Characteristics and prognosis of patients with decompensated right ventricular failure during the course of pulmonary hypertension

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Abstract

Background: New therapies for pulmonary arterial hypertension have prolonged survival but simultaneously increased the number of hospital admissions because of decompensated right heart failure (DRHF). The optimal approach in DRHF has not been established yet.

Aim: Analysis of clinical course of DRHF in a group of patients with pulmonary hypertension treated in a single referral centre.

Methods: We retrospectively analysed 60 episodes of DRHF in 37 patients (29 females, mean age 44±17 years) with pulmonary arterial hypertension and chronic thromboembolic pulmonary hypertension admitted to our hospital between 2005 and 2007. We assessed the cause of decompensation, vital signs at admission, functional class and laboratory values. We classified all episodes into four haemodynamic profiles using the value of systolic blood pressure together with presence of peripheral perfusion abnormalities (profile cold vs. warm) and symptoms of venous congestion (profile wet vs. dry). Primary end-point was in-hospital mortality.

Results: The most common causes of DRHF were infection (27%), drug noncompliance (20%), and pulmonary embolism (3%). In 48% no causative factor was indentified. There were 19 (32%) in-hospital deaths. The highest mortality was observed among patients with connective tissue disease (61%). The haemodynamic profile 'warm-wet' was the most common (48%) and the profile 'cold-dry' was the rarest but was associated with a 100% mortality. Patients who died had higher value of functional class (3.84±0.38 vs. 3.51±0.55, p=0.01) and higher activity of aspartate transaminase (61±61 vs. 42±78 U/l, p=0.02) compared with those who survived. In multivariate analysis higher dopamine dose (RR 2.0/1 μg/kg/min, 95% CI 1.00-5.00, p <0.001) was an independent factor of in-hospital death. In contrast 'rescue therapy' with iloprost or treprostinil decreased mortality (RR 0.09, 95% CI 0.01-0.99, p=0.04). Mortality in patients receiving dopamine was higher (60 vs. 18%, p=0.001) than in patients treated without dopamine.

Conclusion: Mortality in patients with pulmonary hypertension and DRHF remains very high and seems to be related to haemodynamic profile on admission. The newly introduced therapy with parenteral prostanoids may be more beneficial than dopamine infusion.

Key words: pulmonary hypertension, heart failure, right ventricle, mortality, treatment, hospitalisation

Kardiol Pol 2008; 66: 1033-1039

Introduction

Therapy of chronic right ventricular failure induced by increased right ventricular afterload and pulmonary hypertension (PH) involves the use of methods aimed at diminishing pulmonary vascular resistance and simultaneous treatment of the related symptoms with diuretics, oxygen and digitalis glycosides [1]. The introduction to the therapy of pulmonary hypertension of such drugs as prostanoids, endothelin receptor antagonists and sildenafil has changed the disease considered as untreatable and leading in a short period to death to chronic disease however still associated with poor prognosis. The number of chronic patients is increasing, and they are relatively stable in terms of the pulmonary circulation haemodynamic parameters, although at the same time at risk for deterioration decompensated right heart failure (DRHF) [2]. Optimal management in right ventricular HF is not so well established as treatment of left ventricular HF [3].

A lack of standard and commonly accepted management encouraged us to make a retrospective analysis of the clinical

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Received: 02 July 2008. **Accepted:** 03 September 2008.

course of DRHF in a group of patients with PH treated in a single referral centre.

Methods

In the years 2005-2007, 172 patients with pulmonary arterial hypertension (PAH) (n=140) and severe chronic thrombo-embolic PH CTEPH (n=32) with distal thrombosis were treated the Department of Chest Medicine. Within this period, there were overall 60 episodes of DRHF that required hospital admission and treatment in 37 patients (29 females and 8 males, at the age 18 to 74 years, mean 44±17 years).

In the examined group in 17 patients, diagnosis of idiopathic PAH was established, in 10 cases PAH was associated with systemic connective tissue disease and in 6 individuals with congenital heart malformation. In 2 cases thromboembolic PH not suitable for surgery was diagnosed, in 2 patients pulmonary veno-occlusive disease was detected, and in 1 case PAH was associated with porto-pulmonary shunt (porto-pulmonary PH). In all patients diagnosis of PH was confirmed by the haemodynamic examination and the following haemodynamic parameters were calculated: mean pulmonary artery pressure – 55.0±13.8 mmHg, cardiac output – 4.40±1.41 l/min, pulmonary vascular resistance – 12.2±5.4. Wood units and mean right atrial pressure – 9.9±4.1 mmHg.

In the stable period of disease, patients received optimal standard medical therapy that involved appropriate anticoagulation, diuretics, digitalis glycosides, oxygen therapy and chronic treatment with sildenafil (n=10), bosentan (n=2), sitaxentan (n=22), treprostinil (n=8), and iloprost (n=1). Seventeen percent of patients were treated with more than one drug.

Retrospective analysis included the cause of DRHF, vital parameters evaluated at admission, functional class according to NYHA classification and haemodynamic profile assessed on the basis of the clinical status. Patients were then allocated to the haemodynamic profile on the basis of systolic arterial pressure (RRsys <100 mmHg) accompanied by disturbances in the peripheral perfusion (cold vs. warm profile) and presence of lower extremities oedema, ascites, and transudates to the cavities (wet vs. dry profile). In-hospital mortality was the end-point of our analysis. The results of biochemical examinations such as sodium and creatinine concentration as well as aminotransferase activity at the time of admission to hospital were collected. Creatinine clearance was calculated using the Cockroft-Gault formula. Oxygen as well as carbon dioxide arterial pressures were measured in the arterialised capillary blood. Serum troponin T concentration was assessed by means of the high sensitivity III generation test (ECLIA, Roche Diagnostics) with detection threshold of 0.01 µg/l. According to data provided by the test manufacturer concentration of >0.01 µg/l is considered as the detection threshold of troponin T in human serum. The measurements of NT-proBNP concentration at admission were carried out with the electrochemiluminescent test (Elecsys proBNP, Roche Diagnostics).

During hospitalisation patients were treated using standard methods. In 20 episodes (33%) an infusion with dopamine was initiated at doses of 1.2 to 9.5 µg/kg/min due to hypotonia or a drop in daily diuresis and according to accepted indications. Only in 5 cases did the maximal dose exceed 3 µg/kg/min. In 10 cases so-called 'rescue treatment' was employed that involved short-term administration of drugs decreasing pulmonary vascular resistance such as iloprost (Ventavis) in the inhalations (n=2) and treprostinil (Remodulin) subcutaneously or intravenously (n=8).

Statistical analysis

Statistical analysis was performed using the statistical software package Statistica 6.1. A comparison of variables with normal distribution was done using Student's t-test. Continuous variables with abnormal distribution were compared by means of Mann-Whitney U test. Qualitative variables were compared using the χ^2 test with Yates' correction. Univariate followed by multivariate logistic regression analyses were carried out to find the factors independently associated with patient death resulting from clinical deterioration. The results are expressed as the means ± standard deviation or as relative risk (RR) with 95% confidential interval. Values of $p \leq 0.05$ were considered statistically significant.

Results

Out of 60 detected episodes of DRHF the predominant cause was infection (27%), drug and diet noncompliance (20%) and pulmonary embolism (3%). In 8% of cases, there were 2 or more causes of DRHF. In the group of infection-induced DRHP pneumonia was noted in 6 cases, inflammation of the upper respiratory tract in 4, gastro-intestinal one in 3, and skin or subcutaneous infection in 3 other subjects. In 48% of the analysed episodes the cause of DRHF was not established so we assumed that it resulted from a natural progression of the disease.

Nineteen episodes (32%) of DRHF were fatal during hospitalisation. An analysis of the correlation between mortality and PH aetiology revealed higher mortality rate in the group of patients with systemic connective tissue diseases compared to patients with either Eisenmenger syndrome or idiopathic PAH – 61, 50 and 18%, respectively (p=0.01). When analysing the relation between mortality and DRHF we noted that the death rate during episodes with an established causative factor was 32% (10 fatal cases of 31 episodes) whereas in the group of infection-related deteriorations – 50% (8 deaths during 16 episodes). In the group without an evident causative factor the mortality rate was 31% (9 deaths from 29 episodes).

The most commonly observed haemodynamic profile was 'warm-wet' (48%), the rarest one was the 'cold-dry'

Table I. In-hospital mortality in relation to the haemodynamic profile evaluated at admission to hospital

Haemodynamic profile	Prevalence [n] (%)	Deaths [n]	Mortality [% within group]	Mortality [% of all]
Cold-dry	4 (7)	4	100*	7
Warm-dry	9 (15)	2	22*	3
Cold-wet	18 (30)	4	22	7
Warm-wet	29 (48)	9	31	15

** χ^2 test; p=0.02*

Table II. Comparison of clinical and biochemical parameters between survivors and non-survivors

Parameter	Non-survivors n=19	Survivors n=41	p
Age [years]	47±18	43±16	0.40
Heart rate	98±15	91±13	0.06
Systolic blood pressure	97±13	103±13	0.06
Diastolic blood pressure	68±13	68±9	0.80
Pulse pressure [%]	29±14	34±6	0.11
Mean functional NYHA class before deterioration	2.84±0.50	2.68±0.52	0.27
Functional NYHA class before deterioration	II – 4 (21%) III – 14 (74%) IV – 1 (5%)	II – 14 (34%) III – 26 (64%) IV – 1 (2%)	0.52
Mean functional NYHA class during deterioration	3.84±0.38	3.51±0.55	0.02
Functional NYHA class during deterioration	II – 0 III – 3 (16%) IV –16 (84%)	II – 1 (2%) III – 18 (44%) IV – 22 (54%)	0.05
Body weight gain in relation to the period prior to hospitalisation [kg]	2.01±3.39	3.25±4.14	0.28
Na^+ [mmol/l]	133.3±6.7	135.8±5.9	0.15
Creatinine clearance [ml/min]	67±34	76±36	0.38
AspAT [U/l]	61±61	42±78	0.02
AlAT [U/l]	35±32	25±20	0.24
NT-proBNP	8791±6591	6907±5276	0.30
Troponin T concentration >0.01 ng/ml	9 (47%)	21 (51%)	0.78
Troponin T concentration [ng/ml]	0.048±0.071	0.031±0.034	0.36
pO_2 [mmHg]	53±12	57±15	0.39
pCO_2 [mmHg]	25±6	27±4	0.23
pH	7.48±0.06	7.49±0.03	0.28

profile (7%, p=0.03). Mortality reached 100% in the 'cold-dry' profile and was significantly higher than in the other profiles. The prevalence of the type of haemodynamic profiles and related mortality are outlined in Table I.

Clinical indices and the results of laboratory examinations at the time of admission were compared between the group of patients who died during DRHF and those who survived (Table II). Patients who died were found in higher functional NYHA class at the time of clinical deterioration and had higher serum aspartate aminotransferase activity. A tendency towards lower systolic arterial pressure and higher heart rate in the group of fatal DRHF events was observed. No differences in the NT-proBNP, troponin T, serum sodium concentrations, renal failure and gas analysis parameters were noted. Troponin T concentration in 30 (50%) observed episodes was elevated above the threshold of the ultra-sensitive test detection, although it exceeded only the cut-off value of 0.1 µg/l in two cases (3%).

Univariate logistic regression analysis (Table III) identified such variables as higher function class, a need to treat with intravenous furosemide and continuous infusions with dopamine as independent factors increasing

mortality rate in the examined group. Moreover, it was shown that mortality in DRHF increased gradually together with higher doses of dopamine infusion. In the multivariate analysis (Table IV) the dose of dopamine and emergency treatment with drugs decreasing pulmonary resistance were independent factors with a significant impact on outcome prognosis. Mortality increased together with dopamine dose rise, while rescue treatment reduced the number of fatal cases.

An additional analysis concerning episodes of DRHF treated with or without dopamine was carried out (Table V). In the group receiving dopamine infusion higher mean functional class and higher percentage of patients found in NYHA class IV were observed. Moreover, baseline heart rate was increased while systolic arterial pressure decreased. Higher aspartate aminotransferase activity, NT-proBNP concentration and lower partial carbon dioxide pressure (pCO_2) were noted in the dopamine-treated group. Patients who required dopamine infusion were more frequently found in the 'cold' haemodynamic profile and more often required intravenous furosemide administration and rescue treatment. Mortality in the group of patients with dobutamine infusion was three times higher than in the other cases (p=0.001).

Discussion

In this analysis, a high mortality rate associated with DRHF during the course of PH was found. Thirty-two percent of DRHF episodes were fatal, which is markedly higher than in decompensation of left ventricular PH. In the group of 150 patients described by Roguin et al. [4] admitted to the internal ward with acute pulmonary oedema, mortality was 12%. On the other hand, in the Euro Heart Survey 13.5% of patients hospitalised due to suspicion or established HF died during 12-week follow-up [5]. In another registry, OPTIMIZE-HF, which involved 48 612 patients admitted to hospital with HF, in-hospital mortality was approximately 3.8% [6]. In the analysis of the first 100 thousand patients with decompensated left ventricular failure recruited to the ADHERE registry in-hospital mortality was 4% [7].

An important fact is that in approximately 50% of cases of DRHF the cause remained not established. This indicates that PAH is a progressive disease and administered drugs only modify its course but without complete cure [8]. A clinical manifestation of DRHF may involve peripheral oedema, hepato-splenomegaly, ascites and also hypotonia and systemic hypoperfusion resulting from low pulmonary flow and in consequence from low cardiac index. Combined evaluation of venous congestion symptoms and peripheral perfusion provides 4 variants of the haemodynamic profile, as in left ventricular heart failure [9]. An assessment of the haemodynamic profile of left ventricular heart failure has become a routine step in the primary patient evaluation and has a significant impact on the planning of further management [10]. In this study for the first time the concept of haemodynamic profile evaluation to diagnose the form of right ventricular heart failure was used retrospectively and differences with respect to in-hospital mortality related to

Table III. Impact of clinical markers evaluated at admission to hospital on the mortality rate during an episode of right ventricular heart failure deterioration assessed by means of univariate logistic regression method

	Relative risk of death	95% CI	p
Functional NYHA class during deterioration	4.50	1.12-18.00	0.03
Systolic blood pressure [/10 mmHg]	0.64	0.40-1.04	0.07
NT-proBNP [/$\log_{10}$]	2.55	0.50-13.00	0.25
Detected serum TnT	0.86	0.28-2.61	0.78
Cold vs. warm haemodynamic profile	1.40	0.45-4.39	0.55
Dry vs. wet haemodynamic profile	2.24	0.62-8.15	0.22
Need for dopamine administration	7.07	2.05-24.35	0.02
Dopamine dose [/1 µg/kg/min]	2.3	1.40-4.00	<0.001
Treatment decreasing pulmonary resistance	0.20	0.02-1.77	0.14
Therapy with intravenous furosemide	3.57	0.98-12.95	0.05

Table IV. Multivariate logistic regression analysis

	Relative risk of death	95% CI	p
Functional NYHA class during deterioration	4.28	0.80-22.74	0.08
Systolic blood pressure [/10 mmHg]	1.23	0.65-2.35	0.51
Dopamine dose [/1 µg/kg/min]	2.00	1.00-5.00	<0.001
Therapy with drugs decreasing pulmonary resistance	0.09	0.01-0.99	0.04

χ^2 for model = 21.21, p <0.0003

Table V. Comparison of clinical and biochemical parameters between patients who were treated with dopamine (dopamine +) or who were not receiving dopamine (dopamine –)

Parameter	Dopamine (+) n=20	Dopamine (–) n=40	P
Mean functional NYHA class	3.90±0.52	3.47±0.52	0.002
Functional NYHA class during deterioration			
II	0	1 (2%)	
III	2 (10%)	19 (48%)	
IV	18 (90%)	20 (50%)	0.005
Heart rate [/min]	102±13	89±13	<0.001
Systolic blood pressure [mmHg]	94±14	105±11	0.001
Diastolic blood pressure [mmHg]	65±12	69±9	0.12
Pulse pressure [%]	30±14	34±7	0.16
Na^+ [mmol/l]	131±6	137±5	0.007
AspAT [U/l]	86±119	29±12	0.004
AlAT [U/l]	39±33	23±17	0.02
NT-proBNP during deterioration [pg/ml]	9733±4767	6312±5889	0.004
Detected TnT (+) [>0.01 ng/ml]	11 (55%)	19 (48%)	0.78
PO_2 [mmHg]	52±12	57±15	0.19
pCO_2 [mmHg]	24±4	28±5	0.004
pH	7.48±0.06	7.48±0.04	0.63
Therapy with intravenous furosemide	17 (85%)	19 (48%)	0.003
Therapy with drugs decreasing pulmonary resistance	4 (20%)	6 (15%)	0.44
Cold profile	13 (65%)	9 (23%)	0.003
Death	12 (60%)	7 (18%)	0.001

admission haemodynamic profile were confirmed. It reveals variability of the clinical forms of DRHF and also encourages us to search for various therapeutic options in relation to the haemodynamic status. It seems that particularly patients manifesting haemodynamic disturbances of the 'cold-wet' profile should receive treatment focused on a rapid drop in pulmonary vascular resistance.

Functional, haemodynamic, echocardiographic and biochemical indices are considered as the factors of accepted predictive value with respect to PAH [11, 12]. They seem to be reliable to estimate patient prognosis if they are evaluated in the stable phase of the disease. However, in the case of deterioration, particularly caused by a reversible causative factor, their prognostic value may fall [13]. The factors with survival prediction power that could be evaluated during DRHF are less known. In a group of 31 patients hospitalised in a French referral centre due to deterioration of right ventricular heart failure that required admission to the Intensive Care Unit or administration of continuous catecholamines infusion, in-hospital mortality was 36% [14]. Prior to hospital admission the group of patients who survived did not differ from the patients who died with respect to functional NYHA class, haemodynamic indices and administered drugs. Among indices assessed at admission, a low mean arterial blood pressure, elevated BNP and C-reactive protein concentrations as well as decreased serum sodium concentration were found in the group of patients who died. A role of systolic blood pressure was also indicated in the analysis of OPTIMIZE-HF registry data. It showed that patients with systolic blood pressure <105 mmHg had a two-fold higher mortality rate than patients with higher arterial blood pressure [6]. In our study systolic blood pressure did not significanty influence the outcome. Moreover, the importance of hyponatraemia was not confirmed. Previously, it was observed by Forfia et al. in a group of 40 patients that hyponatraemia correlated strongly with heart failure deterioration and it increased by more than ten-fold the risk of death [15].

In the multivariate analysis a higher dose of administered dopamine was shown to be an independent predictor of unfavourable outcome, contrary to so-called rescue therapy, which was associated with lower mortality rate. However, due to methodological limitations it is difficult to estimate the adverse impact of dopamine use on patient mortality. We showed that the group of patients requiring treatment with dopamine had more severe baseline clinical as well as haemodynamic status. Our study justifies the assumption that dopamine is not an effective life-saving option in cases of DRHF. It seems that emergency treatment aimed at rapid and at least partial decrease in pulmonary vascular resistance

may be beneficial rather than utilisation of the routinely used positive-inotropic drugs. Efficacy of rescue therapy with inhaled iloprost (Ventavis, Bayer-Schering AG) was previously documented in a group of patients undergoing atrial septostomy [16]. Analogical conclusions regarding the advantages of therapy decreasing afterload over positive inotropic drugs in patients with decompensated left ventricular heart failure were drawn from the analysis of the ADHERE registry data [17]. In a group of over 15 thousand patients administration of nesiritide and nitroglycerin decreased mortality while treatment with dobutamine and milrinone increased risk of death during hospitalisation.

Study limitations

During the study, the commercial availability in Poland of many drugs currently used in the therapy of PAH was limited. This involved drugs used either in the chronic treatment before hospitalisation or for acute therapy. We did not have access to epoprostenol, which according to recommendations is a drug of choice in the treatment of patients found in NYHA functional class IV [1, 18]. The small number of patients, retrospective nature of the study and non-standard management during hospitalisation did not justify the drawing of firm conclusions. However, all patients were treated by the same team according to the best knowledge, in circumstances of similar availability of drugs within the analysed period of time.

Conclusion

Mortality associated with DRHF in patients with pulmonary hypertension is high compared with medically treated deterioration in cases of left ventricular heart failure. Risk stratification on the basis of haemodynamic profile evaluated at the time of admission may be of importance concerning management in DRHF. Clinical usefulness of inotropic drugs and rescue treatment to decrease pulmonary vascular resistance needs to be determined in prospective randomised trials.

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Charakterystyka i rokowanie chorych z dekompensacją prawokomorowej niewydolności serca w przebiegu nadciśnienia płucnego

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Streszczenie

Wstęp: Poprawa skuteczności farmakoterapii tętniczego nadciśnienia płucnego (TNP) powoduje wydłużenie czasu przeżycia chorych i sprawia, że wzrasta liczba hospitalizacji z powodu dekompensacji prawokomorowej niewydolności serca (DPNS). Optymalne postępowanie w DPNS nie zostało dotychczas określone.

Cel: Analiza przebiegu klinicznego zaostrzeń DPNS w grupie chorych z nadciśnieniem płucnym leczonych w ośrodku referencyjnym.

Metodyka: Analizie poddano 60 epizodów DPNS u 37 chorych (29 kobiet, średni wiek 44±17 lat) z TNP o różnej etiologii oraz NP zakrzepowo-zatorowym wymagających leczenia w warunkach szpitalnych w latach 2005–2007. Retrospektywnej analizie poddano przyczynę dekompensacji, parametry życiowe mierzone w chwili przyjęcia, klasę czynnościową wg NYHA, wskaźniki biochemiczne. Chorych kwalifikowano do profili hemodynamicznych na podstawie wartości ciśnienia tętniczego (RRsk <100 mmHg), obecności zaburzeń perfuzji obwodowej (profil „zimny" *vs* „ciepły") oraz objawów retencji płynów (profil „mokry" *vs* „suchy"). Punktem końcowym była śmiertelność wewnątrzszpitalna.

Wyniki: Najczęstszymi przyczynami dekompensacji prawokomorowej były infekcja (27%), nieprzyjmowanie leków (20%) i zatorowość płucna (3%). W 48% nie stwierdzono uchwytnej przyczyny. Dziewiętnaście (32%) epizodów zakończyło się zgonem w okresie hospitalizacji. Stwierdzono wyższą śmiertelność w grupie chorych z układowymi chorobami tkanki łącznej w porównaniu z chorymi z zespołem Eisenmengera oraz idiopatycznym TNP (odpowiednio 61, 50, 18%, p=0,01). Najczęstszym profilem hemodynamicznym był „ciepły-mokry" (48%), najrzadszym „zimny-suchy" (6,7%, p=0,03), który we wszystkich 4 przypadkach zakończył się zgonem. Chorych zmarłych w trakcie DPNS w porównaniu z chorymi, którzy przeżyli, charakteryzowała wyższa wartość klasy czynnościowej w czasie zaostrzenia (3,84±0,38 *vs* 3,51±0,55, p=0,01) i wyższa aktywność transaminazy asparaginianowej (61±61 *vs* 42±78 U/l, p=0,02). Nie stwierdzono różnic w stężeniu sodu w surowicy oraz NT-proBNP. W analizie wieloczynnikowej dawka stosowanej dopaminy była niezależnym czynnikiem złego rokowania (RR 2,0/1 µg/kg/min, 95% CI 1,00–5,00, p <0,001). Leczenie ratunkowe, czyli włączenie w trakcie hospitalizacji dodatkowego leczenia analogami prostacykliny, zmniejszało natomiast śmiertelność (RR 0,09, 95% CI 0,01–0,99, p=0,04). Liczba zgonów w grupie otrzymującej dopaminę była większa niż w pozostałych przypadkach (60 *vs* 18%, p=0,001).

Wnioski: Śmiertelność w DPNS u chorych z NP jest wysoka i wydaje się zależna od profilu hemodynamicznego. Leczenie ratunkowe, zmniejszające naczyniowy opór płucny, może mieć korzystniejszy wpływ na przeżycie niż stosowanie dopaminy.

Słowa kluczowe: nadciśnienie płucne, niewydolność serca, prawa komora, dekompensacja, śmiertelność, leczenie, hospitalizacja

Kardiol Pol 2008; 66: 1033-1039

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Praca wpłynęła: 02.07.2008. **Zaakceptowana do druku:** 03.09.2008.