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The prognostic role of exercise echocardiography in heart failure

Paweł Rubiś, Leszek Drabik, Grzegorz Kopeć, Maria Olszowska, Wojciech Płazak, Piotr Podolec

Department of Cardiac and Vascular Disease, John Paul II Hospital, Institute of Cardiology, Faculty of Medicine, Jagiellonian University, Krakow, Poland

Abstract

Background: Gradual impairment of exercise tolerance is the commonest sign of heart failure (HF). Little is known as to which cardiac contributors of poor exercise capacity carry an independent prognostic information in HF.

Aim: We investigated the prognostic role of exercise echocardiography (ex-echo) in HF patients.

Methods: We studied 85 consecutive, symptomatic HF patients (66 males, mean age 62.5 ± 11.8 [range 21–83] years, mean left ventricular ejection fraction [LVEF] 27.2 ± 9.5%). The end-point was all-cause mortality. During the follow-up period (mean 43 ± 21 months) 21 patients died. Resting echocardiography and ex-echo, with the simultaneous measurement of peak oxygen uptake (VO_{2peak}), was performed in each patient using a semi-supine ergometer (20 W, 2-min increments). Apart from conventional assessment of systolic and diastolic function (EF, E/A, DT, IVRT) or right ventricular systolic pressure (RVSP), tissue Doppler imaging was used for the assessment of LV and RV peak velocity (IVV) as well as acceleration during isovolumic contraction (IVA), peak velocity during ejection phase (S'), peak early diastolic velocity (E'), peak late diastolic velocity (A'), and ratio of early diastolic mitral/tricuspid velocity to peak early diastolic velocity (E/E').

Results: Patients who died were significantly older, had lower exercise capacity, more advanced HF, greater impairment of baseline systolic function, higher baseline pulmonary artery systolic pressure, and most importantly a lack of improvement in EF, diastolic function, and further increase of RVSP during exercise. Out of all echocardiographic parameters, only peak stress EF (χ^2 6.1; $p = 0.01$), baseline and peak exercise RVSP (χ^2 12.5 and χ^2 18.7; $p < 0.001$; respectively), and mitral E/E' ratio (χ^2 8.9; $p < 0.01$) were univariate predictors of prognosis and remained independently prognostic when adjusted for age and sex but were eliminated from the model by NT-proBNP.

Conclusions: During exercise, more severe systolic and diastolic dysfunction with the elevation of pulmonary arterial pressure is more prevalent in HF patients who have a poorer outcome. The estimation of common parameters such as EF, RVSP and E/E' using ex-echo, provides prognostic information in HF.

Key words: heart failure, stress echocardiography, prognosis, exercise

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INTRODUCTION

Despite enormous progress in pharmacotherapy and electrotherapy, the individual outlook for subjects with systolic heart failure (HF) is still bleak [1]. Prognostic assessment of patients with overt HF plays a major role in holistic and up-to-date approaches to this complex syndrome. A great number of variables have been associated with poor outcome, of which a few have been tested and validated using robust prognostic models [2].

By the nature of HF, gradual impairment of exercise tolerance is the commonest sign of the disease, and often the primary reason why patients seek medical attention [1]. The pathology of poor functional tolerance in HF is complex and the mechanisms responsible can be briefly

Address for correspondence:
Pawel Rubiś, MD, PhD, Department of Cardiac and Vascular Disease, John Paul II Hospital, Institute of Cardiology, Faculty of Medicine, Jagiellonian University, ul. Prądnicka 80, 31–202 Kraków, Poland, e-mail: pawelrub@poczta.onet.pl, rubispp@gmail.com
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divided into two types: cardiac (central) and non-cardiac (peripheral) [3]. Lack of augmentation of left ventricle (LV) contractility, worsening of LV compliance, increased functional mitral regurgitation or increased pulmonary hypertension are typical cardiac contributors of early termination of exercise.

However, other mechanisms, such as neuro-hormonal imbalance, ergo-reflex activation, breathing pattern or adverse changes in peripheral muscles have all been shown to affect exercise in HF [4]. There have been numerous theories put forward to explain the reduction of exercise capacity in HF; yet still little is known as to whether the aforementioned cardiac contributors of poor exercise capacity assessed during exercise carry any independent prognostic information in HF. Therefore, the main objective of this study was to establish the prognostic role of cardiac functional parameters assessed during stress, in patients with HF.

METHODS

Study population

This study constitutes an outcome analysis of our project on exercise echocardiography in systolic HF [5]. Briefly, we included patients with a confirmed diagnosis of HF based on the criteria proposed by the European Society of Cardiology, sinus rhythm, no more than trivial mitral and aortic secondary heart valve disease and an ability to perform an exercise test on a bicycle [5]. All patients had chronic, stable, symptomatic HF (NYHA class I to III). Clinical stability was defined as no hospitalisations, no symptomatic worsening and no change in HF medications for at least three months prior to the study. Patients with any unstable condition, including acute coronary syndrome, were excluded from the trial. The long-term follow-up was obtained via chart review and telephone contact and was complete for the 85 patients who constituted the study group. The status of all patients was known and documented up to and including the census date of 1 September 2009. The end-point of the study was all-cause mortality. The study was approved by our local Ethics Committee and all participants gave their written informed consent.

Baseline and stress echocardiography

All echocardiographic examinations were recorded on commercially available equipment (Vivid 7 GE Medical System, Horten, Norway) with a phased-array 3.5-MHz transducer and tissue Doppler imaging (TDI) software. For the purpose of stress echocardiography, we utilised a semi-supine ergometer (Ergoline 9000 Ergoline GmbH, Bitz, Germany) according to a protocol previously described (increasing the workload by 20 watt increments at two minute intervals) [6]. Each variable represents an average of 3–5 cardiac cycles. Pharmacotherapy was continued without change at the time of the stress tests.

The conventional M-mode, B-mode and Doppler parameters were measured according to the current Polish and American guidelines [7, 8]. Left ventricular ejection fraction (LVEF) was calculated using a Simpson biplane method of discs [8]. As a surrogate for pulmonary artery systolic pressure, we used right ventricular systolic pressure (RVSP) after confirming the absence of any obstruction to flow between the RV and pulmonary artery [9, 10]. The RVSP was measured using the simplified Bernoulli equation.

Tissue Doppler indices were also measured both at rest and during stress. Real-time colour Doppler myocardial velocities were acquired as data superimposed on an underlying two-dimensional grey-scale image at a frame rate > 150/s and sector angle of 60 degrees. Both LV and RV were sampled in the apical four-chamber (A4Ch) standard projection, where a 5 × 5 mm sample volume was positioned at the lateral border of the tricuspid annulus for RV interrogation, and at the septal and lateral borders of the mitral annulus for LV interrogation. The following systolic and diastolic myocardial tissue Doppler signals from LV and RV were measured: peak velocity (IVV) and acceleration during isovolumic contraction (IVA) and peak velocity during ejection phase (S′); peak early diastolic velocity (E′); and peak late diastolic velocity (A′). Additionally, we non-invasively estimated the filling pressure of LV and RV, as a ratio of early diastolic mitral/tricuspid velocity to peak early diastolic myocardial velocity (sampled from the mitral or tricuspid annulus), namely: E/E′ ratio. The IVA was calculated, as previously described by Vogel et al. [11], as a peak velocity during isovolumic contraction divided by the acceleration time, necessary to achieve peak velocity.

Cardiopulmonary exercise test

Alongside exercise echocardiography, we performed the cardiopulmonary exercise test using a ZAN Messgeraete GmbH Ferraris cardiorespiratory instrument (Oberthulba, Germany), with continuous measurements of peak oxygen uptake (VO_2peak), carbon dioxide production and one-minute ventilation. All study protocols and measurements were in accordance with the guidelines of the American Thoracic Society and the American College of Chest Physicians [12].

Statistical analysis

Continuous variables are presented as a mean (standard deviation — SD) when normally distributed, or as median (inter-quartile range — IQR) if not, and discrete variables are presented as frequency counts and percentages. The Kolmogorov-Smirnov test was used to verify the normality of distribution of continuous variables. Comparisons were conducted using the unpaired Student's t-test for normally distributed variables and the Mann-Whitney U-test for non-normally distributed data and χ^2 test. Cox proportional hazard models were constructed to explore the relationship

between variables and outcome. If significant, these variables were then tested in a forward, stepwise multiple Cox regression survival model to determine independent predictors of death. Results of these analyses were summarised as hazard ratios (HR) with 95% confidence intervals (CI) and associated log-likelihood ratio χ^2 and p values. Differences were considered significant when a p value was < 0.05. The analyses were carried out using STATISTICA software (StatSoft, Inc., version 6, 2001).

RESULTS

Eighty eight consecutive and eligible HF patients, referred to our echo laboratory over a period of 12 months, were enrolled in the study. Three patients were lost from follow-up, and therefore are not included in this analysis.

A summary of the characteristics of the 85 patients of the total study group, and a comparison of the baseline characteristics of the patients who died (n = 21) with those who survived (n = 64) is shown in Table 1. The study group was treated according to contemporary guidelines, with ACE-inhibitor and beta-blocker usage at 94% and 92%, respectively. During the follow-up of 43 ± 21 months (IQR 35–49 months; minimum 33 and maximum 51 months), 21 (25%) patients died. Patients who died were significantly older and had worse HF at baseline, as shown by a higher NYHA class, N-terminal prohormone of brain natriuretic peptide (NT-proBNP) value and worse LV function. Ischaemic heart disease was more prevalent in non-survivors. Haemoglobin, sodium and glomerular filtration rate were higher in the survivors. The use of ACE-I/ARB/spironolacton was similar in both groups, but patients with favourable outcomes more often received beta-blockers and less often loop diuretic. Patients who died during follow-up exercised significantly less and consequently achieved lower peak oxygen uptake.

The comparison of echocardiographic parameters is depicted in Table 2. All the participants performed the stress echo without chest discomfort, significant ST changes, or echocardiographic evidence of inducible ischaemia. The primary reason for exercise termination was dyspnoea on exertion and fatigue. As already mentioned, baseline LVEF was significan-

Table 1. Baseline clinical characteristics

Parameters	Total cohort (n = 85)	Non-survivors (n = 21)	Survivos (n = 64)	P
Age [years]	62.5 ± 11.8	67.4 ± 10.7	61.3 ± 12.6	0.05
Women/men [%]	19 (22%)/66 (78%)	5 (24%)/16 (76%)	14 (22%)/50 (78%)	0.85
BMI [kg/m^2]	28.6 ± 4.9	27.9 ± 4.4	28.9 ± 5.3	0.18
NYHA class	1.94 ± 0.8	2.7 ± 0.47	1.81 ± 0.77	< 0.001
ICM/DCM [%]	53 (62%)/32 (38%)	17 (81%)/4 (19%)	36 (56%)/28 (44%)	0.04
Hypertension [%]	49 (57.6%)	13 (62%)	36 (56%)	0.65
Diabetes mellitus [%]	19 (22%)	5 (24%)	14 (22%)	0.85
Dyslipidemia [%]	52 (61%)	15 (71%)	37 (58%)	0.27
Haemoglobin [g/dL]	13.2 ± 1.8	11.6 ± 1.9	14.3 ± 2.7	< 0.001
GFR [mL/min]	80.3 ± 26.7	73.7 ± 21.6	89.5 ± 19.3	0.005
NT-proBNP [pg/mL]	402.9 ± 206.5	444.6 ± 146.4	251.3 ± 80.7	< 0.001
Sodium [mmol/L]	137.8 ± 11.4	135.3 ± 9.3	139.4 ± 8.5	< 0.001
Beta-blocker [%]	78 (92%)	17 (81%)	61 (95%)	0.05
ACE-I/ARBs [%]	80 (94%)	18 (85%)	62 (97%)	0.06
Loop diuretic [%]	51 (60%)	18 (86%)	33 (52%)	0.006
Spironolacton [%]	68 (80%)	15 (71%)	53 (83%)	0.26
Time of stress [s]	415.9 ± 207.6	311.5 ± 107.8	459.5 ± 228.1	0.02
Load [Watts]	82.6 ± 41.4	57.7 ± 11.1	92.9 ± 52.4	0.04
HR rest [beats/min]	66.3 ± 12.4	69.2 ± 15.7	64.6 ± 13.8	0.21
HR stress [beats/min]	112.7 ± 20.4	102.6 ± 31.4	118.4 ± 27.2	0.14
LBBB [%]	22 (26%)	6 (30%)	14 (22%)	0.5
$VO_{2\ peak}$ [mL/kg/min]	15.2 ± 4.9	11.9 ± 2.4	15.6 ± 4.7	0.01
VE/CO_2	34.8 ± 7.2	39.3 ± 7.2	30.5 ± 8.7	0.01

BMI — body mass index; NYHA — New York Heart Association; ICM — ischaemic cardiomyopathy; DCM — dilated cardiomyopathy; GFR — glomerular filtration rate (Cockcroft-Gault equation); NT-proBNP — N-terminal prohormone of brain natriuretic peptide; ACE-I — angiotensin converting enzyme inhibitor; ARB — angiotensin receptor blocker; HR — heart rate; LBBB — left bundle branch block; VO_2 peak — peak oxygen uptake; VE/CO_2 — ventilatory equivalent for carbon dioxide

Table 2. Rest and peak stress echocardiographic parameters

Parameters	Total cohort (n = 85)	Non-survivors (n = 21)	Survivos (n = 64)	P
$LVEDV_{rest}$ [mL]	177.6 ± 56.4	197 ± 67.7	167.5 ± 72.5	0.08
$LVESV_{rest}$ [mL]	133.6 ± 63.8	143.9 ± 53.2	123.7 ± 61.5	0.05
$LVEF_{rest}$ [%]	27.2 ± 9.5	21.7 ± 7.6	28.3 ± 9.4	0.05
$LVEF_{stress}$ [%]	30.4 ± 13.3	23.4 ± 7.9	33.2 ± 13.4	0.001
$LV\text{-}IVV_{rest}$ [cm/s]	3.8 ± 1.6	3.7 ± 1.7	3.8 ± 1.6	0.78
$LV\text{-}IVV_{stress}$ [cm/s]	4.6 ± 1.6	4.1 ± 1.3	4.7 ± 1.7	0.17
$LV\text{-}IVA_{rest}$ [m/s^2]	1.3 ± 0.7	1.1 ± 0.6	1.3 ± 0.7	0.32
$LV\text{-}IVA_{stress}$ [m/s^2]	2.1 ± 1.1	1.8 ± 0.8	2.2 ± 1.07	0.37
$LV\text{-}S'_{rest}$ [cm/s]	4.6 ± 1.6	4.3 ± 2	4.7 ± 1.6	0.49
$LV\text{-}S'_{stress}$ [cm/s]	5.7 ± 1.9	4.9 ± 1.8	5.8 ± 2.1	0.05
$LV\text{-}E/E'_{rest}$	15.2 ± 6.4	16.3 ± 6.5	15 ± 6.4	0.64
$LV\text{-}E/E'_{stress}$	18 ± 6.4	21.4 ± 3.1	17.5 ± 5.6	0.02
$RV\text{-}S'_{rest}$ [cm/s]	9.3 ± 3.5	9.2 ± 3.3	9.4 ± 3.4	0.94
$RV\text{-}S'_{stress}$ [cm/s]	11.3 ± 4.8	9.5 ± 4.2	11.7 ± 4.8	0.21
$RV\text{-}E/E'_{rest}$	6.5 ± 3.3	7.6 ± 4.1	6.3 ± 3.1	0.21
$RV\text{-}E/E'_{stress}$	7.1 ± 4.1	7.8 ± 3.2	6.9 ± 4.2	0.45
$RVSP_{rest}$ [mm Hg]	28.2 ± 19.5	45.4 ± 23.9	25.3 ± 17.3	0.003
$RVSP_{stress}$ [mm Hg]	36.3 ± 17.4	56.8 ± 18.6	31.4 ± 21.4	0.001

LVEDV — left ventricular end-diastolic volume; LVESV — left ventricular end-systolic volume; LVEF — left ventricular ejection fraction; LV-S′ — left ventricular peak velocity during ejection phase; LV-E/E′ — left ventricular ratio of early diastolic mitral velocity to peak early diastolic myocardial velocity; RVSP — right ventricular systolic pressure; IVV — peak velocity during isovolumic contraction from lateral border of tricuspid annulus; IVA — myocardial acceleration during isovolumic contraction; S′ — peak velocity during ejection phase; E/E′ — ratio of early diastolic tricuspid velocity to peak early diastolic myocardial velocity

tly lower in non-survivors; more importantly, this difference even widened at peak stress. Patients who died had higher values of both rest and peak stress RVSP, which may indicate more prevalent secondary pulmonary hypertension in this group. Surprisingly, among all TDI indices, only two parameters from LV, both measured during stress, differed significantly between the groups. The high reproducibility of the echocardiographic data has been previously tested and reported by our study group [5, 13].

Univariate predictors of mortality are shown in Table 3. The NT-proBNP was the strongest univariate predictor, followed by VO_{2peak} and age. Of the 12 variables that were significant in the univariate analysis, only four remained so in the multivariable model, and again NT-proBNP remained the strongest independent predictor of mortality (Table 4). Peak exercise LVEF and both rest and peak stress RVSP remained independently prognostic, when adjusted only for age and sex, but when NT-proBNP was incorporated into the multivariable model, all of them lost all statistical significance.

DISCUSSION

Although establishing prognosis on an individual basis may often be problematic, estimating outcomes in HF is important and can be routinely conducted using a wide selection of prognostic models. Nevertheless, debate still exists as to how to choose the most informative predictors of prognosis and the best prognostic model. Furthermore, it is unknown whether echocardiographic parameters assessed during stress provide independent and incremental prognostic information. Over the last three decades, a sizable number of survival analyses have been performed in a wide selection of HF patients. Many of the previously powerful models are plainly out of date since they do not include modern neuro-hormonal pharmacotherapy, nor very strong predictors such as natriuretic peptides. Therefore, any up-to-date survival model in HF should be validated and adjusted for these factors; otherwise its value may be questionable [14].

In our study, we endeavoured to ascertain whether adding echocardiographic functional parameters, measured during stress tests, would improve the predictive power of survival models in HF. The main findings of our study are somewhat disappointing, as all analysed cardiac functional parameters, measured either at rest or peak stress, failed to be independent predictors of outcome. Only four parameters, namely baseline and stress RVSP, and LV-E/E′ and LVEF both at peak stress, were significant in the univariate model but this significance vanished when adjusted for the NT-proBNP in the multivariable analysis.

Our study is a helpful contribution to the discussion concerning the role of echocardiographic parameters in the con-

Table 3. Univariate predictors of outcome

Parameters	χ^2	P	HR	95% CI
NT-proBNP [pg/mL]	117.6	< 0.001	3.5	2.8–3.9
$VO_{2\ peak}$ [mL/kg/min]	73.8	< 0.001	1.6	1.3–1.8
Age [years]	71.5	< 0.001	1.1	0.97–1.2
Loop diuretic	31.8	< 0.001	2.3	2–2.6
Sodium [mmol/L]	30.3	< 0.001	0.95	0.93–0.98
Haemoglobin [g/dL]	27.9	< 0.001	0.89	0.84–0.91
NYHA class	21.7	< 0.001	1.5	1.3–1.8
$RVSP_{stress}$ [mm Hg]	18.7	< 0.001	1.3	1.1–1.7
ICM	17.5	< 0.001	1.36	1.09–1.48
$RVSP_{rest}$ [mm Hg]	12.5	< 0.001	0.97	0.95–1
$LV\text{-}E/E'_{stress}$	8.9	< 0.01	1.6	1.3–1.8
$LVEF_{stress}$ [%]	6.1	0.01	1.2	1–1.5

Abbreviations as in Tables 1 and 2

Table 4. Multivariate independent predictors of outcome

Parameters	χ^2	P	HR	95% CI
NT-proBNP [pg/mL]	54.8	< 0.001	2.4	1.7–2.9
$VO_{2\ peak}$ [mL/kg/min]	32.7	< 0.001	1.4	0.9–1.7
Age [years]	27.6	< 0.001	1.32	1.2–1.52
Sodium [mmol/L]	12.6	0.001	0.96	0.94–1.1

Abbreviations as in Tables 1 and 2

text of prognosis in HF. There are studies which have reported that certain echocardiographic variables assessed either during stress or more commonly at rest are independently associated with the outcome [15–18]. On the other hand, some investigations did not support these findings and actually reported a lack of prognostic significance of echocardiography [19, 20]. It is difficult to draw robust conclusions from previous studies, as they greatly differed from each other in terms of studied population, aetiology of HF, pharmacotherapy, mode of stress test, or the utilisation of natriuretic peptides in survival analysis.

In 1997, Aaronson et al. [2] published their hallmark study in which they prospectively validated prognostic models in 268 HF patients referred for cardiac transplant evaluation. This formed a cornerstone for all subsequent survival analysis. One of the strongest predictors was LVEF, alongside VO_{2peak} and serum sodium. Similarly, we confirmed VO_{2peak} and serum sodium to be powerful, independent predictors; but in the case of LVEF, we did not find such a strong relation with the outcome as in the Heart Failure Survival Score. It may be that we included a relatively small number of patients in the analysis. However, the pioneering work of Aaronson et al. [2] was completed in the pre-beta-blocker era, which makes any direct comparisons futile, as beta-blockers greatly affect the outcome in HF. The other issue is that Aaronson et al. [2] did not incorporate natriuretic peptides into survival analysis. Natriuretic peptides, particularly BNP, being the most powerful marker of prognosis in HF, can lead to the situation that even strong predictors may become simply too 'weak' when directly challenged in the multivariable analysis.

Studies by Metra et al. [15] and Marmor and Schneeweiss [16] have reported that peak exercise stroke work index determined invasively during stress and contractile reserve respectively, measured non-invasively during dobutamine infusion, were independent predictors of survival. Similarly, Nagaoka et al. [17] showed that myocardial contractile reserve, measured during exercise radionuclide angiography, can provide independent information on prognosis in mildly symptomatic patients with DCM. Quantitative assessment of functional mitral regurgitation during exercise also proved valuable for the determination of the outcome, as elegantly showed by Lebrun et al. [18].

However, there have also been some 'negative' studies. Our findings are in agreement with the results of Griffin et al. [19] who reported a lack of independent prognostic power

of common echocardiographic parameters, including LVEF measured both at rest and during stress in HF. The conclusions of our study are also in line with the trial by Ennezat et al. [20], who reported that only resting LV end-diastolic volume and tricuspid annular plane systolic excursion were independent predictors of death in multivariable analysis, and that not a single echocardiographic parameter measured during exercise added any new prognostic information beyond that already provided by resting measurements.

Pulmonary hypertension is a well-known negative predictor in HF [21]. The increase of pulmonary artery systolic pressure during exercise was the strongest echocardiographic factor in our univariate analysis, which is in line with some previous trials [21, 22]. Nevertheless, once again, it did not withstand the predictive power of BNP. Similarly, LV diastolic dysfunction and elevated LV filling pressure are strong markers of disease severity and important contributors of low exercise capacity in HF [23]. Lack of improvement of LV filling during exercise was associated with worse survival, as determined non-invasively by mitral E/E' ratio, which disproportionally rose only in patients who died, in contrast to those with better outcomes.

Probably another important finding of our study is the fact that we did not observe any advantage of TDI parameters over 'classical' echocardiographic indices such as EF or RVSP in the determination of prognosis. Even novel, less popular indices of isovolumic contraction (IVV and IVA), measured either at rest or peak stress, turned out to be insignificant in terms of prognosis in our patients. This was clearly not an anticipated result, as many trials have already confirmed the value of TDI parameters in different clinical settings. By way of explanation, it may be that even though TDI parameters such as S' or E' are generally perceived as indices of global cardiac function, in the context of ischaemic cardiomyopathy and regional rather than global functional impairment, single measurements from a few cardiac sites may not fully reflect the true impairment. As our study population was too small to perform a separate survival analysis for dilated or ischaemic cardiomyopathy, we cannot say whether TDI indices would have been of prognostic value in cases of DCM. At this stage, we can only speculate that if the global systolic function is severely reduced, as it was in a great number of our patients, this fact has a more pronounced effect on the outcome than the impact of the relatively preserved regional contraction in remote areas of the LV.

Surprisingly, myocardial velocities, measured by means of TDI, are frequently detected in non-functional (akinetic) areas of the myocardium because of the tethering effect to the adjacent tissue. To overcome this potential source of error and misinterpretation, novel indices of strain rate and strain have been incorporated. However, the main limitation of wider usage of those indices is the difficulty in obtaining reliable measurements, especially during stress (low quality of scans or too high a noise-signal ratio).

In this trial, we included patients with longlasting, stable, optimally treated HF, who were not considered for myocardial revascularisation, even if they had ischaemic cardiomyopathy, as no inducible ischaemia was detected during stress test. Therefore our subjects significantly differed from those individuals with preserved viability, who were subsequently revascularised, and in some instances, their outcome had been greatly improved [24, 25].

Limitations of the study

Our study has a number of limitations, which we acknowledge. Undoubtedly, the relatively small number of patients is a weakness of the study. The other issue concerns the baseline characterisation of the study group, which is at least 10 years younger than a 'real life' HF population. Another limiting factor was the exclusion of patients with severe mitral regurgitation or atrial fibrillation which may jeopardise direct comparisons with the general HF population.

CONCLUSIONS

During exercise, more severe systolic and diastolic dysfunction with elevation of pulmonary arterial pressure is more prevalent in HF patients who have a poorer outcome. Exercise echocardiography, involving the estimation of common parameters such as EF, RVSP and E/E' provides some moderate prognostic information in HF. No advantage for the TDI parameters over commonly used echocardiographic variables such as EF or RVSP has been observed in the determination of prognosis. Larger studies in HF patients are necessary to determine the real prognostic role of 'classical' and novel cardiac functional parameters.

Conflict of interest: *none declared*

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Znaczenie prognostyczne echokardiografii wysiłkowej w niewydolności serca

Paweł Rubiś, Leszek Drabik, Grzegorz Kopeć, Maria Olszowska, Wojciech Płazak, Piotr Podolec

Klinika Chorób Serca i Naczyń, Uniwersytet Jagielloński, *Collegium Medicum*, Krakowski Szpital Specjalistyczny im. Jana Pawła II, Kraków

Streszczenie

Wstęp i cel: Celem pracy była ocena prognostycznego znaczenia echokardiografii wysiłkowej u chorych z przewlekłą niewydolnością serca (CHF).

Metody: Badaniem objęto 85 kolejnych pacjentów z CHF [66 mężczyzn w wieku 62,5 ± 11,8 (21–83) lat i frakcją wyrzutową lewej komory 27,2 ± 9,5%]. Punktem końcowym była śmiertelność całkowita. W okresie obserwacji trwającej średnio 43 ± 21 miesięcy 21 pacjentów zmarło. Spoczynkowe i wysiłkowe badanie echokardiograficzne, z jednoczesnym pomiarem szczytowego zużycia tlenu (VO_{2peak}), wykonano na pół-leżącym ergometrze rowerowym (protokół obciążeń 20 W co 2 min). Oceniono konwencjonalne wskaźniki funkcji skurczowej i rozkurczowej (FW, E/A, DT, IVRT) oraz skurczowe ciśnienie w prawej komorze (RVSP). W lewej i prawej komorze zmierzono także parametry TDI: szczytową prędkość (IVV) i akcelerację w czasie skurczu izowolumetrycznego (IVA), szczytową prędkość w fazie wyrzutowej (S'), wczesną (E') i późną (A') prędkość w fazie rozkurczu oraz stosunek fali wczesnego napływu mitralnego/trójdzielnego do wczesno-rozkurczowej prędkości miokardium (E/E').

Wyniki: Pacjenci, którzy zmarli, byli starsi i stwierdzono u nich niższą wydolność wysiłkową, bardziej zaawansowaną CHF, bardziej upośledzoną spoczynkową funkcję skurczową lewej komory, wyższe spoczynkowe RVSP, brak poprawy FW i funkcji rozkurczowej oraz dalsze narastanie RVSP podczas obciążenia. Spośród wszystkich parametrów echokardiograficznych jedynie FW na szczycie wysiłku (χ^2 6,1; p = 0,01), spoczynkowe i wysiłkowe RVSP (χ^2 12,5 i χ^2 18,7; p < 0,001; odpowiednio) oraz wysiłkowe mitralne E/E' (χ^2 8,9; p < 0,01) były istotnymi predyktorami zgonu w jednoczynnikowej analizie Coxa i pozostały istotne po skorygowaniu względem wieku i płci, natomiast zostały odrzucone w analizie wieloczynnikowej po wprowadzaniu do modelu NT-proBNP.

Wnioski: Podczas wysiłku większe upośledzenie funkcji skurczowej i rozkurczowej oraz wyższe wartości ciśnienia w tętnicy płucnej są częstsze u pacjentów z niekorzystnym rokowaniem. Wysiłkowa ocena typowych parametrów echokardiograficznych: FW, RVSP czy E/E' dostarcza informacji prognostycznych w CHF.

Słowa kluczowe: niewydolność serca, echokardiografia obciążeniowa, prognostyka, wysiłek

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Adres do korespondencji:
dr n. med. Paweł Rubiś, Klinika Chorób Serca i Naczyń, Uniwersytet Jagielloński, *Collegium Medicum*, Krakowski Szpital Specjalistyczny im. Jana Pawła II, ul. Prądnicka 80, 31–202 Kraków, e-mail: pawelrub@poczta.onet.pl, rubispp@gmail.com
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