

Clinical, haemodynamic and echocardiographic features of early cardiac graft dysfunction

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Abstract

Background: The main cause of early death after heart transplantation (HTx) is so-called early primary or secondary graft failure (GF). The risk of profound GF has not declined in the past decade, as the consequence of the liberalisation of donor acceptance criteria because of the scarcity of donors. It is therefore important to try to diagnose graft failure and recognise the mechanisms of early graft dysfunction.

Aim: To establish haemodynamic and echocardiographic criteria of early GF to define patients who should be considered for assist device support or re-transplantation.

Methods: Between January 2000 and March 2009, 116 HTx patients were studied. On the basis of echocardiography and continuous invasive monitoring, three groups were identified: (1) The true graft failure group (GF) consisted of 46 patients; (2) The latent right ventricular (RV) dysfunction group (RV-D) consisted of 25 patients with small left ventricular (LV) chamber (< 39 mm) and RV ejection fraction (RVEF) < 50%; (3) The control group consisted of 45 consecutive HTx patients without any haemodynamic complications.

Results: Postoperatively, only the GF group required large doses of norepinephrine (> 0.3 µmg/kg/min) and inhalative NO (40 ppm). Nevertheless, right and left filling pressures were significantly higher than in the controls (right 12 ± 3.6 vs. 9.0 ± 2 and left atrial pressure 13.0 ± 3.2 vs. 9.6 ± 2 mm Hg, both $p < 0.001$). Cardiac index was significantly smaller (2.9 ± 0.7 vs. 3.7 ± 0.9 , $p < 0.001$) but neither pulmonary artery pressure (29.5 ± 6 vs. 29.7 ± 7 mm Hg) nor transpulmonary gradient (6 ± 5 vs. 5.1 ± 5 mm Hg) nor pulmonary vascular resistance (273 ± 97 vs. 287 ± 144 dyn \times s \times cm⁻⁵) differed significantly from those of the control group. In the GF group, LV end diastolic dimension (LVEDD) was significantly smaller and function poorer than in controls (39.8 ± 5 vs. 44.4 ± 5 mm, respectively, $p = 0.001$). RV function was also significantly worse (RVEF $42.2 \pm 14\%$ vs. $56.0 \pm 9\%$), respectively, $p = 0.001$), whereas RV dimension did not differ significantly. Mechanical support after failure of the initial medical treatment was necessary in 37% of patients; 29 (63.0%) patients from the GF group died, the cause of death being sepsis with multi-organ failure. In the RV-D group, remodelling was quite similar but LVEF was excellent and maximal systolic velocity from the posterior wall was significantly higher than in GF. No death occurred.

Conclusions: True early GF represents a grave haemodynamic situation with high mortality. Bedside echocardiography helps to distinguish between latent RV dysfunction and true GF.

Key words: heart transplantation, graft failure, echocardiography

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INTRODUCTION

The main cause of early death after heart transplantation (HTx) is so-called early primary or secondary graft failure (GF). Primary GF is defined as severe dysfunction of the cardiac allo-

graft without any obvious anatomic or immunologic cause [1, 2]. Secondary GF failure can be attributed to unresponsive pulmonary hypertension or, rarely, to hyperacute rejection. Some authors indicate that primary GF has to be treated

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by the early institution of mechanical support [3–5] and that secondary GF, especially in cases of elevated pulmonary vascular resistance (PVR), should be treated primarily by medical means, such as nitric oxide (NO) inhalation [6].

The risk of profound GF has not declined in the past decade [7], as the consequence of the liberalisation of donor acceptance criteria because of the scarcity of donors. It is therefore important to try to diagnose GF and recognise the mechanisms of early graft dysfunction.

The aim of this study was to establish haemodynamic and echocardiographic criteria of early GF to define patients who should be considered for assist device support or re-transplantation [8].

METHODS

Patient population

Heart transplantation was performed in a total of 1,651 patients (1,331 men and 320 women) at the Deutsches Herzzentrum Berlin between April 1986 and October 2010. This prospective haemodynamic and echocardiographic study was carried out between January 2000 and March 2009 to compare patients with and without clinical signs of GF. Altogether, 116 patients were extensively studied after transplantation. On the basis of echocardiographic examination and invasive data, the patients were assigned to the true GF group or the latent right ventricular (RV) dysfunction group, and compared to a control group (Fig. 1).

The true GF group. The true GF group, collected prospectively between 2000 and 2009, consisted of 46 patients (mean age 50 ± 13.5 years, 37 men, nine women). Early graft dysfunction was diagnosed on the basis of clinical, haemodynamic and echocardiographic criteria: (1) the main criteria were clinical signs of shock or hypotension refractory to treatment with filling pressures elevated above 12 mm Hg as the main haemodynamic criterion, and the need for excessive doses of norepinephrine (exceeding $0.3 \mu\text{mg/kg/min}$); (2) the secondary criteria were clinical signs of low cardiac output (CO), collapsed form of haemodynamics (hypotension with normal or reduced left ventricular [LV] filling pres-

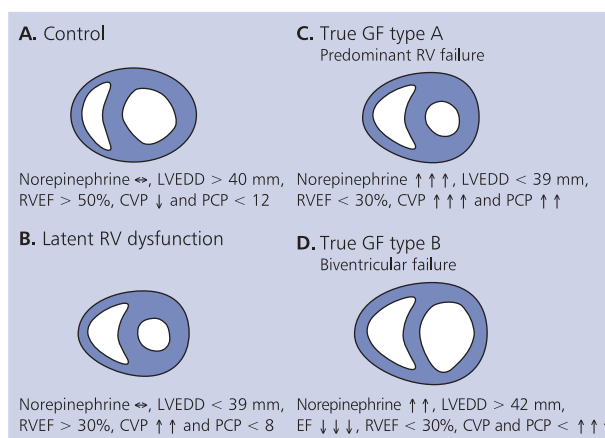


Figure 1. A. Normal relationship between right ventricle (RV) and left ventricle (LV) after transplantation, in the short axis view; B. Small LV chamber and increased dimension of RV in the same projection; C. Same relationship between RV and LV as in B in patients suffering from true graft failure (GF) (a less typical case but a very clear pattern of RV dysfunction); D. Relation between RV and LV in patients with biventricular GF; \leftrightarrow indicates moderate dose of epinephrine; LVEDD — left ventricular end diastolic diameter; RVEF — right ventricular ejection fraction; CVP — central venous pressure; PCP — pulmonary capillary pressure

ures but elevated RV filling pressures) and the necessity for norepinephrine (exceeding $0.3 \mu\text{mg/kg/min}$) to maintain proper blood pressure with one echocardiographic sign of heart dysfunction (reduced RV ejection fraction [RVEF] < 50% and/or relatively small LV cavity ≤ 39 mm). However, in four patients, a restrictive form of RV failure was found (small, stiff and hyperkinetic RV).

This group of patients required mechanical support for haemodynamic stabilisation. The reasons for HTx are presented in Table 1. Based on echocardiography (LV dimension > 42 mm), the GF group is subdivided into A and B (Figs. 1C, D).

The latent RV dysfunction group. The latent RV dysfunction (RV-D) group consisted of 25 consecutive patients

Table 1. Preoperative characteristics of studied patients

	Control group (n = 45)	Graft failure group (n = 46)	P
Recipient age [years]	50 ± 13	50 ± 14	0.47
Female	7 (16.3%)	9 (19.6%)	0.3
Recipient body surface area (preoperative assessment)	1.86 ± 02	1.88 ± 02	0.3
Dilated cardiomyopathy	32 (71.1%)	28 (60.9%)	0.03
Ischaemic cardiomyopathy	11 (24.4%)	14 (30.4%)	0.41
Other myocardial diseases	2 (4.4)*	4 (8.7)**	NS
Preoperative use of ventricular assist device	10 (23.3%)	9 (19.6%)	0.24
Ischaemic time [min]	203 ± 48	209 ± 48	0.32

*Marfan syndrome (n = 1), congestive heart failure after double valve replacement; **hypertrophic obstructive cardiomyopathy (n = 2), complex congenital heart failure (n = 1), congestive heart failure after double valve replacement (n = 1)

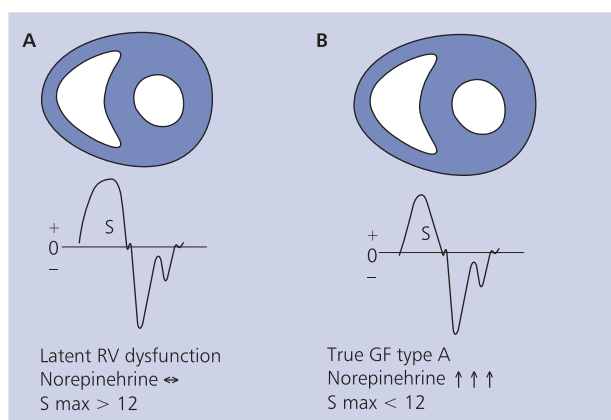


Figure 2. **A.** Latent right ventricle (RV) dysfunction with normal values of systolic myocardial velocity of left ventricle (LV) posterior wall; **B.** True graft failure (GF) type A with same RV/LV relations as in 2A; maximal systolic velocity (S max) is significantly smaller in the true GF group

(mean age 49 ± 12 years, 21 men, four women) who fulfilled our echocardiographic criteria of RV dilation and/or dysfunction (RV > 35 mm in parasternal view, EF < 50%) and had normal or hyperkinetic systolic function of the LV (EF > 65%), small LV chamber (< 39 mm) and systolic velocity of the posterior wall exceeding 12 cm/s recorded from the parasternal view (Fig. 2A). These patients did not suffer from haemodynamic instability but were treated with moderate norepinephrine (not exceeding $0.3 \mu\text{g}/\text{kg}/\text{min}$) and moderate doses of NO inhalation (27 ± 15 ppm) during mechanical ventilation in accordance with our institutional policy. Pretransplant diagnoses were: dilated cardiomyopathy ($n = 18$), ischaemic cardiomyopathy ($n = 5$), Marfan syndrome ($n = 1$) and congestive heart failure after double valve replacement ($n = 1$).

The control group. The control group consisted of 45 patients who were our historical group examined by the same investigator early after HTx between 2000 and 2003 who had no haemodynamic complications or any echocardiographic signs of dysfunction (Fig. 1A, Table 1). The reasons for HTx are presented in Table 1. This group needed only minimal pharmacological support (norepinephrine not exceeding $0.3 \mu\text{g}/\text{kg}/\text{min}$) and did not receive any other medication to support haemodynamic function.

Exclusion. Patients suffering from graft rejection were not included in the study.

Echocardiography and invasive monitoring

All patients received serial echocardiography using the ALOKA Japan 5500 or $\alpha 10$ machine, including transoesophageal mode, in the first 2–6 hours and were followed according to their haemodynamic situation. In unstable patients, echocardiography was performed every second day, according to

a protocol published elsewhere [9]. RV assessment by Simpson's method was made in the 2D parasternal, four-chamber and short axis views using a transthoracic approach and the transoesophageal mode of investigation. A tissue Doppler study was performed from transthoracic echocardiography of the parasternal long axis. Maximal systolic velocity (S max) from the posterior wall was recorded online using pulsed Doppler.

In addition, the patients were monitored on the basis of Swan-Ganz catheterisation for pulmonary artery pressure (PAP) and left and right filling pressures. CO was assessed according to thermodilution principles. The values indicating 'borderline' patients — those between latent and true graft dysfunction — were selected according to clinical development and necessity of pharmacological support.

Management of immunosuppression and heart biopsy was undertaken in accordance with our institutional policy. None of the patients died of an acute rejection reaction. In all patients, the heart rate was maintained by pacing.

Statistical analysis

Data was entered into a commercially available statistical program for analysis. Values are expressed as mean \pm SD. Statistical significance was assumed for $p < 0.05$.

RESULTS

Pretransplant factors

The recipient characteristics of the control group and GF group are outlined in Table 1 and Figure 1A. There were no differences between the groups in terms of age, body surface area, incidence of dilated or ischaemic cardiomyopathy, or ventricular assist device implantation as a bridge to HTx. The ischaemia time of the donor heart was almost identical in both groups.

Postoperative findings

Haemodynamic instability present in the **GF group** of patients required aggressive medical treatment (mean suprenalin $0.38 \pm 19 \mu\text{g}/\text{kg}/\text{min}$ for this cohort of patients and inhalative NO doses 34.5 ± 9 ppm). Nevertheless, the right and left filling pressures were significantly higher than in the control group (12 ± 3.6 vs. 9 ± 2 mm Hg, $p < 0.001$, and 13 ± 3.2 vs. 9.6 ± 2 mm Hg, $p = 0.003$, respectively). The shock symptoms and/or low CO dominated the clinical situation. The cardiac index (CI) in the GF group was significantly smaller than in the control group (2.9 ± 0.7 vs. 3.67 ± 0.9 , $p = 0.0001$); neither PAP nor transpulmonary gradient (TPG) nor PVR differed significantly from those of the control group (Table 2).

Echocardiographic examination showed S max from the posterior wall to be significantly lower in the GF group than in the control group (11.2 ± 3 vs. 14.9 ± 2.3 cm/s, $p = 0.05$) (Table 3). The left ventricular end-diastolic diameter (LVEDD) was significantly smaller, and RV function significantly

Table 2. Postoperative haemodynamic characteristics of the control group (n = 45), the true graft failure (GF) group, and the latent right ventricular dysfunction group (RV-D)

	Control group (n = 45)	True GF group (n = 46)	P: Control vs. GF	Latent RV-D group (n = 25)	P: GF vs. Latent RV-D
Heart rate [bpm]	107 ± 10	112 ± 12	0.02	115 ± 9	0.4
Central venous pressure [mm Hg]	9 ± 2	12 ± 3.6	0.001	8.9 ± 3	0.003
Left atrium [mm Hg]	9.6 ± 2	13 ± 3.2	0.003	9.7 ± 3	0.09
Pulmonary artery pressure systolic [mm Hg]	29.7 ± 7	29.5 ± 6	0.2	33 ± 7	0.07
Cardiac index [L/min/m ²]	3.7 ± 0.9	2.9 ± 0.7	0.0001	3.3 ± 0.9	0.3
Transpulmonary gradient [mm Hg]	5.1 ± 5	6 ± 5	0.3	11 ± 4	0.002
Pulmonary vascular resistance [dyn × s × cm ⁻⁵]	287 ± 144	273 ± 97	0.3	322 ± 146	0.09

Bold type indicates statistical significance

Table 3. Echocardiographic characteristics of the control group, the true graft failure (GF) group, and the latent right ventricular dysfunction group (RV-D)

Echocardiography	Controls (n = 45)	True GF group (n = 46)	P: Controls vs. GF	Latent RV-D group (n = 25)	P: GF vs. Latent RV-D
Left ventricular end diastolic diameter [mm]	44.4 ± 5	39.8 ± 5	0.001	35.5 ± 5	0.003
Left ventricular end diastolic volume [mL]	92 ± 15	72 ± 16	0.001	67 ± 16	0.05
Left ventricular ejection fraction [%]	65 ± 9	60.5 ± 11	0.05	70.1 ± 7	0.003
Left ventricular systolic volume [mL]	59.8 ± 9	43.9 ± 9	0.05	47 ± 9	0.3
Right ventricular end diastolic diameter [mm]	29.9 ± 4	29.8 ± 4.5	0.2	29.5 ± 5	0.3
Right ventricular ejection fraction [%]	56 ± 9	42.2 ± 14	0.001	50.7 ± 13	0.2
S max [cm/s]	14.9 ± 2.3	11.2 ± 3	0.05	15.2 ± 3	0.001

Bold type indicates statistical significance; S max — maximal myocardial velocity of posterior wall (pulsed Doppler)

poorer, in the GF group than in the control group (39.8 ± 5 mm vs. 44.4 ± 5 mm, p = 0.04 and 49% ± 14.2 vs. 56.0 ± 10.3%, p = 0.001, respectively). LV function was significantly worse than in the controls (60.5 ± 11% vs. 65 ± 9%, p = 0.05), whereas RV dimension did not differ significantly. Relatively small LV chamber (even with acceptable EF value) in the GF group was responsible for low CO (2.9 ± 0.7 L/min/m²) (Table 2).

Two different forms of GF according to echocardiographic measurements were observed in the GF group and the patients were divided into subgroups accordingly.

The first subgroup (group A, the majority of the patients, 89%, 41/46) suffered from predominant RV myocardial failure. In these patients, RVEF was reduced to 45 ± 20%, the LV was smaller than in controls (38.1 ± 5 vs. 44.4 ± 5 mm, p = 0.001) and under intensive inotropic stimulation, the LVEF was 61 ± 14% (Fig. 1C).

The second subgroup (group B) suffered from primary biventricular failure (only five patients; 5/46, 11%). The patients required the same intensive doses of catecholamines

and demonstrated poor quality of RV function (EF 42 ± 15%) but significantly larger LV and worse systolic function than in the subgroup A (48 ± 1.4 vs. 38.1 ± 5 mm and for EF 54 ± 11 vs. 61 ± 14%, for both p = 0.07) (Fig. 1D).

In the GF group, surgical intervention (mechanical support) to keep patients alive was required in 15/46 patients: intraaortic balloon pump (32.6%) and ventricular assist device (BerlinHeart) in two (4.4%) patients.

High early mortality was observed in the GF group (29/46, 63.0%). The cause of death in all cases was sepsis with multi-organ failure that developed after prolonged treatment of heart failure. None of the patients suffered from haemodynamically relevant rejection or other 'anatomical' causes or tamponade.

Latent RV dysfunction group was characterised by small LV chamber (< 40 mm) and/or relatively poor RVEF (< 50%) as studied by echocardiography, but patients in this group did not need large doses of catecholamines. The type and grade of remodelling were quite similar to those of the GF group, but LVEF was excellent without large doses of norepi-

nephrene (70.1 ± 7 vs. $60.5 \pm 11\%$ in the GF group, $p = 0.003$) and S_{\max} from the posterior wall was significantly higher than in the GF group (15.2 ± 3 cm/s vs. 11.2 ± 3 , $p = 0.001$) (Table 3, Fig. 2).

Invasive haemodynamic values demonstrated almost identical central venous pressure and left filling pressure as in the control group (8.9 ± 3 and 9.7 ± 3 mm Hg and 9.0 ± 2 vs. 9.6 ± 3 mm Hg, respectively) (Table 3). These values were significantly different from those of the GF group (12 ± 3.6 mm Hg, $p < 0.001$; 13 ± 3.2 mm Hg, $p = 0.09$ respectively). Systolic PAP and TPG were more highly elevated in the latent RV dysfunction group than in the control group (33 ± 7 and 11 ± 4 vs. 29.7 ± 7 and 5.1 ± 5 mm Hg, respectively) and were also higher than in the GF group ($p = 0.07$ for TPG and $p = 0.09$ for PVR) (Table 2). CI was not significantly higher than in the GF group; however, pulmonary resistance was significantly higher (3.3 ± 0.9 vs. 2.9 ± 0.7 mL/kg/m² and 322 ± 146 vs. 373 ± 97 PVR dyn \times s \times cm⁻⁵; $p = 0.09$, respectively) (Table 2).

The control group and the latent RV dysfunction group were characterised by stable haemodynamics and no death was recorded.

DISCUSSION

Graft failure group

Early diagnosis of GF is crucial in order to undertake adequate treatment. In general, invasive postoperative values (PAP and PVR and TPG) did not discriminate 'good' from 'bad' patients (Table 2), and only CI and filling pressures of the RV and LV may help to distinguish three patient groups (Table 2). Additional echocardiographic examination revealed the origin of haemodynamic problems more precisely. LVEDD in the GF group was significantly smaller ($p = 0.001$) and EF was worse than in the control group. Recording of LV posterior wall velocity helped to differentiate between the GF group and the latent RV-D group (both characterised by similar invasive and echocardiographic values). Generally speaking, the velocity of the myocardial wall (and calculated values like strain) early after transplantation fluctuates, depending on the right and left afterload and inotropic stimulation. However, this method helps to identify the condition of the myocardium and is regularly used for follow-up of ambulatory patients by our institution and others [10].

Recognised remodelling of the LV in the GF group has to be explained by interaction with the failing RV which produces smaller stroke volume supplying the LV. The LV, not being adequately supplied, has to retract to adapt to the actual filling. This explanation is based on the significantly smaller RVEF in the GF group than in the control group. It is also important to remember that the GF group required high doses of catecholamines. In the GF group, systolic PAP was significantly lower after transplantation than in the control group. A significant drop in systolic PAP and elevated pulmo-

nary resistance indicates that the transplanted heart was not able to 'match' the host pulmonary resistance in the GF group. In fact, the same explanation has to be used for the control group but probably the 'grade' of mismatch was not significant and was tolerated haemodynamically; in the control group, normal CO was recorded, while in the GF group, low CO was one of the main signs of haemodynamic failure.

GF patients were divided into groups A and B according to the type of graft failure: group A had RV failure and group B biventricular failure. The larger group, group A (89%, 41/46), demonstrated a relatively small but significantly better functioning LV. These patients suffered from predominant loss of RV performance and probably should have been supported by an RV assist device only. Group B, which was smaller (5/46, 11%), had similar invasive parameters as group A, but the LV chamber was dilated and characterised by very poor function.

It should be noted that, in our study, early after transplantation the RV was typically not able to generate adequate pressure to face the pulmonary resistance. The LV was probably underloaded in all groups. Nevertheless, in such a situation, elevation of the LV filling pressure indicates severe diastolic dysfunction of the chamber [11, 12] which surely has to be attributed to graft incompetence. From this viewpoint, it can be stated that invasive parameters alone, such as low PAP, are misleading and may obscure the diagnosis.

No single parameter was proven to be a good marker of early GF. Non-invasive and invasive parameters taken together were helpful in diagnosing GF.

Latent RV dysfunction group

The patients with latent RV dysfunction were distinguished from patients in the control group on the basis of echocardiographic investigation only, as the controls did not have any haemodynamic instability or suffer from clinical problems. However, CO was only insignificantly higher than in the GF group. According to our policy, these patients received moderate doses of inhalative NO and follow-up was necessary to avoid the risk of complications.

As stated above, the distinction between the GF and latent RV dysfunction groups was based on tissue Doppler. S_{\max} velocity assessed by pulsed Doppler of the posterior wall was significantly higher in the latent RV-D group than in the GF group.

Postoperative risk factors for bad outcome

There is a scarcity of literature describing the risk factors in the early period after transplantation. Donor related factors are considered important [13]; among them, recipient ventilator support, number of prior sternotomies, older age and abnormal donor echocardiogram are risk factors as determined in multi-institutional, multivariable analyses [14]. In other studies, prolonged ischaemic time was found to be a risk factor for fatal GF but it did not cause a worse long term outcome [15, 16]. Some studies demonstrate that restriction of

the LV after transplantation later in time challenged the outcome [17].

Some authors have suggested differentiating between primary and secondary GF [18]. GF is, in fact, a more complex issue. The majority of our patients with haemodynamic problems early after HTx demonstrated a mixed clinical picture affecting predominantly the right side but also the left side of the heart. In most patients, leading signs of collapse (hypotension with normal LV filling pressure) or shock (elevated left filling pressures) or a combination of the two were present, and in the later course some of them developed a septic reaction or sepsis-like syndrome leading to death. In the very early period (hour/hours) after transplantation, RV failure dominated LV failure. In the latent GF group, these signs were stable and in the later course normalisation was observed without special treatment. However, the true GF patients suffered from LV failure as well but the mean capillary pressure was not strongly elevated.

In this study, evidence suggests that these two pathological situations, i.e. elevated PVR and pure myocardial failure, play a combined role in cases with GF. Elevated pulmonary resistance does not cause elevation of pulmonary systolic pressure. However, it probably protects the LV from filling pressure elevation.

In the literature, these two different pathological situations have rarely been treated together [19]. In both forms of GF, effective reduction of PVR, based, for example, on pre-treatment with substances that lower pulmonary resistance before transplantation, would probably have a great influence on the outcome.

CONCLUSIONS

The great majority of GF patients demonstrated a mixed clinical picture of shock and collapse (small undervolumed LV chamber, low LV posterior wall velocity [< 12 cm/s] and hypotension and at the same time overdistended RV chamber).

It is important to note that it is not elevated PAP, but rather rising filling pressure at the right side of the heart with reduced CI, that indicates early true GF. On the other hand, isolated low filling pressure of the LV can also be the first sign of GF. In such patients, echocardiographic examination is very helpful to verify whether RV failure is present (overdistended and reduced EF of RV) with parallel reduced LV dimensions.

The latent form of GF has to be distinguished from true GF. This particular group can be diagnosed almost conclusively by echocardiographically documented small LV (often smaller in size than the RV), which speaks for a relatively overdimensioned RV. This group of patients did not require mechanical support and was characterised by good outcome; however, treatment with NO is required to avoid possible transmission of this haemodynamic state into true GF.

Findings from this study indicate that neither invasive monitoring nor echocardiography alone can diagnose early GF. Early diagnosis should be based on clinical signs and invasive and echocardiographic monitoring.

Early identification of patients at risk for GF is crucial for adequate treatment and better outcome.

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Obraz kliniczny, echokardiograficzny i hemodynamiczny wczesnej niewydolności przeszczepu serca

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Streszczenie

Wstęp: Celem niniejszej pracy, opartej na wynikach badań prospektywnych, jest określenie parametrów hemodynamicznych i echokardiograficznych wczesnej postaci niewydolności przeszczepu serca.

Metody: Badania przeprowadzono u 116 chorych po transplantacji w latach 2000–2009 w Centrum Zdrowia Serca w Berlinie. Na podstawie wyników badań inwazyjnych i echokardiograficznych oraz przebiegu klinicznego chorych podzielono na 3 grupy: grupa określona jako jawna niewydolność pooperacyjna przeszczepu (GF) składała się z 46 chorych, grupa latentnej niewydolności prawej komory (RV-D) — z 25 chorych, a grupa kontrolna — z 45 chorych. Badane grupy nie różniły się pod względem wieku, płci, wskaźnika masy ciała, przyczyny niewydolności serca, częstości wspomaganie sztucznym sercem ani czasem przedoperacyjnego niedokrwienia graftu. Wyniki uzyskane w grupach GF i RV-D porównywano z grupą kontrolną charakteryzującą się prawidłowym przebiegiem klinicznym, zbadaną w latach 2000–2003.

Wyniki: W okresie wczesnym po operacji tylko grupa GF była leczona dużymi dawkami norepinefryny ($> 0,3 \mu\text{mg/kg/min}$) oraz inhalacyjnie tlenkiem azotu — NO 40 ppm. Mimo tej terapii ciśnienia napełniania prawej (OCŻ) i lewej komory (ciśnienie zaklinowane, CZ) były znacząco wyższe niż w grupie kontrolnej, odpowiednio $12,3 \pm 3,6$ vs. $9,0 \pm 2$ i $13,0 \pm 3,2$ vs. $9,6 \pm 2$ mm Hg ($p < 0,001$). Także wartość wskaźnika sercowego była znacząco niższa ($2,9 \pm 0,7$ vs. $3,7 \pm 0,9$ ml/kg/min; $p < 0,001$), natomiast wartości ciśnienia skurczowego w tętnicy płucnej ($29,5 \pm 6$ vs. $29,7 \pm 7$ mm Hg, NS), gradientu przepłucnego czy oporu płucnego nie różniły się znacząco. Wartość echokardiograficzna wymiaru końcoworozkurczowego lewej komory (LVEDD) była znacznie mniejsza w grupie GF w porównaniu z grupą kontrolną i wynosiła odpowiednio $39,8 \pm 5$ vs. $44,4 \pm 5$ (p = 0,001). Grupa RV-D charakteryzowała się m.in. podobnym do grupy GF stopniem remodelingu serca określonym w badaniu echokardiograficznym (LVEDD < 40 mm), ale wartości ciśnienia napełniania lewej i prawej komory nie różniły się od mierzonych w grupie kontrolnej; OCŻ i CZ wynosiły odpowiednio $8,9 \pm 3$ vs. $9,7 \pm 3$ oraz $9,0 \pm 2$ vs. $9,6 \pm 3$ mm Hg (NS) dla obu parametrów. Natomiast wskaźnik sercowy w grupie RV-D był podobny jak w grupie GF i wynosił odpowiednio $3,3 \pm 0,9$ vs. $2,9 \pm 0,7$ l/min/m² (NS). Istotne różnice dotyczące grupy RV-D w stosunku do grupy GF wystąpiły w pomiarze maksymalnej prędkości skurczowej mięśnia tylniej ściany lewej komory, które wynosiły $15,2 \pm 3$ i $11,2 \pm 3$ cm/s ($p = 0,001$). Wysoką śmiertelność z powodu niewydolności organów zanotowano jedynie w grupie GF i wynosiła ona 63% (29/46) mimo stosowania kontrapulsacji wewnątrzortalnej u 32,6% (15/46) oraz sztucznego serca u 2 chorych. Nie obserwowano odrzutu przeszczepu.

Wnioski: Postać wczesnej, rzeczywistej niewydolności przeszczepu po transplantacji serca nie wiąże się z odrzuceniem przeszczepu i wiąże się z dużym ryzykiem śmiertelności. Choć stan hemodynamiczny chorego wynikający z niewydolności przeszczepu odbiega znacząco od przebiegu normalnego, to często typowe wykładniki ostrej niewydolności nie muszą być dominujące (ciśnienie w tętnicy płucnej). Wczesne rozpoznanie jest możliwe po analizie danych klinicznych hemodynamicznych i echokardiograficznych.

Słowa kluczowe: transplantacja, niewydolność serca, niewydolność przeszczepu serca

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