Prognostic significance of reactive pulmonary hypertension in patients with chronic heart failure listed for elective heart transplantation

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A b s t r a c t

Background: Pulmonary hypertension (PH) is recognised in about 60% of patients referred for heart transplantation (HTx), and it influences the patient's prognosis.

Aim: To assess the prognostic value of reactive PH in patients listed for elective HTx.

Methods: A total of 522 patients were enlisted from the Polish National Registry (POLKARD), listed for HTx, in whom complete haemodynamic data were reported. The endpoint was all-cause death before HTx. Heart transplantation, elective or urgent, was considered as an end of the follow-up (mean follow-up 1.47 ± 1.26 years). Patients were divided into three subgroups: no PH, passive PH, reactive PH taking into account mean pulmonary artery pressure (PAP) of 25 mm Hg, pulmonary capillary wedge pressure (PCWP) 15 mm Hg, and pulmonary vascular resistance (PVR) 3.0 Wood units. Haemodynamic, clinical, echocardiographic, and biochemical assessments (including NT-proBNP, hs-CRP) were performed. The Heart Failure Survival Score (HFSS) was calculated.

Results: Estimated death rate was comparable between patients with no PH, passive PH, and reactive PH, despite the fact that the patients with reactive PH had the worst pulmonary haemodynamic scores (mean PAP 38.4 ± 7.8, PCWP 27 ± 7.4 mm Hg and PVR 4.8 ± 1.7 Wood units). In patients with reactive PH, in multifactor Cox analysis only the serum Na level influenced survival. According to the Kaplan-Meier method, patients with reactive PH, with mean PAP ≥ 41.4 mm Hg had the worst prognosis, estimated survival was: one year — 83%, two years — 65%.

Conclusions: Reactive PH is common among patients with chronic HF listed to HTx (28%). It has no further influence on short-term prognosis as compared with patients with no PH and passive PH. Serum Na concentration and mean PAP ≥ 41.4 mm Hg influence the prognosis of patients with reactive PH.

Key words: heart transplantation, pulmonary hypertension

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INTRODUCTION

Left ventricular (LV) dysfunction and failure causes an increase in LV filling pressure and is responsible for the rise in left atrial pressure and in pulmonary venous and arterial bed. The mean pulmonary artery pressure (mPAP) is closely related to pulmonary capillary wedge pressure (PCWP), left atrial pressure, and with LV filling pressure in patients with LV heart failure (HF). Elevated PCWP can cause remodelling of the pulmonary vascular bed. Arterialisation of pulmonary veins is the first step to reactive pulmonary hypertension (reactive PH) with further structural changes of pulmonary arteries [1–4].

Increased pulmonary vascular resistance (PVR) and increased transpulmonary gradient (TPG) are the result of reactive PH and have an influence on the morbidity and mortality of patients with HF [3–9]. Pulmonary hypertension (PH) is recognised in 50–60% of patients referred for heart transplantation (HTx) and has, according to some authors, a significant influence on patient's prognosis. Contrary to

that, some authors have reported that patients with chronic HF have poor prognosis and that PH has no further influence on prognosis at this stage of disease [9, 10].

Patients with chronic HF listed for HTx are a highly selected population because of the strict qualification/disqualification criteria. PH and elevated PVR are considered as a relative contraindication when there is no response to "vasodilator challenge". Nevertheless, numerous studies have shown that this model of PH (reversible) is associated with worse prognosis [10].

The main aim of the study was to assess the prognostic value of reactive PH (reversible) in patients with chronic HF waiting for HTx in comparison with the rest of the qualified patients (with no PH and with passive PH).

The results could improve allocation criteria to HTx among patients listed for elective transplantation.

METHODS

Within five years (2003–2007) 658 patients with chronic HF according to the United Network for Organ Sharing (UNOS-2) were listed for HTx in Poland and were included into the registry. The average period of observation was 601 days (1–1462 days). All patients underwent standard evaluation including right heart catheterisation [11]. Further analysis concerned only those in whom the complete right heart haemodynamic protocol was reported (522 patients; 79.3%). Apart form the haemodynamic measurements (mPAP, PCWP, TPG, PVR), precise clinical, echocardiographic, electrocardiographic and biochemical assessments (including N-terminal pro B-type natriuretic peptide [NT-proBNP], and high-sensitivity C-reactive protein [hs-CRP]) were performed.

Heart Failure Survival Score (HFSS) according to Aaronson was calculated. The HFSS was derived in each patient from the seven clinical parameters: heart rate (HR), LV ejection fraction (LVEF), serum sodium (Na), peakVO$_2$max, intraventricular conduction delay (IVCD), and ischaemic heart disease (IHD; yes = 1, no = 0). The seven products were summed and the absolute value determined according to the following equation: HFSS = [(0.0216 × resting HR (0.025 × mean blood pressure) + (0.0464 × LVEF) + (0.047 × serum Na) + (0.0546 × peakVO$_2$) + (0.608 × presence or absence of IVCD) + (0.6931 × presence or absence of IHD)]. For the HFSS, risk strata were defined as low risk (≥ 8.10), medium risk (7.20–8.09), or high risk (≤ 7.19), using previously described cut-offs [4].

The whole group (522 patients) was divided into three subgroups: no PH, passive PH, reactive PH according to the Aronson definitions: no PH subgroup — mPAP < 25 mm Hg; passive PH subgroup — mPAP ≥ 25 mm Hg, PCWP ≥ 15 mm Hg, and PVR < 3.0 Wood Units (WU), reactive PH subgroup — mPAP ≥ 25 mm Hg, PCWP ≥ 15 mm Hg, and PVR ≥ 3.0 WU [5].

The endpoint of the study was all-cause death. Heart transplantation, elective or urgent, was considered as an end of the follow-up. All patients were followed up and treated with optimal doses of angiotensin converting enzyme inhibitors, beta-blockers, and diuretics by cardiologists in the Heart Failure and Transplantology Departments in four participating transplantology centres in Poland and appropriate outpatient's clinics.

Informed consent was obtained from each patient participating in the study, according to the protocol approved by the Local Ethics Committee. The investigation conformed to the principles outlined in the Declaration of Helsinki.

The results presented are part of the national POLKARD--HF programme (gov.no.NCT 00690157).

Statistical analysis

All calculations were performed with SAS version 9.2 software. Continuous data are reported as mean ± standard deviation. In the case of a skewed distribution the median with the range is presented. Differences between groups for continuous data were tested with nonparametric analysis of variance by the Kruskal-Wallis test. Differences between groups for proportions were tested with the χ^2 test. Since both cardiac variables (hs-CRP and NT-proBNP) are not normally distributed, these variables were transformed logarithmically to meet the assumptions for linear regression analysis. Cardiac survival was estimated with the Kaplan-Meier method; differences were tested with log rank test — the endpoint of the study was all-cause death, the end of observation (censored) — HTx urgent or elective. Univariable and multivariable Cox-regression analyses were performed to investigate the association of pre-measurements with incidents of death. Independent variables in multivariable analysis were selected by stepwise option.

For all analyses, a two-sided p-value < 0.05 was considered to indicate statistical significance.

RESULTS
Patient's characteristics

A total of 658 patients with stable chronic HF were listed for elective (UNOS 2) HTx and registered in a national registry. In 522 (79.3%), complete haemodynamic data concerning pulmonary circulation were obtained. The main causes of HF were ischaemic cardiomyopathy (46.4%) and dilated cardiomyopathy (47.7%). The average age of patients was 50 years; most of them were male (87.8%). There were patients with chronic HF — 84% of them were in New York Heart Association (NYHA) functional classes III and IV. The mean value of LVEF was 20.7 ± 7.7%. The end-diastolic diameter of LV (LVEDD) was 72.2 ± 10.3 mm. More than 60% of patients had PH — mPAP was 28.5 ± 11.2 mm Hg; PCWP exceeded 20 mm Hg (21.0 ± 9.5), TPG was 10.1 ± 5.6 mm Hg. The mean value of cardiac index (CI) was less than 2.0 liters (1.9 ± 0.9 L/min/m²). NT-proBNP median value was 2703 pg/mL. The Aaronson score (HFSS) indicating medium risk 7.7 ± 0.9 (Table 1). All

Table 1. Characteristics of all the patients

Age [years]	50.0 ± 10.3
LVEF [%]	20.7 ± 7.7
LVEDD [mm]	72.2 ± 10.3
LVESD [mm]	60.9 ± 12.4
HR [/min]	78.4 ± 15.3
SBP [mm Hg]	100.9 ± 14.1
DBP [mm Hg]	66.1 ± 10.4
Na [mEq/L]	136.2 ± 4.4
VO$_2$max [mL/min/kg]	12.2 ± 3.6
HFSS	7.7 ± 0.9
mPAP [mm Hg]	28.5 ± 11.2
PCWP [mm Hg]	21.0 ± 9.5
TPG [mm Hg]	10.1 ± 5.6
Cardiac index [L/min/m^2]	1.9 ± 0.9
PVR [WU]	3.2 ± 2.4
hs-CRP [mg/L]	3.3 (0.03–347.0)
NT-proBNP [pg/mL]	2703 (77–45515)

Data are presented as mean ± standard deviation or median and range (min–max); LVEF — left ventricular ejection fraction; LVEDD — left ventricular end-diastolic diameter; LVESD — left ventricular end-systolic diameter; HR — heart rate; SBP — systolic blood pressure; DBP — diastolic blood pressure; HFSS — Heart Failure Survival Score; mPAP — mean pulmonary artery pressure; PCWP — pulmonary capillary wedge pressure; TPG — transpulmonary gradient; PVR — pulmonary vascular resistance; WU — Wood Unit; hs-CRP — high-sensitivity C-reactive protein; NT-proBNP — N-terminal pro B-type natriuretic peptide
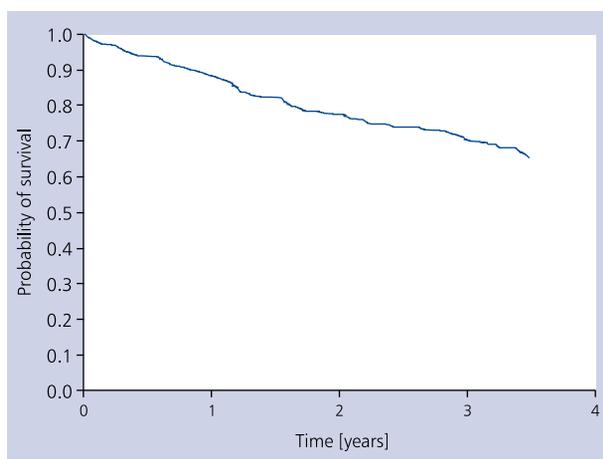
Figure 1. Probability of survival in patients with chronic heart failure — all group

group one-year survival (Kaplan-Meier method) was about 88%, two years — about 77% (Fig. 1).

In total 325 (49.39%) patients were transplanted. Ninety (27.7%) died after HTx. The univariate Cox regression model revealed: end-systolic diameter of LV (LVESD), LVEDD, LVEF, mean PCWP, NT-proBNP, VO$_2$max, HFSS, and serum Na levels influenced the patient's prognosis (Table 2). The multifactor Cox regression model indicated that only LVEDD (HR 1.04; CI 1.010–1.070), VO$_2$max (HR 0.899, CI 0.827–0.978) level, and serum Na (HR 0.937, CI 0.889–0.988) level influenced survival.

The characteristics and prognosis of patients with no PH listed for elective HTx

This was the oldest group as compared with the others — either passive or reactive PH.

The mean value of LVEF (22 ± 8.5%) was higher than LVEF in patients with passive or reactive PH. Systolic and diastolic diameters of LV assessed by echocardiography were comparable with those in the two other groups. The median level of NT-proBNP was the lowest in comparison with the other groups. HFSS was 7.8 ± 0.9. The mean value of: mPAP was 19.9 ± 3.1 mm Hg, PCWP 14.1 ± 3.0 mm Hg, TPG 7.7 ± 3.9 mm Hg, PVR 2.2 ± 1.3 WU, CI 2.2 ± 1.2 L/min (Table 3).

The estimated death rate (Kaplan-Meier method) of patients with HF and no PH was comparable with the death rate of patients with passive or reactive PH (Fig. 2).

The univariate Cox regression model revealed that only NT-proBNP, HFSS, and serum Na levels influenced the patient's prognosis (Table 2). The multifactor Cox regression model indicated that LVEDD (HR 1.048, CI 1.007–1.092), NT-proBNP (HR 1.002, CI 1.001–1.003) level, and serum Na (HR 0.910, CI 0.836-0.991) level influenced survival.

Characteristics and prognosis of patients with passive PH

There were 163 (31%) patients with LVEF: in 20.7 ± 7.3% it was comparable with LVEF in patients with reactive PH. Haemodynamic parameters were as follows: PCWP was 26.6 ± 6.5 mm Hg, mPAP 34.0 ± 6.6 mm Hg, TPG 8.1 ± 3.5 mm Hg, CI 1.9 ± 0.5 L/min, PVR 2.2 ± 0.7 WU. No differences between groups were recorded in terms of LVESD and LVEDD. There were also no differences in the levels of NT-proBNP, VO$_2$max, HFSS, and serum Na level between the groups of patients with passive or reactive PH (Table 3). No differences were found in estimated death rates between patients with no PH or reactive PH (Fig. 2).

Only NT-proBNP and VO$_2$max significantly influenced the patient's prognosis in the univariate Cox regression model (Table 2). Multifactor Cox regression analysis revealed that hs-CRP (HR 1.127, CI 1.037–1.223) and VO$_2$max (HR 0.824, CI 0.713–0.951) influenced the survival of patients with passive PH.

Characteristics and prognosis of patients with reactive PH

This group consisted of 146 (28%) patients. It was the youngest group as compared to others (49.8 ± 10.4 years). Left ventricular dimensions assessed in echocardiography were comparable with respective echo measurements in patients

Table 2. Univariate Cox regression analysis factors influencing survival

	All group		No PH mPAP < 25 mm Hg		Passive PH mPAP ≥ 25 mm Hg PCWP ≥ 15 mm Hg PVR < 3.0 WU		Reactive PH mPAP ≥ 25 mm Hg PCWP ≥ 15 mm Hg PVR ≥ 3.0 WU	
	HR	95% CI	HR	95% CI	HR	95% CI	HR	95% CI
Age [years]	0.998	0.997–1.019	0.998	0.958–1.026	1.003	0.970–1.037	1.004	0.960–1.050
LVEF [%]	0.958	0.927–0.990	0.951	0.903–1.002	0.979	0.929–1.030	0.938	0.872–1.009
LVEDD [mm]	1.031	1.009–1.053	1.026	0.994–1.059	1.009	0.970–1.051	1.060	1.019–1.103
LVESD [mm]	1.025	1.007–1.044	1.024	0.997–1.051	1.006	0.973–1.039	1.050	1.013–1.088
HR [/min]	1.01	0.997–1.023	1.009	0.990–1.028	1.013	0.991–1.035	1.010	0.981–1.039
SBP [mm Hg]	0.988	0.973–1.003	0.996	0.975–1.017	0.994	0.963–1.026	0.965	0.932–0.999
DBP [mm Hg]	0.991	0.972–1.011	1.012	0.985–1.040	0.979	0.939–1.021	0.965	0.926–1.005
Na [mEq/L]	0.922	0.884–0.962	0.91	0.853–0.971	0.927	0.850–1.010	0.927	0.861–0.999
VO$_2$max [mL/min/kg]	0.916	0.861–0.974	0.929	0.844–1.022	0.878	0.777–0.993	0.924	0.823–1.038
HFSS	0.648	0.518–0.811	0.661	0.460–0.951	0.680	0.451–1.024	0.594	0.387–0.913
mPAP [mm Hg]	1.015	0.997–1.033	0.989	0.924–1.059	1.034	0.988–1.083	1.051	1.008–1.097
PCWP [mm Hg]	1.023	1.002–1.044	0.988	0.917–1.064	1.039	0.992–1.088	1.052	1.014–1.091
TPG [mm Hg]	1.000	0.914–1.095	1.021	0.933–1.012	0.981	0.917–1.048	1.006	0.992–1.020
Cardiac index [L/min/ m^2]	0.827	0.551–1.241	1.111	0.764–1.616	0.589	0.274–1.266	0.260	0.078–0.862
PVR [WU]	1.003	0.932–1.079	1.104	0.919–1.326	1.598	0.916–2.786	0.979	0.868–1.105
hs-CRP [mg/L]	1.006	0.989–1.026	1.008	0.977–1.040	1.007	0.973–1.043	1.006	0.989–1.026
NT-proBNP [pg/mL]	1.002	1.001–1.003	1.002	1.001–1.003	1.002	1.001–1.003	1.002	1.001–1.003

CI — confidence interval; HR — hazard ratio; PH — pulmonary hypertension; other abbreviations as in Table 1
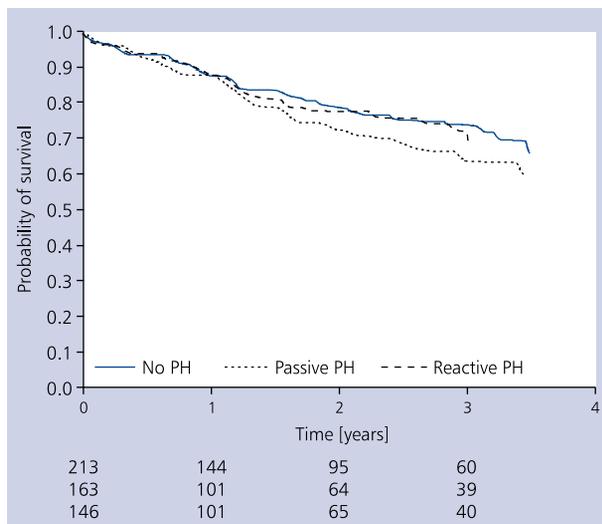
Figure 2. Probability of survival in patients with chronic heart failure and with no-pulmonary hypertension (PH), passive PH, and reactive PH; p = NS

with no PH or passive PH. Mean PAP was 38.4 ± 7.8 mm Hg, PCWP 27.0 ± 7.4 mm Hg, TPG 15.6 ± 5.8 mm Hg, CI 1.6 ± 0.5 L/min, PVR 4.8 ± 1.7 WU. There were no differences in NT-proBNP, VO$_2$max, HFSS, and Na level between patients with passive or reactive PH.

The prognosis of patients with reactive PH (Kaplan-Meier method) was comparable with patients with no or with passive PH (Fig. 2).

Among the parameters analysed, LVEDD, LVESD, systolic blood pressure, mPAP, PCWP, CI, NT-proBNP, HFSS, and Na level significantly influenced patient survival in the univariate Cox regression model (Table 2). Multifactor regression analysis revealed that only serum Na level (HR 0.900, CI 0.823–0984) influenced the survival of patients with reactive PH.

DISCUSSION

There are varied opinions concerning the influence of PH on the prognosis of patients who are candidates for HTx. Some authors have reported that patients with HF qualified for HTx have poor survival and that PH has no additive influence on their prognosis [2, 9, 12, 13].

The main aim of our study was to assess the prognostic value of reactive PH in ambulatory patients waiting for HTx. That is a group of patients in whom functional as well as structural changes in pulmonary circulation can be suspected [5–9].

Table 3. Characteristics of patients according to pulmonary hypertension subgroups

	No PH mPAP < 25 mm Hg	Passive PH mPAP ≥ 25 mm Hg PCWP ≥ 15 mm Hg PVR < 3.0 WU	Reactive PAH mPAP ≥ 25 mm Hg PCWP ≥ 15 mm Hg PVR ≥ 3.0 WU	Group I vs. group II	Group I vs. group III	Group II vs. group III
Age [years]	52.4 ± 8.5	48.3 ± 10.4	49.8 ±10.4	< 0.05	< 0.05	NS
LVEF [%]	22.0 ± 8.5	20.7 ± 7.3	19.4 ± 6.1	< 0.05	< 0.05	NS
LVEDD [mm]	72.3 ± 10.2	72.2 ± 11.8	72.6 ± 8.8	NS	NS	NS
LVESD [mm]	60.2 ± 13.3	61.1 ± 12.0	61.5 ± 12.7	NS	NS	NS
HR [/min]	76.8 ± 15.4	77.7 ± 14.7	78.7 ± 14.1	NS	NS	NS
SBP [mm Hg]	103.8 ± 15.7	98.5 ± 11.6	101.0 ± 12.8	< 0.05	NS	NS
DBP [mm Hg]	67.1 ± 11.6	64.8 ± 9.0	67.1 ±9.7	< 0.05	NS	< 0.05
Na [mEq/L]	136.6 ± 4.2	136.3 ± 4.2	136.0 ± 4.8	NS	NS	NS
VO$_2$max [mL/min/kg]	12.6 ± 3.7	12.3 ± 3.5	11.8 ± 3.6	NS	NS	NS
HFSS	7.8 ± 0.9	7.6 ± 0.9	7.5 ± 0.9	< 0.05	< 0.05	NS
mPAP [mm Hg]	19.9 ± 3.1	34.0 ± 6.6	38.4 ± 7.8	< 0.05	< 0.05	< 0.05
PCWP [mm Hg]	14.1 ± 3.0	26.6 ± 6.5	27.0 ± 7.4	< 0.05	< 0.05	NS
TPG [mm Hg]	7.7 ± 3.9	8.1 ± 3.5	15.6 ± 5.8	NS	NS	NS
Cardiac index [L/min/ m^2]	2.2±1.2	1.9 ± 0.5	1.6 ± 0.5	NS	< 0.05	< 0.05
PVR [WU]	2.2 ± 1.3	2.1 ± 0.7	4.8 ± 1.7	NS	< 0.05	< 0.05
hs-CRP [mg/L]	3.0 (0.1–114.3)	3.0 (0.1–68.7)	3.8 (0.1–62.1)	NS	NS	NS
NT-proBNP [pg/mL]	1695 (123–29873)	3113 (77–45515)	4240 (88–38829)	< 0.05	< 0.05	NS

PH — pulmonary hypertension; other abbreviations as in Table 1

Structural changes in venous and arterial pulmonary circulation are secondary to elevated LV filling pressure, and decreased left atrial and pulmonary vein compliance. Such structural consequences are well known in mitral stenosis, but there are few papers focused on such changes in patients with advanced HF [14–17]. In both models the rise of pressure in pulmonary circulation leads to certain changes in the function and morphology of arteries and veins. There are selected publications taking into account the problems above. Some of them underline that patients with higher levels of pre HTx PAP have a residual PH even three years after transplantation, suggesting the presence of structural changes [14, 18]. There are also some suggestions that PH referred to as "fixed" because of little or no reversibility with pharmacological tests or treatments before HTx, represents a major risk factor for prognosis after HTx, because of structural vascular remodelling. Delgado described advanced changes in media thickness assessed after HTx in a group of patients with reactive PH as compared to structural changes among those with passive PH [19]. A similar pathology was described by Hasleton and Brooks [20]. Khush et al. [9], using comparable methods to describe patients with mixed (reactive) PH, proved that this kind of PH was common in patients with HF but did not predict poor short-term clinical outcomes. They underlined that

patients with advanced HF in NYHA class IV generally had poor prognosis, and PH or no PH was no longer a significant prognostic factor.

Remodelling of pulmonary vasculature could be assessed taking into account the difference between diastolic PAP and PCWP when PVR is known. According to Engelberg and Dubios [21], experimental findings show that the difference (diastolic pulmonary gradient) could be used to assess diastolic PAP compliance. Tedford et al. [22], using the UNOS database, selected patients with PH (mPAP ≥ 25 mm Hg). Diastolic pulmonary gradient has no effect on patient's survival until five years after HTx, irrespective of TPG and PVR. The authors underlined that TPG is independent of cardiac output and so better reflects vascular compliance than PVR alone [22]. Results of Tedford extended our findings concerning prognosis of patients with PH after HTx. There is also important information (not published) that, irrespective of vasodilator testing for reversibility of PH, there is no difference in six-month survival (21% vs. 22%) between those with or without reactive PH [9]. Among patients with advanced HF and PH, right ventricular function significantly influences their prognosis, but right ventricular dysfunction with normal PAP poses no additional risk [23, 24]. Aronson et al. [5], whose methodology describing PH was used in our study, found

significant differences in prognosis between patients with reactive PH as compared to patients with no PH or passive PH. It should be underlined that the population of patients with HF was different. They were patients with acute HF included in a randomised trial, so the patients and the way of assessing the results were different. The authors estimated survival according to post-treatment PH categories. In a retrospective analysis, when PH was assessed before treatment, the survival of compared groups was nearly identical [5] and comparable to ours. Our registry covered a homogenous cohort of patients with chronic HF listed for elective HTx. There were patients treated according to the guidelines by appropriate in- and outpatient departments.

The most intriguing finding of our study was that the survival of patients with reactive PH, who met the criteria of reversibility, was comparable with patients with no PH or passive PH.

Limitations of the study

The results of the study were based on the data taken from the national registry of patients listed for HTx. The measurements of pulmonary haemodynamicity were not standardised; moreover, there were no available data concerning PH reversibility. Because of a necessary simplification of the registry, some data were not recorded, namely data concerning right ventricular function and details concerning treatment.

We decided to follow the Aronson classification of PH because of specificity of patients qualified to HTx [5]. It is a group of patients in whom PCWP is high, and consequently TPG is relatively low; moreover, TPG has no influence on patient survival irrespective of the analysed group.

Finally, the groups of patients with no PH, passive PH, or reactive PH were relatively small and the follow-up was short, not including the patient's survival after HTx. Thus the data concerning factors influencing survival and prognosis should be interpreted with caution.

CONCLUSIONS

Pulmonary hypertension is the result of longstanding left ventricular HF. Our results indicate that reactive PH is common in ambulatory patients with advanced HF listed for elective HTx. Reactive PH has no further influence on prognosis at this stage of disease, despite the fact that patients with reactive PH had the worst pulmonary haemodynamic scores.

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Znaczenie prognostyczne reaktywnego nadciśnienia płucnego u pacjentów z przewlekłą niewydolnością serca kwalifikowanych do transplantacji serca

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Streszczenie

Wstęp: Nadciśnienie płucne (PH) rozpoznaje się u ok. 60% pacjentów rozpatrywanych jako kandydatów do transplantacji serca (HTx), co wpływa na przebieg kliniczny.

Cel: Celem pracy było określenie wartości prognostycznej reaktywnego PH pacjentów kwalifikowanych w trybie planowym do HTx.

Metody: Analizowano dane 522 pacjentów biorących udział w rejestrze POLKARD kwalifikowanych do HTx, u których wykonano cewnikowanie prawego serca. Punktem końcowym badania były wszystkie przypadki zgonu przed transplantacją serca. Przeprowadzenie HTx w trybie planowym lub pilnym uznano za koniec obserwacji (średni czas obserwacji 1,47 ± 1,26 roku). Pacjenci zostali podzieleni na trzy podgrupy: bez PH, z PH, z reaktywnym PH, a jako punkt odniesienia brano pod uwagę wartości średniego ciśnienia w tętnicy płucnej (PAP) 25 mm Hg, ciśnienia zaklinowania we włośniczkach płucnych (PCWP) 15 mm Hg i naczyniowego oporu płucnego (PVR) 3,0 j. Wooda. Przeprowadzano ocenę kliniczną oraz wykonywano badania hemodynamiczne, echokardiograficzne i biochemiczne krwi (m.in. NT-proBNP, hs-CRP). Wyliczano też wartość Heart Failure Survival Score (HFSS).

Wyniki: Śmiertelność była porównywalna między grupami, mimo że w badaniach hemodynamicznych pacjentów z reaktywnym PH wyniki były najwyższe (średnie PAP 38,4 ± 7,8, PCWP 27 ± 7,4 mm Hg, PVR 4,8 ± 1,7 j. Wooda). W grupie pacjentów z reaktywnym PH w wieloczynnikowej analizie Coxa tylko stężenie sodu wpływało na przeżycie. Na podstawie analizy metodą Kaplana-Meiera stwierdzono, że pacjenci z reaktywnym PH, ze średnim PAP ≥ 41,4 mm Hg, charakteryzowali się najgorszym rokowaniem, a szacunkowe przeżycie jednoroczne i dwuletnie wynosiło odpowiednio 83% i 65%.

Wnioski: Reaktywne PH jest częstym zjawiskiem u osób z przewlekłą niewydolnością serca kwalifikowanych do HTx. Nie ma to dalszego wpływu na krótkotrwałe rokowanie w porównaniu z pacjentami bez PH lub z PH. Stężenie sodu i średnie PAP ≥ 41,4 mm Hg wpływa na rokowanie pacjentów z reaktywnym nadciśnieniem płucnym.

Słowa kluczowe: transplantacja serca, nadciśnienie płucne

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