Persistent high NTpro-BNP concentration as a negative prognostic factor in patients with decompensated heart failure

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Abstract

Introduction: Monitoring of natriuretic peptide concentration may be useful for the identification of high-risk patients presenting with decompensated chronic heart failure (CHF).

Aim: Assessment of the predicting value of a significant decrease (by ≥20% vs. baseline) of N-terminal proBNP (NTpro-BNP, ROCHE) concentration during hospitalisation in patients with decompensated CHF.

Methods: This study involved 54 patients admitted to our centre because of CHF decompensation. Concentration of NTpro-BNP was measured on admission and at discharge from hospital. Primary end-points of this study were overall mortality and mortality with a number of cardiovascular-related readmissions.

Results: Mean NTpro-BNP concentration on admission was 7435±10040 pg/ml and at the time of discharge from hospital – 4816±7822 pg/ml. In 31 (57%) patients a significant decrease (≥20% *vs* baseline value) in NTpro-BNP level (mean: –58%±21%) was noted, while in the remainder (23 patients; 43%) neither an increase nor a decrease in NTpro-BNP levels was observed (mean: +72%±132%) despite optimal treatment and stabilisation of the clinical status. The mean follow-up duration was 358±240 days. Cox analysis showed that the absence of significant NTpro-BNP level decrease was associated with an increased risk of death – RR: 3.69 (95% CI: 1.10–12.37; p=0.035) and was the single independent risk factor for readmission due to cardiovascular-related reasons and/or death – RR: 2.29 (95% CI: 1.20–4.35; p=0.01). In the group of 23 patients with an increase or decrease in NTpro-BNP concentration of more than or equal to 20%, the survival rate was 65% vs. 87% in the remainder (p=0.02).

Conclusions: The lack of a significant (≥20%) decrease of NTpro-BNP level during hospitalisation correlates with a higher mortality and rate of readmissions. NTpro-BNP level monitoring may be of clinical importance for risk stratification in patients hospitalised for decompensated CHF.

Key words: congestive heart failure, brain natriuretic peptide, prognosis

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Introduction

Patients with decompensated chronic heart failure (CHF) are a particular group requiring special attention due to high mortality and frequent readmissions [1, 2]. It is estimated that half of these patients die or are readmitted to hospital within 6 months following discharge. It is of paramount importance to identify the highest risk patients with a poor prognosis.

Despite advances in the treatment of cardiovascular disorders, the number of patients with CHF is higher and is constantly increasing. The results of epidemiological analyses show that around 1 to 3% of the adult population suffer from CHF [3]. Paradoxically, the development of new therapeutic methods, especially those used in patients with acute coronary syndromes, enable any disease to transit from the acute to the

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chronic phase and cause a higher incidence of late CHF. The ideal solution would be to find a simple, predicting parameter measured just prior to discharge from hospital that would also enable the identification of patients with the highest risk of readmission and death.

One of the biochemical parameters that may be useful is brain natriuretic peptide (type B natriuretic peptide, BNP), released from cardiomyocytes in response to pressure or volume overload of cardiac ventricles [4], promoting diuresis and natriuresis, alleviating the effects of renin-angiotensin-aldosterone system activation, regulating the capacity of vasculature, as well as controlling arterial pressure and inhibiting the sympathetic nervous system [5]. A number of published reports have documented the usefulness of BNP level measurement in the diagnosis of CHF [6-8] and currently it is also more often considered a predictive parameter in CHF [9-11]. Practical use of BNP levels to establish the prognosis after aggravation of the disease, i.e. CHF decompensation, is being investigated.

The aim of this study was to estimate the predictive value of a significant decrease (>20% *vs* baseline value) in the N-terminal fragment of pro BNP (NTpro-BNP) concentration during the treatment of patients with decompensated CHF.

Methods

The study involved 54 patients hospitalised for decompensated CHF. Heart failure decompensation was defined as: 1. clinical pulmonary oedema, 2. worsening of heart failure symptoms to NYHA IV or NYHA III/IV functional class in previously stable patients with pulmonary congestion demonstrated on chest X-ray, 3. significant hypotension (cardiogenic shock and/or ventricular arrhythmia accompanied by hypotension). Exclusion criteria were: recent acute myocardial ischaemia, left ventricular ejection fraction (LVEF) >45%, drug-induced hypotension and lack of patient's consent.

After admission to the hospital each patient underwent a physical examination and had routine laboratory tests performed. Standard drugs were administered and adjusted according to the patient's status.

Concentrations of NTpro-BNP were measured on admission before the initiation of appropriate treatment, and at discharge from hospital. NTpro-BNP and physiologically active BNP origin from cleavage of higher molecular weight precursor, proBNP [12]. NTpro-BNP concentrations were measured in our study because NTpro-BNP level correlated well with BNP level while being more stable and durable in serum, having a longer half-life and fewer circadian fluctuations [13]. NTpro-BNP level was measured in venous blood serum by means of a quantitative electrochemiluminescence test using the ELECSYS device and kits manufactured by ROCHE.

In all patients, standard resting echocardiography was performed using the Aloka 4000 system equipped with a 2.5-3.5 MHz probe. Dimensions of cardiac chambers, LVEF calculated from modified Simpson equation, severity of valvular disease and pulmonary artery systolic flow acceleration time were analysed. The grade of mitral regurgitation was estimated according to a semi-quantitative 4-grade scale. The presence of a backward flow through cardiac valves was visualised by means of colour Doppler in the following projections: 4- and 5-chamber apical, 2-chamber apical, and long-, and short-axis parasternal view.

This study involved 54 patients (45 men, mean age of 62±14 years) who are presented in detail in Table I. Patients were admitted to hospital for pulmonary oedema in 20 cases (37%), clinically significant hypotension (symptoms of cardiogenic shock onset and/or ventricular arrhythmias accompanied by hypotension) in 11 (20%) and a significant worsening of CHF symptoms, reaching the III/IV functional NYHA class, in 23 (43%). The origin of CHF was ischaemic in 34 (63%) patients and dilated nonischaemic cardiomyopathy in the remaining 20 (37%).

The following parameters were analysed as possible prognostic factors:

1. essential demographic and clinical data: age, gender, CHF aetiology (ischaemic *vs* nonischaemic), comorbidities (diabetes, renal failure)
2. clinical pattern of decompensation (one of 3 categories: pulmonary oedema, significant hypotension and worsening of symptoms to III/IV functional NYHA class.
3. heart rate and arterial blood pressure on admission
4. results of standard laboratory tests on admission (e.g. sodium, potassium, creatinine levels) and NTpro-BNP concentration on admission, at the time of discharge and admission – discharge difference (ΔBNP)
5. necessity for inotropic support
6. echocardiographic parameters: cardiac chambers dimensions, LVEF, significant valvular disease.

Statistical analysis

The results are expressed as a mean ± standard deviation. The number of patients as well as the percentage distribution in the studied population are presented for selected parameters. An analysis of variance (ANOVA) was used to compare groups and linear regression analysis to estimate the correlation between NTpro-BNP and clinical parameters.

Overall mortality and combined mortality and number of cardiovascular-related hospitalisations were the primary endpoint of this study.

A Cox proportional hazards regression model was used to identify the association between endpoint

Table I. Patients' clinical characteristics

	Patients with decompensated CHF, n=54
Age [years]	62±14 (22–90)
Gender (male/female)	45/9 (83%/17%)
CHF aetiology	
ischaemic heart disease	34
Dilated cardiomyopathy	20
Concomitant diseases	
Arterial hypertension	25 (46%)
Diabetes	18 (33%)
Atrial fibrillation	21 (39%)
Renal failure	8 (15%)
COPD	3 (6%)
Indications for admission	
Marked CHF symptoms worsening	23 (43%)
Pulmonary oedema	20 (37%)
Clinically significant hypotension (shock or ventricular arrhythmia)	11 (20%)
Systolic arterial pressure on admission [mmHg]	121±29 (70–200)
Diastolic arterial pressure on admission [mmHg]	77±15 (40–110)
Heart rate on admission [beats/min]	99±27 (50–150)
Echocardiography	
LVEF [%]	28±8 (15–45)
LVEDD [mm]	67±10 (48–87)
Mitral regurgitation [% of patients]	14 (26%)
Aortic regurgitation [% of patients]	2 (4%)
Laboratory tests	
Creatinine [mg/do]	1.2±0.4 (0.7–3.7)
Sodium [mEq/L]	140±6 (120–147)
Potassium [mEq/L]	4.6±0.7 (3.3–6.1)
Haemoglobin [g/do]	13.9±1.6 (9.3–16.9)
NTpro-BNP on admission [pg/ml]	7435±10040 (601–51 800)
NTpro-BNP at discharge [pg/ml]	4816±7822 (329–47 000)
ΔBNP [pg/ml]	2619±6835
ΔBNP [%]	–3±108

Abbreviations: CHF – chronic heart failure, COPD – chronic obstructive pulmonary disease, LVEF – left ventricular ejection fraction, LVEDD – left ventricular end-diastolic dimension

events and the aforementioned clinical parameters. Uni- and multivariate analysis was conducted including in the analysis the time of endpoint events. Finally, the effect of significant negative risk factors on survival was evaluated using Kaplan-Meier curves.

The study protocol was accepted by the Bioethical Committee of Wrocław Medical University as approval No. KB-763/2003.

Results

NTpro-BNP concentration on admission and clinical characteristics

The highest NTpro-BNP concentrations were observed in patients admitted to hospital for clinically substantial hypotension (shock or ventricular arrhythmia accompanied by hypotension) and slightly lower in patients hospitalised for pulmonary oedema (14 559±16 007 pg/ml *vs* 7460±8882 pg/ml; p=0.10). Patients admitted to hospital because of worsening CHF had lower NTpro-BNP concentrations in comparison with the aforementioned groups (4006±4534 pg/ml; p <0.05).

The NTpro-BNP concentration on admission to hospital:

– did not correlate with LVEF or left ventricular dimension (r <0.2; p >0.2);
– correlated with heart rate and systolic arterial blood pressure (r=0.33 and r=–0.28, respectively; p <0.05);
– correlated with sodium concentration (r=–0.40; p=0.003).

Patterns of NTpro-BNP changes during hospitalisation

The mean NTpro-BNP concentration on admission was 7435±10040 pg/ml, at discharge 4816±7822, the mean admission – discharge difference (ΔBNP) was

Table II. Clinical characteristics of patients with or wthout significant decrease in NT-pro-BNP concentration

	Patients with significant decrease of NTpro-BNP concentration (n=31)	Patients without significant decrease of NTpro-BNP concentration (n=23)	p
Indication for hospitalisation			
Pulmonary oedema	13 (42%)	7 (30%)	NS
Significant hypotension	6 (19%)	5 (22%)	NS
CHF worsening	12 (39%)	11 (48%)	NS
Age [years]	61±12	65±16	NS
ischaemic aetiology	20 (65%)	14 (61%)	NS
LVEF [%]	27±6	29±9	NS
LVEDD [mm]	68±6	66±12	NS
RVEDD [mm]	26±6	26±8	NS
LA [mm]	49±7	48±6	NS
Heart rate on admission	97±26	102±28	NS
Systolic arterial blood pressure	123±28	117±29	NS
Creatinine [mEq/L]	1.2±0.3	1.3±0.6	NS
Sodium [mEq/L]	140±5	139±6	NS
NTpro-BNP on admission [pg/ml]	8052±7872	6604±12 532	NS
Atrial fibrillation	45%	30%	NS

Abbreviations: RVEDD – right ventricular end-diastolic diameter, LA – left atrial diameter. Rest of abbreviations as in Table I

2619±6835 pg/ml and the mean admission – discharge difference expressed as a percentage (ΔBNP%) was 3±108%.

In 31 (57%) patients (57%) a significant (≥20% *vs* baseline) decrease in the NTpro-BNP level (mean: –58±21%) was noted, while in the remaining 23 (43%) no drop or increase in the NTpro-BNP concentration in spite of appropriate treatment and clinical stabilisation (mean 72±132%) was seen.

Clinical characteristics of patients with NTpro-BNP decrease

Patients with a significant decrease of the NTpro-BNP level during treatment did not differ significantly from patients with increased values. No differences between examined groups regarding essential clinical and echocardiographic as well as laboratory parameters were observed (Table II).

Predictive analysis

During the follow-up (mean 358±240 days) 12 (22%) patients died after 37±122 days. Among 42 (78%) survivors, 28 (52%) were readmitted to hospital 1 to 6 times (mean 2.1±1.5 times) for cardiovascular-related complications, 147±195 days after discharge.

Univariate Cox analysis revealed that among analysed parameters no significant decrease of the NTpro-BNP concentration [RR: 3.69 (95% CI: 1.10-12.37; p=0.035)] and lower sodium level (hyponatremia ≤138 mEq/L) on admission [RR: 3.29 (95% CI: 1.04–10.40; p=0.04)] were associated with a higher risk of death. Both parameters were the independent risk factors of mortality (p <0.05). The only factor indicating a higher risk of readmission to hospital for cardiovascular-related reasons combined with mortality was the lack of a significant decrease in the NTpro-BNP concentration (≥20% *vs* baseline) – RR 2.29 (95% CI: 1.20–4.35; p=0.01).

A Kaplan-Meier analysis showed increased mortality in the group of 23 patients without a significant decrease in NTpro-BNP concentration (survival 65% *vs* 87% in this group and the remainder, p=0.02) (Figure 1A). The percentages of survivors and patients free of rehospitalisation during the follow-up (secondary endpoint) were also significantly lower in the group without lowering of the NTpro-BNP level in comparison with the rest of the patients (17% *vs* 39%; p=0.007) (Figure 1B).

Discussion

Patients with a history of CHF decompensation form a group at a high risk of recurrent cardiovascular events. Identification of the highest risk individuals is of paramount clinical importance. A number of essential clinical parameters (i.e. age, LVEF, systolic arterial pressure and heart rate at admission, diabetes mellitus,

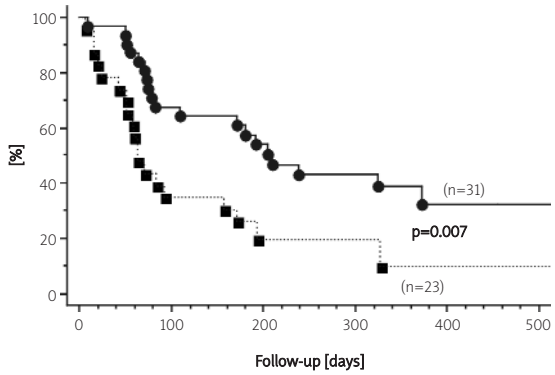

Figure 1a. Mortality rates in patients with (solid line) or without (dotted line) NTpro-BNP decrease

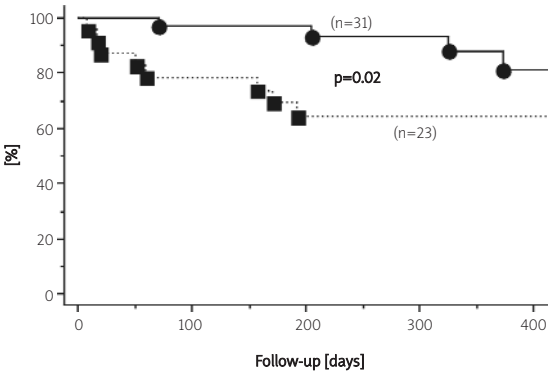

Figure 1b. Survival + no rehospitalisation rates in patients with (solid line) or without (dotted line) NTpro-BNP decrease

atrial fibrillation, creatinine concentration) presented herein failed to select the group of patients at a high risk of death and/or readmission.

We documented, however, that no significant NTpro-BNP concentration reduction during hospitalisation despite optimal and complete treatment qualifies the patient to the group with a higher risk of death and readmission. Another variable associated with higher mortality was hyponatremia on admission. Such a high predictive value of lower sodium concentration seems to be a reflection of the processes associated with water and electrolyte balance including impaired renal perfusion and is also an indicator of severe neurohormone activation.

The dynamics of NTpro-BNP concentration changes and their consequences presented in this study require more comments. So far only a few prospective studies analysing this problem have been published. Cheng et al. measured BNP concentrations in patients with acute CHF on admission to hospital and then daily during hospitalisation. They found that a decrease of BNP (mean, 215 pg/ml) in response to the treatment was associated with a more favourable prognosis while no drop or increase in BNP concentration (mean, 233 pg/ml) was noted in patients with significantly higher mortality and the necessity for readmission within 30 days after discharge [14]. Gackowski et al. demonstrated that a relative decrease of plasma BNP by more than 10% after the first day of decompensated CHF treatment or BNP concentration below 300 pg/ml at day 7 correlated with a lower incidence of adverse events (death, cardiac arrest, readmission, heart transplantation) during the 60-day follow-up [15]. It must be emphasised that BNP measurements in that study were more predictive than clinical signs and symptoms or the results of echocardiographic examination. Ischii et al. showed that increased BNP values exceeding 440 pg/ml (accompanied by higher troponin I) on admission due to worsening CHF correlated with an incremental increase in mortality and cardiac event rate either in hospital or during the following several months [16]. Similar conclusions were drawn by Yu et al., who noted a significantly higher annual mortality among patients after an acute CHF event with elevated BNP concentration during in-hospital stay [17].

The results of the above-mentioned studies justify more common use of BNP measurements in patients with decompensated CHF in order to identify the highest risk patients so that they may receive more individual and complex treatment, for example in specialist outpatient clinics, or detailed patient education. Currently it is difficult to establish the exact magnitude of BNP concentration reduction that would be clinically relevant. Similarly to others, we used an arbitrary level of 20% reduction vs. baseline. Undoubtedly, a more definitive recommendation of such a threshold to be used in clinical practice must be preceded by prospective clinical observational studies involving more patients and designed for appropriate data analysis, for example using the ROC (*receiver-operator-curve*) method which would help to define the level of NTpro-BNP decrease for the optimal identification of patients with the worst prognosis.

It has not been established so far how many BNP measurements should be made during hospitalisation to draw practical conclusions. In the first two referred reports, many examinations were carried out – in the first study on a daily basis, in the second one on admission, then after 24 h and at discharge. In the other two studies, only a single measurement on admission was performed. It seems, also based on our results, that an investigation of the dynamics of theses changes, i.e. at least two measurements, gives a more complete overview of the course of treatment and enables a more accurate clinical prognosis to be established. In contrast, Logeart et al. have indicated that the most predictive of all is the

predischarge BNP measurement, even more relevant than changes of concentrations during in-hospital stay [18]. On the other hand, Wu et al. concluded that the monitoring of BNP daily or every other day was not justified. They suggested that the minimum measurement interval should be 6 to 7 days [19].

An interesting summary for this discussion could be a manuscript including a clinical management algorithm adopted in the Cleveland Clinic [20]. In decompensated CHF, serial BNP concentration measurements are performed and on occasion even discharge from hospital is delayed only because of abnormal test results. Unfortunately, we must be aware of the relative high costs of natriuretic peptide measurements in our country which force us to reduce the frequency of sampling as much as possible.

The presented study is not prospective, but a retrospective analysis of patients with decompensated CHF in whom serial measurements of NTpro-BNP concentration were carried out during in-hospital stay in our centre. This is a preliminary report but the conclusions are interesting enough to encourage further and more extensive studies.

Conclusions

1. The lack of a significant (≥20%) decrease of the NTpro-BNP level during hospitalisation correlates with higher mortality and rate of readmissions.
2. NTpro-BNP level monitoring may be of clinical importance for risk stratification in patients hospitalised for decompensated CHF.

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Utrzymujące się wysokie stężenie NT-proBNP jest czynnikiem złego rokowania u chorych ze zdekompensowaną niewydolnością serca

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Streszczenie

Wstęp: Wśród chorych ze zdekompensowaną niewydolnością serca (NS) monitorowanie stężenia peptydów natriuretycznych może okazać się przydatne w selekcji grupy wysokiego ryzyka.

Cel pracy: Ocena wartości prognostycznej istotnego spadku (≥20% wartości wyjściowej) stężenia N-końcowego fragmentu proBNP (Nt-proBNP, ROCHE) w trakcie hospitalizacji w grupie chorych ze zdekompensowaną NS.

Metodyka: Do badania włączono 54 pacjentów, hospitalizowanych w naszym ośrodku z powodu dekompensacji NS. Stężenie Nt-proBNP oznaczano przy przyjęciu oraz przy wypisie chorego ze szpitala. Pierwotnymi punktami końcowymi była śmiertelność całkowita oraz śmiertelność wraz z liczbą hospitalizacji z przyczyn sercowo-naczyniowych.

Wyniki: Stężenie Nt-proBNP przy przyjęciu wynosiło średnio 7435±10040 pg/ml, przy wypisie 4816±7822. U 31 chorych (57%) obserwowano istotny spadek (o ≥20% wartości wyjściowej) wartości Nt-proBNP (średnio: -58%±21%), natomiast u pozostałych 23 chorych (43%) brak istotnego spadku lub wzrost wartości Nt-proBNP (średnio: +72%±132%) pomimo leczenia i stabilizacji klinicznej. Analiza Coxa wykazała, że brak istotnego spadku Nt-proBNP wiązał się z podwyższonym ryzykiem zgonu – RR: 3,69 (95%CI: 1,10–12,37; p=0,035) oraz był jedynym czynnikiem wskazującym na zwiększone ryzyko hospitalizacji z przyczyn sercowo-naczyniowych i zgonu (łącznie) – RR: 2,29 (95%CI: 1,20–4,35; p=0.01). W grupie 23 chorych, u których wystąpił wzrost lub spadek stężenia Nt-proBNP o ≥20% przeżycie wyniosło 65% *vs* 87% u pozostałych chorych (p=0,02).

Wnioski: Brak istotnego (≥20%) spadku stężenia Nt-proBNP podczas hospitalizacji wiąże się z częstszym występowaniem zgonów i ponownych hospitalizacji. Monitorowanie stężenia Nt-proBNP może mieć praktyczne zastosowanie w stratyfikacji ryzyka u chorych ze zdekompensowaną NS.

Słowa kluczowe: niewydolność serca, mózgowy peptyd natriuretyczny, rokowanie

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