ORIGINAL ARTICLE

Incidence of chronic heart failure with preserved left ventricular ejection fraction in patients with hypertension and isolated mild diastolic dysfunction

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KEY WORDS

heart failure with preserved ejection fraction, impaired left ventricular relaxation

ABSTRACT

INTRODUCTION Heart failure (HF) with preserved ejection fraction (HFPEF) is still a challenge in clinical practice. The prognosis of patients with HFPEF is similar to or only slightly better than that of patients with HF with reduced ejection fraction (HFREF). Impaired relaxation is the mildest form of diastolic dysfunction, which should not be accompanied by symptoms of HFPEF.

OBJECTIVES The aim of the study was to assess the incidence of chronic HFPEF in patients with hypertension and isolated mild diastolic dysfunction.

PATIENTS AND METHODS It was a cross-sectional study including 210 patients (mean age, 56.11 ±6.24 years; women, 58%) with isolated abnormalities of left ventricular relaxation and arterial hypertension. In addition, we identified patients with type 2 diabetes to compare the incidence of HFPEF between patients with and without diabetes. HFPEF was diagnosed when clinical symptoms of HF were present simultaneously with echocardiographic markers of elevated left ventricular diastolic pressure, pulmonary congestion on chest X-ray, or elevated serum brain natriuretic peptide (BNP) levels.

RESULTS HFPEF was diagnosed in 42% of the patients with impaired relaxation. An elevated left atrial volume index (>34 ml/m²) was observed in 38% of the patients; E/e' ratio exceeding 8, in 37%; elevated BNP levels, in 39%; and pulmonary congestion on chest X-ray, in 41%. Independent predictors of HFPEF were age, systolic blood pressure of 140 mmHg or higher, type 2 diabetes, coronary artery disease, and an estimated glomerular filtration rate of less than 60 ml/min/1.73 m². In diabetic patients, a positive correlation was found between an insulin dose (>80 units/day) and BNP levels.

CONCLUSIONS Patients with isolated relaxation abnormalities constitute a clinically heterogeneous group because some of these individuals present with symptoms of HFPEF and a simultaneous increase in BNP levels. Therefore, the question of whether diastolic dysfunction is mild should be readdressed, and it should be emphasized that these patients have a serious prognosis with the risk of HF. In diabetic patients, a positive correlation between high insulin doses and BNP levels requires further research.

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INTRODUCTION In approximately 30% to 50% of newly diagnosed patients with heart failure (HF), systolic function assessed by measuring the left ventricular ejection fraction (LVEF) is normal or near normal. These patients have diastolic HF, known today as HF with preserved ejection fraction (HFPEF), which may be either isolated or, more often, accompanies systolic HF with reduced ejection fraction (HFREF). The etiology of HFPEF is often complex. Apart from old age, its common causes include hypertension, obesity, left ventricular (LV) systolic dysfunction, chronic

coronary artery disease (CAD), hypertrophic cardiomyopathy, congenital heart defects associated with ventricular hypertrophy, and diabetes.[1-15]

According to the European Society of Cardiology (ESC), the diagnosis of HFPEF requires 4 criteria to be met: the presence of signs and symptoms typical of HF, normal or only mildly reduced LVEF, no LV dilation, and relevant structural heart disease or left ventricular diastolic dysfunction (or both).

The prognosis of patients with HFPEF is similar to or slightly better than that of patients with HFREF and has not improved over the recent decades.[3,4] The full onset of HFPEF is preceded by a clinically asymptomatic stage characterized only by echocardiographic signs of diastolic dysfunction. Based on the echocardiographic pattern, 3 grades of diastolic dysfunction have been distinguished: mild, called an "abnormal relaxation pattern"; moderate, called a "pseudonormal filling pattern"; and severe, known as a "restrictive filling pattern". An abnormal relaxation pattern without systolic dysfunction is believed to be the earliest and mildest form of diastolic dysfunction that generally should not lead to HFPEF. This common belief makes clinicians less alert to the possible signs and symptoms of HFPEF because they do not suspect it in their patients. However, our previous unpublished findings indicate that patients with an abnormal relaxation pattern constitute a diverse group and differ not only in clinical symptoms but also in the results of noninvasive cardiac tests conducted to evaluate myocardial function. This indicates the need not only to intensify the causative treatment of HFPEF but also to question the general belief that impaired relaxation is a relatively "mild" form of diastolic dysfunction.

The aim of the study was to assess the relationship between the incidence of chronic HFPEF in patients with hypertension and isolated relaxation abnormalities (without systolic dysfunction) and hypertension.

PATIENTS AND METHODS It was a cross-sectional cohort study. At baseline, the study included 410 hypertensive patients treated at an outpatient cardiac clinic of the Świętokrzyskie Centre of Cardiology in Kielce, Poland, who gave their informed consent to participate in the study. The study was approved by a local bioethics committee. In all patients, hypertension was diagnosed before enrollment to the study as systolic blood pressure of 140 mmHg or higher and/or diastolic blood pressure of 90 mmHg or higher on at least 2 separate measurements, or hypotensive therapy initiated due to elevated blood pressure.[16] At the time of entry, all participants were treated with antihypertensive agents.

The inclusion criteria were as follows: 1) an abnormal relaxation pattern on echocardiography (an E/A ratio of less than 1.0 and deceleration time [DT] exceeding 200 ms in hypertensive patients younger than 50 years of age, and an E/A ratio of less than 0.75 and DT exceeding 220 ms in patients aged 50 years or older); 2) normal global LVEF on echocardiography (≥50%); and 3) lack of conditions that might hamper the assessment of diastolic dysfunction, such as tachycardia (heart rate exceeding 100 bpm), atrial fibrillation or atrial flutter, or the presence of a cardiac pacemaker.

We excluded patients with global systolic dysfunction (n = 40) and those in whom diastolic function could not be determined (n = 159). Other exclusion criteria were as follows: refusal to participate in the study (n = 1), anemia (hemoglobin levels, <13 g/dl for men and <12 g/dl for women), pulmonary diseases with dyspnea, vascular disease with edema, structural valvular heart defects, constrictve pericarditis, and uncontrolled hyperthyroidism and hypothyroidism. The final study sample included 210 patients with hypertension and isolated abnormalities of LV relaxation. Because diabetic patients are at higher risk of HF, we identified 140 patients with type 2 diabetes (68% of the study group) in order to assess the incidence of HFPEF in this patient group.

All patients underwent a full echocardiographic study (VIVID 9 ultrasound machine, GE, BT 12, Harten, Norway). The echocardiographic measurements were performed according to the guidelines of the American Society of Echocardiography and European Association of Echocardiography.[2,17] The echocardiographic markers of elevated LV end-diastolic pressure included an E/e' exceeding 8 and left atrial volume index (LAVI) exceeding 34 ml/m^2.[1,18] LV hypertrophy was defined as an LV mass index of more than 95 g/m^2 for women and more than 115 g/m^2 for men.[1]

A medical history was taken and a physical examination was performed in all study patients. The clinical symptoms of HF were classified according to the New York Heart Association (NYHA).[1] Routine laboratory tests were performed including the measurement of BNP levels (with a cut-off value of ≥35 pg/ml for chronic HF),[1] complete blood count, measurement of creatinine levels, glomerular filtration rate (GFR) using the Modification of Diet in Renal Disease formula, lipid levels, and thyroid-stimulating hormone levels. In addition, chest X-ray, the Bruce treadmill test for the diagnosis of CAD, and spirometry for a differential diagnosis of dyspnea were performed. The medical records of all patients were also reviewed.

The primary endpoint was the diagnosis of chronic HFPEF in patients with isolated relaxation abnormalities on the basis of the clinical presentation and imaging tests (ie, echocardiography and chest X-ray). The secondary endpoint was an increase in BNP levels to 35 pg/ml or higher in patients with HFPEF.

Statistical analysis A statistical analysis was conducted using the Statistica 7.0 PL software. Continuous variables were first evaluated for normal

TABLE 1 Echocardiographic findings, brain natriuretic peptide levels, chest X-ray results, and symptoms of heart failure with preserved ejection fraction in patients with impaired left ventricular relaxation

Variable	All patients (n = 210)	Men (n = 89)	Women (n = 121)	*P* value, men vs women
E/A ratio	0.51 ±0.12	0.54 ±0.12	0.50 ±0.10	<0.01
DT, ms	296.67 ±35.64	295.74 ±34.73	297.26 ±31.62	0.9
LAVI, ml/m²	32.74 ±6.70	29.14 ±6.17	32.40 ±6.29	0.02
LAVI >34 ml/m²	80 (38)	26 (29)	54 (45)	0.03
E/e' ratio	9.30 ±2.64	8.90 ±2.69	9.74 ±2.75	<0.01
E/e' ratio >8	77 (37)	25 (28)	52 (43)	0.03
LVEF, %	64.52 ±3.45	65.32 ±4.35	59.39 ±3.30	0.12
LV mass, g/m²	110.27 ±32.0	105.20 ±29.0	110.00 ±30.0	0.01
higher LV mass[a]	79 (38)	26 (29)	53 (44)	0.04
BNP, pg/ml	34.67 ±29.60	29.01 ±24.12	32.13 ±28.02	0.001
BNP >35 pg/ml	81 (39)	27 (30)	54 (46)	0.04
pulmonary congestion on chest X-ray	85 (41)	28 (32)	57 (47)	0.03
symptoms of HFPEF	89 (42)	30 (34)	59 (49)	0.04

Data are presented as mean ± standard deviation or number (percentage) of patients.

a increased LV mass was considered as more than 95 g/m² for women and more than 115 g/m² for men

Abbreviations: BNP, brain natriuretic peptide; DT, deceleration time; HFPEF, heart failure with preserved ejection fraction; LAVI, left atrial volume index; LV, left ventricular; LVEF, left ventricular ejection fraction

TABLE 2 Independent predictors of heart failure with preserved ejection fraction in a multivariate regression model

Variable	95% CI	OR	*P* value
age, y	1.07–1.25	1.15	<0.01
type 2 diabetes	2.78–15.67	6.65	<0.01
SBP ≥140 mmHg	2.16–14.36	5.57	<0.01
coronary artery disease	2.17–13.75	5.46	<0.01
GFR <60 ml/min/1.73 m²	1.11–2.89	1.56	0.04

Abbreviations: CI, confidence interval; GFR, glomerular filtration rate; OR, odds ratio; SBP, systolic blood pressure

distribution by the Shapiro–Wilk test. We also checked data distribution. The Mann–Whitney test was used to compare continuous variables. All continuous variables were expressed as mean ± standard deviation, and categorical variables were expressed as percentages. The χ^2 test was used to compare the qualitative data between the groups. The Pearson rank correlation coefficients were calculated to test the association between 2 variables with a normal distribution, respectively. All statistical tests were 2-sided.

A *P* value of less than 0.05 was considered statistically significant. The incidence of HF in the study group was measured. Univariate and multivariate logistic regression models were used to identify the independent predictors of HFPEF. All clinical, laboratory, and echocardiographic variables that showed an association with HFPEF in the univariate model and did not show significant correlations with another independent variables were included in the multiple regression analysis to determine the predictors of HFPEF.

RESULTS The study included 210 patients with hypertension and isolated relaxation abnormalities (women [58%] at a mean age of 60.83 ±5 years; men [42%] at a mean age of 53.11 ±6.32 years; *P* <0.001; mean age of the whole study group, 56.11 ±6.24 years). In the whole study group, the mean systolic blood pressure was 130.56 ±14.67 mmHg, while mean diastolic blood pressure was 79.73 ±10.12 mmHg; most participants (57%) had satisfactory blood pressure control. HFPEF was observed in 42% of the patients. It was observed more often in women than in men (*P* = 0.04). The mean values of systolic blood pressure in patients with HFPEF were higher (135.72 ±17.45 vs 130.12 ±12.3 mmHg; *P* <0.01) than in patients without HFPEF. Also diastolic blood pressure in HFPEF patients was higher than in individuals without HFPEF (80.80 ±11.30 vs 76.64 ±1.77 mmHg; *P* <0.02). The results of imaging tests and BNP measurements as well as the number of patients with HF are presented in TABLE 1.

In patients with impaired relaxation and clinical symptoms of HFPEF, serum BNP levels were from 104.4 to 480.2 pg/ml (mean, 225 ± 45.8 pg/ml). Mild functional mitral regurgitation was observed in 88% of the patients with HFPEF and LAVI exceeding 34 ml/m².

The most common clinical symptoms of HF were dyspnea and reduced exercise tolerance, which were observed in 89% of the patients, of whom about 78% were classified as NYHA class II and 11%—as NYHA class III.

The independent predictors of HFPEF in the multivariate regression model for the whole study group are presented in TABLE 2.

HFPEF was more common in patients with type 2 diabetes (76% of the patients in comparison with 24% of nondiabetic patients; *P* = 0.01). Diabetic patients, apart from diet, received pharmacological treatment. Metformin as the only hypoglycemic agent was administered in 52% of the patients. Sulfonylurea derivatives were used by 48% of the patients, while insulin treatment in combination with metformin—in 42%. The most common insulin therapy was the 2-injection regimen. The differences between patients with and without diabetes are presented in TABLE 3.

The analysis of diastolic function parameters showed a positive correlation (both for women and men) between BNP levels and age (*r* = 0.55; *P* <0.01), DT (*r* = 0.53; *P* <0.01), E/e'(*r* = 0.48; *P* <0.01), systolic blood pressure of 140 mmHg or higher (*r* = 0.41; *P* <0.01), LAVI (*r* = 0.17; *P* <0.01), LV mass (*r* = 0.67; *P* <0.0001), and an insulin dose exceeding 80 units/day in diabetic patients (*r* = 0.44; *P* <0.01) as well as a negative correlation

TABLE 3 Characteristics of patients with and without diabetes

Variable	Patients with diabetes (n = 140)	Patients without diabetes (n = 70)	*P* value
age, y	60.82 ±4.86	56.15 ±5.56	<0.01
BMI, kg/m²	30.68 ±3.67	29.00 ±3.82	<0.01
hypertension	140 (100)	70 (100)	1.0
SBP, mmHg	139.83 ±17.87	133.78 ±16.95	<0.01
DBP, mmHg	85.17 ±9.55	77.74 ±10.77	<0.01
hyperlipidemia	100 (71.4)	31 (44.2)	<0.01
GFR, ml/min/1.73 m²	80.06 ±24.80	87.60 ±18.56	0.03
GFR <60 ml/min/1.73 m²	60 (43)	14 (20)	0.01
coronary artery disease	72 (51)	25 (36)	0.04
history of myocardial infarction	23 (16)	10 (14)	0.8
history of stroke	6 (4)	6 (9)	0.3
smoking	30 (21)	13 (19)	0.8
ACEIs	132 (94)	53 (76)	0.01
diuretics	123 (88)	33 (47)	<0.01
calcium channel blockers	102 (73)	44 (63)	0.18
β-blockers	129 (92)	57 (81)	0.03
aspirin	89 (64)	49 (70)	0.4
angiotensin receptor blockers	2 (1.4)	15 (21)	<0.01
statins	135 (96)	59 (84)	<0.01
fibrates	4 (3.0)	2 (3)	0.7

Data are presented as mean ± standard deviation or number (percentage) of patients.

Abbreviations: ACEI, angiotensin-converting enzyme inhibitor; BMI, body mass index; DBP, diastolic blood pressure; others, see **TABLE 2**

TABLE 4 Independent predictors of heart failure with preserved ejection fraction in patients with type 2 diabetes and impaired left ventricular relaxation in a multivariate regression model

Variable	95% CI	OR	*P* value
age, y	1.10–1.28	1.18	<0.01
SBP >140 mmHg	4.56–24.8	10.66	<0.01
coronary artery disease	2.22–12.40	5.25	<0.01
GFR <60 ml/min/1.73 m²	0.93–0.97	0.95	<0.01
increased LV mass, g/m²	1.37–8.41	3.40	0.01

Abbreviations: see **TABLES 1** and **2**

with E/A (r = –0.49; P <0.01) and body mass index (r = –0.50; P = 0.01).

The independent predictors of HFPEF in the multivariate regression model in patients with diabetes are shown in **TABLE 4**.

DISCUSSION Studies published so far have indicated that, in patients with isolated diastolic dysfunction, the risk of HF is proportional to the degree of diastolic dysfunction.[19-24] However, in our study, we observed HFPEF in 42% of the patients (mean age, 56.11 ±6.24 years) with hypertension and the mildest form of diastolic dysfunction, namely, impaired relaxation. To the best of our knowledge, our study is the first to demonstrate the presence of HFPEF in patients with impaired relaxation, which was confirmed at the same time by different diagnostic methods. However, we believe that this phenomenon must have already been observed by practicing cardiologists. Our patients not only presented with subjective symptoms of HF (mainly dyspnea and reduced exercise tolerance) but also showed signs of pulmonary congestion, elevated LV end-diastolic pressure on echocardiography, and increased BNP levels.

Numerous studies have shown an association between an increase in the incidence of isolated HFPEF and aging.[25-27] However, our study revealed that HFPEF may also develop in younger patients because the mean age of our subjects at diagnosis was 59 years, while the youngest patient was 45 years old. This indicates that the treatment of hypertension and related comorbidities may be insufficient, and physicians should focus on developing methods of assessing treatment efficacy individually in each patient.

It is well known that older age, hypertension, and CAD are common causes of HFPEF. In our study, all patients with impaired LV relaxation had hypertension and, additionally, some of them had CAD and diabetes. In most patients, hypotensive therapy was effective, but in patients with HFPEF, mean blood pressure values were higher than in those without HFPEF. The role of chronic heart ischemia as the cause of HFPEF was confirmed in other clinical studies even without concomitant impaired contractility.[8] In our study, CAD was an independent predictor of HFPEF.

In our study, reduced GFR was also an independent predictor of HFPEF. Lower GFR values were observed more often in diabetic patients compared with nondiabetic ones (P = 0.01). The association between reduced GFR and increased severity of HF symptoms as well as increased incidence of HFPEF and HFREF has also been reported by other authors.[28,29]

We also showed a higher incidence of HFPEF in women, which may be explained by the fact that they were older and more often had obesity, renal failure, type 2 diabetes, higher blood pressure, and greater LV hypertrophy, all of which are known risk factors for HFPEF. In addition, most women in our study were postmenopausal, which might have been an additional risk factor for the development of HFPEF because of reduced vascular and heart muscle compliance due to lower estrogen levels.[30-32]

Another independent predictor of HFPEF is type 2 diabetes. In our study, diabetic patients constituted 68% of the study group. The etiology of HFPEF in patients with type 2 diabetes is complex. The available data indicate, among others, the role of chronic hyperglycemia, which may lead to cardiac fibrosis and secondary reduction in LV compliance. This in turn may manifest itself in the early stages only during exertion.[32-34] However, in our study, patients with diabetes had a wide range of other possible causes of HFPEF

apart from hyperglycemia, such as hypertension, obesity, CAD, LV hypertrophy, and impaired renal function, which makes it difficult to assess the role of abnormal glucose metabolism alone in the development of HFPEF. This might have been possible if the study included also patients with type 2 diabetes and no comorbidities. The lack of such patient subgroup in our study may be considered as one of the limitations; however, it was very difficult to identify such patients in clinical practice. In addition, in our study, all patients with diabetes were older, had greater LV hypertrophy, higher body mass index, and higher blood pressure despite a more frequent use of antihypertensive therapy, compared with nondiabetic patients. They also had a lower GFR and higher incidence of CAD. All these factors increased the risk of HFPEF in comparison with nondiabetic subjects in our study population.

Although our multivariate regression analysis did not show insulin therapy to be an independent predictor of HFPEF, we observed a positive correlation between an insulin dose (>80 units/day) and BNP levels. In particular, high levels of endogenous insulin in obese patients with type 2 diabetes who received insulin treatment may be one of the risk factors for HFPEF. Based on pathophysiological data, we believe that high insulin levels related to insulin resistance in type 2 diabetes in combination with exogenous insulin lead to hypercatecholaminemia, activation of the renin–angiotensin–aldosterone system, an increase in peripheral resistance, and heart overload. In turn, heart overload results in diastolic dysfunction and HFPEF. The relationship between chronic heart overload in diabetic patients treated with insulin and diastolic dysfunction was also discussed in a study by Konduracka et al.[33]

Recent data have shown that BNP levels are increased in patients with HF, which is especially important in the differential diagnosis of dyspnea.[35] The degree of an increase in BNP levels was examined in different types of HF in a number of studies, and it is known that HFREF increases plasma BNP concentrations to a greater extent than HFPEF does.[35-37] In our study, mean BNP levels in patients with symptoms of HFPEF were 225 pg/ml, whereas in a study by Lubien et al,[37] mean BNP levels in patients with impaired relaxation and dyspnea were slightly higher than those in our study (about 310 pg/ml), which was probably related to older age of those patients compared with our population. On the other hand, in a review article, Dahlström[38] stated that natriuretic peptides are not activated in patients with diastolic dysfunction in the form of impaired relaxation. Additionally, in our study, increased BNP levels correlated positively with echocardiographic markers of increased LV end-diastolic pressure, which, in combination with clinical presentation, proves the usefulness of this marker in the diagnosis of chronic HFPEF.

The presence of isolated HFPEF in a substantial number of patients with impaired relaxation indicates that this stage of diastolic dysfunction is not necessarily as mild as is commonly believed. It is important to intensify the treatment of any conditions leading to HF before any echocardiographic signs of diastolic dysfunction are observed. Not only the classification grades but also some diagnostic markers of HFPEF may vary between individual patients. Sometimes, the parameters are conflicting and there are variations between studies depending on the study population. In a recent study, Kuwaki et al[39] investigated a group of patients in whom diastolic dysfunction could not be graded according to the standard classification on the basis of echocardiography, similarly to our patients.These patients had an E/A ratio of 0.75 or higher, DT exceeding 140 ms, and an E/e' ratio of 10 or higher, so despite having mild diastolic dysfunction, they had a high probability of increased LV end-diastolic pressure. The authors postulated that a new degree of diastolic dysfunction should be recognized and emphasized that these patients have a serious prognosis with the risk of serious cardiovascular events and that such cases are not uncommon in clinical practice.

In conclusion, isolated impaired LV relaxation may be accompanied by HFPEF; therefore, the issue of whether this stage of diastolic dysfunction is relatively mild should be readdressed. Increased BNP levels in symptomatic patients with impaired relaxation prove the usefulness of this marker in the diagnosis of chronic HFPEF. In addition, because the modifiable predictors of HFPEF in patients with impaired LV relaxation include systolic blood pressure of 140 mmHg or higher, obesity, increased LV mass, impaired renal function (GFR <60 ml/min/1.73 m^2), CAD, and type 2 diabetes, prevention of all those possible causes of HF is necessary (mainly through lifestyle modification). Moreover, in patients who already suffer from those conditions, it is necessary to intensify the treatment so that the onset of diastolic dysfunction is delayed. In diabetic patients, a positive correlation between higher doses of insulin (>80 units/day) and BNP levels requires further research.

Contribution statement HB conceived the idea for the study. HB and EK contributed to the design of the research. HB was involved in data collection. HB, EK, and GG analyzed the data. All authors edited and approved the final version of the manuscript.

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ARTYKUŁ ORYGINALNY

Występowanie przewlekłej niewydolności serca z zachowaną frakcją wyrzutową mięśnia lewej komory u osób z nadciśnieniem tętniczym i izolowaną łagodną postacią dysfunkcji rozkurczowej

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SŁOWA KLUCZOWE

niewydolność serca z zachowaną frakcją wyrzutową, zaburzenia relaksacji lewej komory

STRESZCZENIE

WPROWADZENIE Niewydolność serca z zachowaną frakcją wyrzutową (*heart failure with preserved ejection fraction* – HFPEF) pozostaje wyzwaniem w praktyce klinicznej. Rokowanie pacjentów z HFPEF jest podobne lub tylko nieznacznie lepsze w porównaniu do pacjentów z niewydolnością serca z obniżoną frakcją wyrzutową (*heart failure with reduced ejection fraction* – HFREF). Zaburzenia relaksacji są najłagodniejszą postacią dysfunkcji rozkurczowej, której nie powinny towarzyszyć objawy HFPEF.

CELE Celem badania była ocena występowania przewlekłej HFPEF u osób z nadciśnieniem tętniczym i izolowaną łagodną postacią dysfunkcji rozkurczowej.

PACJENCI I METODY W badaniu przekrojowym wzięło udział 210 pacjentów (średnia wieku 56,11 ±6,24 roku; 58% kobiet) z izolowanymi zaburzeniami relaksacji mięśnia lewej komory i nadciśnieniem tętniczym. Dodatkowo w badanej grupie wyodrębniono chorych z cukrzycą typu 2 w celu porównania częstości występowania HFPEF z pacjentami bez cukrzycy. HFPEF była rozpoznawana, kiedy kliniczne objawy niewydolności serca współwystępowały z echokardiograficznymi wskaźnikami podwyższonego ciśnienia rozkurczowego lewej komory, z cechami zastoju w krążeniu płucnym w RTG klatki piersiowej lub ze zwiększonym stężeniem peptydu natriuretycznego (*brain natriuretic peptide* – BNP).

WYNIKI HFPEF rozpoznano u 42% badanych z zaburzeniami relaksacji. Podwyższony indeks objętości lewego przedsionka (>34 ml/m²) stwierdzono u 38% badanych, wskaźnik E/e' >8 – u 37%, zwiększone stężenie BNP – u 39%, natomiast cechy zastoju w krążeniu płucnym w RTG klatki piersiowej – u 41%. Niezależnymi predykatorami HFPEF były: wiek, skurczowe ciśnienie tętnicze krwi ≥140 mm Hg, cukrzyca typu 2, choroba wieńcowa oraz oszacowana wielkość przesączania kłębuszkowego <60 ml/min/1,73 m². U pacjentów z cukrzycą wykazano dodatnią korelację między dawką insuliny >80 j./d a stężeniem BNP.

WNIOSKI Pacjenci z izolowanymi zaburzeniami relaksacji są niejednorodną klinicznie grupą, gdyż u części z nich występują objawy HFPEF, którym towarzyszy zwiększenie stężenia BNP. Dlatego należy zrewidować czy ten stopień dysfunkcji rozkurczowej rzeczywiście jest łagodny oraz podkreślić, że ci pacjenci mają poważne rokowanie z ryzykiem rozwoju niewydolności serca. U pacjentów z cukrzycą dodatnia korelacja pomiędzy wysoką dawką insuliny a stężeniem BNP wymaga dalszych badań.

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