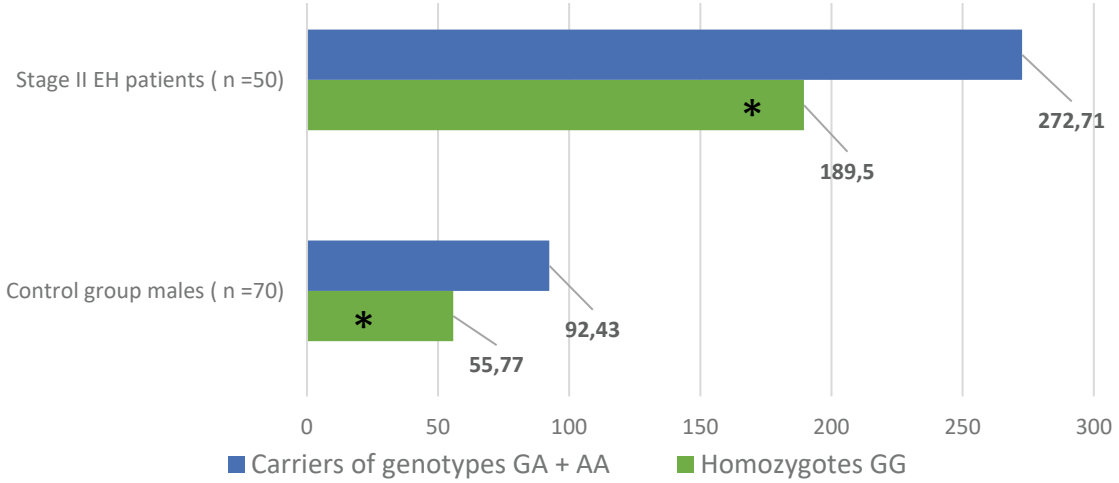

Figure. **Blood plasma cardiotrophin-1 concentration in the control group males, Stage II EH patients and carriers of various variants of the CT-1 gene, pg/ml. Note: the difference is considered statistically reliable (p<0.001) when compared with: * – GG genotype of the CT-1 gene within the group.**

concentric and eccentric LVH occurred in 38 (76 %) and 12 (24 %) individuals, respectively (p<0.05).

The next stage of the study involved the analysis of intracardiac and systemic hemodynamics in carriers of various variants of CT-1 gene. Stage II EH genotype GA + AA carriers had statistically significantly higher LV wall parameters, such as PWT, SWT, relative wall thickness (RWT) and left atrial size (LA), than those of genotype GG males (p<0.05) *(Table 1)*. According to the obtained data, Stage II EH patients had CT-1 blood serum concentration significantly higher than this in the control group males (p<0.05). The highest peptide concentration was determined in Stage II EH carriers of CT-1 genotypes GA + AA (p<0.05) *(Figure)*.

We used the Spearman's rank correlation, which indicates a possible statistically significant relationship between two variables, to measure a correlation between blood plasma CT-1 concentration and parameters of intracardiac and systemic hemodynamics in male subjects. Stage II CT-1 blood serum concentration in EH males demonstrated a statistically significant correlation with parameters of wall thickness and myocardial mass, such as PWT, SWT, RWT, LVMI, and DT *(Table 3)*.

While analyzing the correlation between hemodynamic indicators and CT-1 concentration in Stage II EH patients, carriers of polymorphic variants of the CT-1 gene, a weak positive correlation with such indicators as PWT, SWT, RWT, and LVMI in GA + AA genotype carriers was determined *(Table 4)*.

Study of the frequency of LV hypertrophy type distribution in EH patients, carriers of different CT-1 gene genotypes, showed concentric and eccentric LVH in 16 (72.73 %) and 6 (27.27 %) GG genotype male carriers, respectively (p<0.05), while male genotype GA + AA carriers had concentric and eccentric LVH in

Table 3

Correlative indices of blood plasma CT-1 and indicators of intracardiac and systemic hemodynamics in EH male patients (Spearman's rank correlation)

Description	CT-1 concentration	
	Male patients with uncomplicated EH (n=50)	
	R	p
PWT, cm	+0.42	<0.05
SWT, cm	+0.30	<0.05
RWT, RU	+0.33	<0.05
LVMI, g/m$^{2.7}$	+0.36	<0.001
EF, %	–0.27	>0.05
DT, ms	+0.38	<0.05
E/A, RU	–0.028	>0.05
E/E', RU	+0.0 17	>0.05
IVRT, ms	+0.11	>0.05
LA, cm	+0.15	>0.05
SAP, Hg mm	+0.20	>0.05
DAP, Hg mm	+0.20	>0.05
HR, bpm	–0.025	>0.05

R – Spearman correlation coefficient; PWT – posterior wall thickness (cm); SWT – septal wall thickness (cm); RWT – relative wall thickness (relative units); LVMI – left ventricular mass index (g/m$^{2.7}$); EF – ejection fraction (%); DT – deceleration time of early diastolic filling (ms); E/A – ratio of early (E) to late (A) diastolic transmitral flow velocities (relative units); E/E' – ratio of early transmitral flow velocity to early diastolic mitral annular velocity (relative units); IVRT – isovolumic relaxation time (ms); LA – left atrial size (cm); SAP – systolic arterial pressure (mmHg); DAP – diastolic arterial pressure (mmHg); HR – heart rate (beats per minute, bpm).

Table 4

Correlation between blood plasma CT-1 concentration and indicators of intracardiac and systemic hemodynamics in male EH patients, carriers of polymorphic variants of the CT-1 gene (Spearman's rank correlation)

Description	CT-1 concentration			
	Homozygotes GG (n=22)		Genotype GA + AA carriers (n=28)	
	R	p	R	p
PWT, cm	+0.02	<0.05	+0.42	<0.05
SWT, cm	+0.18	>0.05	+0.30	<0.05
RWT, RU	+0.23	>0.05	+0.33	<0.05
LVMI, g/m$^{2.7}$	+0.16	<0.05	+0.36	<0.05
EF, %	–0.31	>0.05	–0.23	>0.05
DT, ms	+0, 31	>0.05	+0.26	>0.05
E/A, RU	–0.0 1	>0.05	–0.0 4	>0.05
E/E', RU	+0.0 5	>0.05	+0.0 7	>0.05
IVRT, ms	+0.21	>0.05	+0, 12	>0.05
LA, cm	+0.35	>0.05	+0.15	>0.05
SAP, Hg mm	+0.40	>0.05	+0.13	>0.05
DAP, Hg mm	+0.10	>0.05	+0.21	>0.05
HR, bpm	–0.032	>0.05	–0.13	>0.05

R – Spearman correlation coefficient; PWT – posterior wall thickness (cm); SWT – septal wall thickness (cm); RWT – relative wall thickness (relative units); LVMI – left ventricular mass index (g/m$^{2.7}$); EF – ejection fraction (%); DT – deceleration time of early diastolic filling (ms); E/A – ratio of early (E) to late (A) diastolic transmitral flow velocities (relative units); E/E' – ratio of early transmitral flow velocity to early diastolic mitral annular velocity (relative units); IVRT – isovolumic relaxation time (ms); LA – left atrial size (cm); SAP – systolic arterial pressure (mmHg); DAP – diastolic arterial pressure (mmHg); HR – heart rate (beats per minute, bpm).

22 (78.57 %) and 6 (21.43 %), respectively (p<0.05). This is an important finding because, according to a number of studies, concentric LVH coronary complications occur more often than systolic dysfunction, which means an unfavorable prognosis for patients [2].

DISCUSSION

It is known, that not only demographic, neuroendocrine, but also genetic factors play a significant role in LVH development and progression. It was established that Stage II EH patients had higher registration frequency of CT-1 genotype GA + AA carriers (rs8046707) than GG homozygotes (p<0.05) [8]. Such regularity was discovered by scientists S.Z. Lutz, O. Franck et al. in the German population, as GA genotype turned out to be the most common among Germans [9, 10]. In both German and Ukrainian populations, the AA genotype is rare one. Although CT-1 protein is normally found in liver, adipose tissue, and the respiratory system, it mainly influences the heart as it is synthesized by the myocardium. As mentioned above, CT-1 peptide is produced by cardiomyocytes and cardiac fibroblasts under the conditions of biomechanical stress and under the influence of humoral factors. Moreover, this peptide has pronounced hypertrophic, hyperplastic and antiapoptotic effects. It is produced to protect the myocardium by triggering cell proliferation and survival, exerts its hemodynamic effects and endocrine properties, and finally makes the heart prepared for pathological conditions. In fact, it induces myocyte hypertrophy and collagen synthesis [11–13]. According to data received from male patients with uncomplicated EH, the highest peptide concentration was found in Stage II EH subjects being genotype GA + AA carriers (p<0.05). This finding became the basis for the analysis of structural and functional indicators of the myocardium in male 40–60 – year old Podillia inhabitants.

The Spearman's rank correlation revealed a relationship between blood plasma CT-1 concentration and parameters of intracardiac and systemic hemodynamics in male subjects. We established a positive correlation of blood plasma CT-1 concentration with LV

parameters (end-systolic size, PWT, end-diastolic and end-systolic volume index, left ventricular myocardial mass index). Also, a correlation with LV indicators was found in genotype GA + AA male carriers. That means that even minor changes in the structure of the heart are reflected to some extent in the blood plasma CT-1 concentration. According to the literature, CT-1 blood plasma concentration is positively correlated with an increase in the mass of the myocardium of the left ventricle and elevates in LVH-diagnosed essential hypertension patients [14–16]. Considering the fact that CT-1 blood serum concentration reflects a number of different processes in cardiomyocytes and connective tissue of the heart during the development of LVH of multiple etiology [9], we believe that this biomarker can become an additional diagnostic indicator of hypertrophic changes in the myocardium.

CONCLUSIONS

1. Left ventricular hypertrophy patients with underlying essential hypertension without clinical signs of chronic heart failure have statistically higher cardiotrophin-1 blood serum concentration compared to individuals without any cardiovascular pathology.

2. Left ventricular hypertrophy male patients, cardiotrophin-1 genotype GA + AA carriers, have statistically higher cardiotrophin-1 concentration.

3. Correlations between blood plasma cardiotrophin-1 concentration and indicators of intracardiac hemodynamics suggest changes in wall thickness and myocardial mass of stage II essential hypertension patients, which are signs of cardiac remodeling in the early stages of the disease.

There is no conflict of interest.

Author contributions: research concept and design – V.Z., S.L., S.F.; collection and/or assembly of data – I.P., L.S., O.S.; data analysis and interpretation – S.L., M.M., I.P., S.F.; writing the article, final approval of the article – V.Z., M.M.

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Параметри внутрішньосерцевої гемодинаміки в чоловіків з есенціальною гіпертензією – носіїв поліморфних варіантів гена кардіотрофін-1

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Мета роботи – поліпшити діагностику особливостей параметрів внутрішньосерцевої та системної гемодинаміки при розвитку гіпертрофії лівого шлуночка (ГЛШ) у чоловіків з есенціальною гіпертензією (ЕГ) за допомогою біомаркера кардіотрофіну-1 (СТ-1) при носійстві поліморфних варіантів відповідного кодувального гена (rs8046707).

Матеріали і методи. Обстежено 70 чоловіків без ознак серцево-судинної патології та ГЛШ іншої етіології віком (48,81±0,78) року та 50 чоловіків із ГЛШ унаслідок ЕГ без ознак хронічної серцевої недостатності віком (50,62±0,73) року. Усім учасникам дослідження проводили стандартне ехокардіографічне обстеження, визначали концентрацію СТ-1 у плазмі крові методом імуноферментного аналізу та визначали поліморфізм (rs8046707) гена СТ-1 у зразках венозної крові методом полімеразної ланцюгової реакції.

Результати. Концентрація СТ-1 у плазмі крові в осіб без ознак серцево-судинної патології гомозигот GG нижча, ніж у носіїв генотипів GA + AA: (55,77±2,53) пг/мл (n=31) проти (92,46±1,54) пг/мл (n=39) (p<0,001). Рівень СТ-1 у плазмі крові у представників із ГЛШ значущо вищий, ніж у контрольній групі: у чоловіків із генотипом GG – (188,22±7,95) пг/мл (n=22); у носіїв генотипів GA + AA – (282,33±11,52) пг/мл (n=28) (p<0,001). Методом рангової кореляції за Спірменом виявлено кореляцію між концентрацією СТ-1 у плазмі крові та показниками ехокардіографії.

Висновки. У хворих з ЕГ II стадії – носіїв генотипів GA AA гена СТ-1 виявлено вищі рівні концентрації СТ-1 у плазмі крові та кореляційні зв'язки з показниками ехокардіографії. Це свідчить про те, що зміни товщини стінки та маси міокарда у пацієнтів з ЕГ II можуть бути діагностовані із застосуванням зазначеного маркера як додаткового показника.

Ключові слова: гіпертрофія лівого шлуночка, есенціальна гіпертензія, кардіотрофін-1.