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Thoracic fluid content as a useful diagnostic marker of resting dyspnoea in patients hospitalised due to acute heart failure

Zawartość płynu w klatce piersiowej jako przydatny marker diagnostyczny duszności spoczynkowej u pacjentów hospitalizowanych z powodu ostrej niewydolności serca

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Abstract

Background: Dyspnoea is one of the most common symptoms in the emergency department, and identification of its cause may require complex diagnostic tests. In everyday practice, some concerns arise when the reported dyspnoea is not fully compatible with objective measures in additional tests. This study was performed to evaluate which objective diagnostic markers differentiate patients admitted to hospital due to acute heart failure with and without resting dyspnoea, with a special emphasis on haemodynamic parameters measured by impedance cardiography. **Methods:** This study enrolled patients over 18 years of age who were hospitalised due to acute heart failure. The admission evaluation included haemodynamic profiling by ICG, with special emphasis on parameters characterising afterload (systemic vascular resistance index), cardiac function (cardiac index; stroke index), and congestion (thoracic fluid content). **Results:** The study population consisted of 102 patients, mostly men (76.5%), with a mean age of 71.4 ± 12.5 years and a left ventricle ejection fraction of 37.3 ± 14.1%. Patients with dyspnoea at rest (n = 41), in comparison with those without this symptom (n = 61), presented with poorer clinical states: more frequent orthopnoea (p = 0.002), tachypnoea (p = 0.001), palpitations (p = 0.004), and peripheral hypoperfusion (p = 0.0005), higher concentration of high-sensitivity troponin T (p = 0.021), and higher thoracic fluid content (p = 0.003). No significant differences were noted for haemoglobin, creatinine, N-terminal pro-brain natriuretic peptide, heart rate, blood pressure, chest X-ray, left ventricular ejection fraction, cardiac index, stroke index, or systemic vascular resistance index. **Conclusions:** Thoracic fluid content assessed by impedance cardiography was found to be a good diagnostic marker for differentiating patients admitted to hospital due to acute heart failure with and without resting dyspnoea, and was superior to chest X-rays, N-terminal pro-brain natriuretic peptide, and left ventricle ejection fraction.

Keywords: heart failure, hypervolaemia, dyspnoea, fluid overload, congestion

Streszczenie

Wprowadzenie i cel: Duszność należy do najczęstszych objawów występujących na oddziałach ratunkowych, a ustalenie jej przyczyny może wymagać skomplikowanych badań diagnostycznych. W codziennej praktyce nierzadko zdarza się, że zgłaszana duszność nie jest adekwatna do wyników badań dodatkowych. Celem pracy była ocena, które obiektywne markery diagnostyczne różnicują pacjentów przyjętych do szpitala z powodu ostrej niewydolności serca z dusznością spoczynkową i bez niej, ze szczególnym uwzględnieniem parametrów hemodynamicznych ocenianych kardiografią impedancyjną. **Materiał i metody:** Do badania włączono pacjentów w wieku powyżej 18 lat, hospitalizowanych z powodu ostrej niewydolności serca. Przy przyjęciu oceniano parametry laboratoryjne oraz hemodynamiczne za pomocą kardiografii impedancyjnej, ze szczególnym uwzględnieniem parametrów charakteryzujących obciążenie następcze, czynność serca (objętość wyrzutowa, rzut serca) i zastój w obrębie klatki piersiowej (zawartość płynu w klatce piersiowej). **Wyniki:** Grupę badaną stanowiło 102 pacjentów, w większości mężczyzn (76,5%); średni wiek 71,4 ± 12,5 roku, frakcja wyrzutowa lewej komory 37,3 ± 14,1%. Pacjenci z dusznością spoczynkową (n = 41) w porównaniu z pacjentami bez duszności w spoczynku (n = 61) prezentowali gorszy stan kliniczny: częstsze *orthopnoe* (p = 0,002), *tachypnoe* (p = 0,001), kołatanie serca (p = 0,004), hipoperfuzję obwodową (p = 0,0005), wyższe stężenie troponiny T o wysokiej czułości (p = 0,021) i wyższą zawartość płynu w klatce piersiowej (p = 0,003). Nie zaobserwowano istotnych różnic w zakresie stężenia hemoglobiny, kreatyniny, N-końcowego promózgowego peptydu natriuretycznego, częstości akcji serca, ciśnienia krwi, badania radiologicznego klatki piersiowej, frakcji wyrzutowej lewej komory, wskaźnika sercowego, wskaźnika udaru mózgu czy

wskaźnika systemowego oporu naczyniowego. **Wnioski:** Zawartość płynu w klatce piersiowej oceniana za pomocą kardiografii impedancyjnej była dobrym markerem diagnostycznym, pozwalającym różnicować pacjentów przyjętych do szpitala z powodu ostrej niewydolności serca z dusznością spoczynkową i bez niej i była skuteczniejsza niż zdjęcie rentgenowskie klatki piersiowej, N-końcowy peptyd natriuretyczny oraz frakcja wyrzutowa lewej komory.

Słowa kluczowe: niewydolność serca, hiperwolemia, duszność, przewodnienie, zastój

INTRODUCTION

Dyspnoea is one of the most frequently reported symptoms in the emergency department and can be caused by heart failure (HF), a common reason for hospitalisation in patients over 65 years of age[1,2]. The identification of the cause of dyspnoea may require complex diagnostic tests, and its severity does not always correspond to objective test results. For example, the widely used chest X-ray has been revealed by current reports to have limited value in HF patients, such that as many as 20% of patients with HF present with normal results[3,4]. The New York Heart Association (NYHA) classification is a well-established tool for assessing the severity of dyspnoea in patients with HF[5–7]. It is mainly based on the level of dyspnoea, but it also comprises nonspecific limitations in functionality in everyday life[8,9]. NYHA class is an independent predictor of mortality, especially in women[10–14]. In everyday practice, some concerns arise when the reported subjective symptoms are not fully compatible with objective measures in additional tests. Thus, the search for effective tools for assessing congestion is ongoing.

Accordingly, this study was performed to evaluate which objective clinical markers differentiate patients admitted to hospital due to acute heart failure (AHF) with and without resting dyspnoea, with a special emphasis on haemodynamic parameters measured by impedance cardiography.

METHODS

This paper presents the results of the secondary analysis of a prospective, observational study that enrolled 102 adult patients hospitalised at the Department of Cardiology and Internal Diseases of the Military Institute of Medicine – National Research Institute between November 2014 and March 2017 (NCT 02355769). Subjects admitted due to AHF (defined according to the European Society of Cardiology guidelines[6,7]) who required intravenous diuretic therapy were included. In brief, the exclusion criteria were as follows: unstable angina, history of acute coronary syndrome and/or coronary artery bypass grafting surgery within the last 12 weeks, non-cardiogenic shock, severe pulmonary hypertension or other severe lung condition, pulmonary embolism, poorly controlled hypertension, acute and/or decompensated non-cardiovascular disease, valvular disease or other acquired heart defects requiring surgical intervention, anaemia, end-stage chronic kidney disease, and neoplastic disease[15–17].

The study protocol was approved by the local bioethics committee (approval no. 14/WIM/2012), and all study participants provided their written informed consent. After enrolment, all patients were subjected to a detailed history and clinical examination with emphasis on the severity of dyspnoea and different types of dyspnoea. On admission, laboratory tests from peripheral venous blood samples were performed (serum creatinine, N-terminal pro-brain natriuretic peptide – NTproBNP, high-sensitivity troponin T – hsTnT, and haemoglobin). Throughout the hospital stay, the patients were treated according to the current guidelines.

To assess the hemodynamic profile, non-invasive impedance cardiography (ICG, Niccomo™ device [Medis, Germany]) was used. All ICG measurements were performed within 24 hours of admission in a sitting position and after 10 minutes of rest; these included assessments of heart rate (HR), blood pressure (BP), systemic vascular resistance index (SVRI), cardiac index (CI), stroke index (SI), and thoracic fluid content (TFC).

Echocardiography was conducted using Vivid S6 (GE-Healthcare, USA) and Vivid 7 (GE-Healthcare, USA) ultrasound systems. The standard assessment included left ventricular ejection fraction (LVEF). There was no defined time limit for echocardiography.

Statistical analysis

Statistical analyses were performed using STATISTICA 12.0 (StatSoft, Inc., Tulsa, USA). The distribution and normality of the data were assessed via visual inspection and the Kolmogorov–Smirnov test. Continuous variables were presented as means ± standard deviation (*SD*), and categorical variables were expressed as absolute and relative frequencies (percentages). For the purpose of the analysis, patients were categorised into two groups: patients with (DR[+]) and those without dyspnoea at rest (DR[−]). The two subgroups were compared in terms of clinical, laboratory, and hemodynamic parameters using the Student's *t*-test or Mann–Whitney *U* test for continuous variables and chi-squared or Fisher's exact test for categorical variables. A p-value of <0.05 was considered statistically significant.

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	Study group ***N*** **= 102**
Age, mean ± *SD*	71.4 ± 12.5
Male, *n* (%)	78 (76.5%)
HR [bpm], mean ± *SD*	87 ± 24
SBP [mm Hg], mean ± *SD*	135 ± 27
DBP [mm Hg], mean ± *SD*	82 ± 14
Symptoms and signs, ***n*** **(%)**	
Dyspnoea at rest	41 (40.2%)
Dyspnoea on effort	100 (98.1%)
Orthopnoea	78 (77.2%)
Paroxysmal nocturnal dyspnoea	44 (43.1%)
Chest pain	25 (24.5%)
Palpitations	33 (32.4%)
Oedema	77 (75.5%)
Tachypnoea	21 (20.6%)
Ascites	16 (15.7%)
Peripheral hypoperfusion	10 (9.8%)
Concomitant disease, ***n*** **(%)**	
Prior MI	42 (41.1%)
Hypertension	68 (66.6%)
Atrial fibrillation	54 (52.9%)
Diabetes mellitus	50 (49.0%)
Chronic obstructive pulmonary disease	15 (14.7%)
Chronic kidney disease (stage ≥3)	30 (29.4%)
Laboratory tests, mean ± *SD*	
NTproBNP [pg/mL]	6197 ± 7057
Creatinine [mg/dL]	1.31 ± 0.51
eGFR [mL/min/1,73 m^2]	62.2 ± 23.9
Haemoglobin [g/dL]	12.6 ± 2.6
Medication at admission	
ACEIs/ARBs	72 (70.6%)
MRAs	33 (32.4%)
Beta-blockers	78 (76.5%)
Diuretics	74 (72.5%)

ACEIs – angiotensin-converting enzyme inhibitors; **ARBs** – angiotensin receptor blockers; **DBP** – diastolic blood pressure; **eGFR** – estimated glomerular filtration rate; **HR** – heart rate; **MI** – myocardial infarction; **MRAs** – aldosterone receptor antagonists; **NTproBNP** – N-terminal pro-brain natriuretic peptide; **SBP** – systolic blood pressure; ***SD*** – standard deviation.

*Tab. 1. Admission characteristics of the study group (*N = *102)*

RESULTS

Study group admission characteristics

The study group was dominated by men (76.5%) and older subjects (mean age 72 years). Almost all patients presented dyspnoea on effort (98.1%), and 40.2% reported resting dyspnoea (Tab. 1). A total of 36 patients (35.3%) were classified as NYHA class IV. The mean LVEF was 37.3 ± 14.1%. The study group was burdened with a high prevalence of ischaemic heart disease, hypertension, atrial fibrillation, and diabetes mellitus. The rates of use of angiotensin-converting enzyme inhibitors/angiotensin receptor blockers (ACEIs or ARBs), aldosterone receptor antagonists (MRAs), beta-blockers, and diuretics were 70.6%, 32.4%, 76.5%, and 72.5%, respectively. The detailed demographics, clinical characteristics, and laboratory tests at admission are summarised in Tab. 1.

Comparison of subgroups regarding the presence of dyspnoea at rest

Patients with dyspnoea at rest (DR[+]), when compared to those without this symptom (DR[–]), presented at admission with poorer clinical states: more frequent orthopnoea ($p = 0.002$), tachypnoea ($p = 0.001$), palpitations ($p = 0.004$), and peripheral hypoperfusion ($p = 0.0005$). hsTnT concentrations were significantly higher in the DR[+] subgroup ($p = 0.021$). LVEF, NTpro-BNP, and chest X-ray findings were similar between both groups. Prior to admission, DR[+] patients received less ACEIs ($p = 0.043$) and diuretics ($p = 0.009$), which may be partly explained by the trend toward a higher prevalence of de novo HF ($p = 0.058$) (Tab. 2).

The differences in the hemodynamic profile manifested as a higher TFC in DR[+], both for absolute values ($p = 0.003$) and the significant cut-off for congestion ($p < 0.0001$) (Tab. 2). However, other ICG parameters, including markers of cardiac performance (SI and CI) and afterload (SVRI), were similar in both groups.

There was no significant intergroup difference in the length of hospital stay (DR[–] vs. DR[+]: 9.6 ± 5.8 vs. 10.2 ± 5.4 days; $p = 0.708$) or in-hospital total dose of intravenous (911 ± 1,258 vs. 883 ± 962 mg per hospitalisation; $p = 0.675$) and oral loop diuretics (363 ± 468 vs. 480 ± 454 mg per hospitalisation; $p = 0.093$).

DISCUSSION

Our study revealed that a high TFC distinguished patients admitted to the hospital with AHF and resting dyspnoea from those without this symptom, while most other diagnostic methods (including chest X-ray, NTproBNP concentration, and LVEF) did not. These findings are in agreement with reports on ICG as a useful method for differentiating the causes of dyspnoea in emergency settings[18,19] and predicting HF decompensation[20]. TFC is an especially good marker of (de)congestion[15,21–23]. We have now proven an additional diagnostic value of TFC in explaining the severity of symptoms.

The wide range of phenotypes of AHF[6] has inspired studies on diagnostic methods that support the objective assessment of patients. The complexity of the clinical presentation of AHF is reflected in some differences between the reported studies. For example, similarly to what Asano et al. reported, in our study, 40% of patients presented with resting dyspnoea[24].

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	DR[–] Mean ± *SD*; median (IQR)/*n* (%)	DR[+] Mean ± *SD*; median (IQR)/*n* (%)	*p*
Age [years]	72.2 ± 11.8; 73 (64–83)	70.2 ± 13.5; 72 (64–80)	0.436
De novo HF	12 (19.7%)	15 (36.6%)	**0.058**
Male	48 (78.7%)	30 (73.2%)	0.519
BMI [kg/m^2]	31.0 ± 6.1; 30.4 (26.6–33.6)	28.8 ± 6.9; 28.0 (24.5–30.9)	0.106
Symptoms and signs			
NYHA class			
Class 3	56 (91.8%)	10 (24.4%)	**<0.0001**
Class 4	5 (8.2%)	31 (75.6%)	
Dyspnoea at rest	0 (0%)	41 (100%)	NA
Dyspnoea on effort	61 (100%)	41 (100%)	NA
Orthopnoea	40 (65.6%)	38 (92.7%)	**0.002**
Paroxysmal nocturnal dyspnoea	24 (39.3%)	20 (48.8%)	0.345
Chest pain	11 (18.0%)	14 (34.2%)	0.063
Palpitations	13 (21.3%)	20 (48.8%)	**0.004**
Oedema	49 (80.3%)	31 (75.6%)	0.570
Tachypnoea	6 (9.8%)	15 (36.6%)	**0.001**
Peripheral hypoperfusion	0 (0%)	10 (24.4%)	**0.0005**
Concomitant disease (in anamnesis)			
Prior MI	23 (37.7%)	19 (46.3%)	0.385
Hypertension	43 (70.5%)	25 (61.0%)	0.318
Atrial fibrillation	35 (57.4%)	19 (46.3%)	0.274
Diabetes mellitus	30 (49.2%)	20 (48.8%)	0.984
Chronic obstructive pulmonary disease	7 (11.5%)	8 (19.5%)	0.261
Chronic kidney disease (stage ≥3)	18 (29.5%)	12 (29.3%)	0.937
Pharmacotherapy			
ACEIs	42 (70.0%)	20 (50.0%)	**0.043**
ARBs	4 (6.7%)	6 (15.0%)	0.173
Beta-blockers	50 (83.3%)	28 (70.0%)	0.115
MRAs	21 (35.0%)	12 (30.0%)	0.602
Diuretics	50 (83.3%)	24 (60.0%)	**0.009**
Laboratory tests			
Creatinine [mg/dL]	1.28 ± 0.4; 1.3 (0.9–1.5)	1.36 ± 0.63; 1.3 (1.0–1.5)	0.392
eGFR [mL/min/1,73 m^2]	62.1 ± 22.9; 57.3 (46.7–75.4)	62.4 ± 25.7; 59.1 (43.8–76.2)	0.943
NTproBNP [pg/mL]	5438 ± 6,768; 3,554 (1,344–6,346)	7,308 ± 7,404; 4751 (3,136–8,585)	0.522
High-sensitivity troponin T [ng/L]	48.7 ± 69.8; 32.6 (23.0–53.1)	151.9 ± 330.7; 40.8 (21.6–122.9)	**0.021**
Haemoglobin [g/dL]	12.7 ± 2.4; 12.6 (11.4–14.4)	12.5 ± 2.0; 12.6 (11.2–13.8)	0.737
Chest X-ray			
Pulmonary oedema	1 (1.7%)	3 (7.5%)	0.156
Congestion	45 (77.6%)	31 (77.5%)	0.992
Haemodynamics			
LVEF [%]	37.9 ± 15.0; 35 (26–51)	36.4 ± 12.8; 34 (28–41)	0.870
HR [bpm]	81 ± 22; 80 (64–90)	82 ± 21; 76 (67–88)	0.765
SBP [mm Hg]	124 ± 24; 120 (107–143)	121 ± 25; 119 (106-135)	0.694
DBP [mm Hg]	74 ± 12; 72 (67–81)	73 ± 11; 73 (65-81)	0.563
SI [mL*m^{-2})	41.2 ± 13.6; 40.7 (31.0–46.0)	38.1 ± 12.2; 41.0 (30.0-47.5)	0.566
CI [mL*m^{-2}*min^{-1}]	3.11 ± 0.90; 3.00 (2.60–3.45)	2.90 ± 0.78; 2.80 (2.40-3.50)	0.376
SVRI [dyn*s*cm^{-5}*m^2]	2,260 ± 728; 2,165 (1,715–2,603)	2,457 ± 807; 2,315 (1,995–2,988)	0.235
TFC [1*kΩ^{-1}]	33.8 ± 7.5; 32.9 (29.1–37.6)	38.5 ± 7.3; 39.0 (35.4–42.6)	**0.003**
TFC ≥35 [1*kΩ^{-1}]	18 (29.5%)	31 (75.6%)	**<0.0001**

ACEIs – angiotensin converting enzyme inhibitor; **ARBs** – angiotensin receptor blockers; **BMI** – body mass index; **CI** – cardiac index; **DBP** – diastolic blood pressure; **DR** – dyspnoea at rest; **eGFR** – estimated glomerular filtration rate; **HF** – heart failure; **HR** – heart rate; **IQR** – interquartile range; **LVEF** – left ventricle ejection fraction; **MI** – myocardial infarction; **MRAs** – aldosterone receptor antagonists; **NA** – not applicable; **NTproBNP** – N-terminal pro-brain natriuretic peptide; **NYHA** – New York Heart Association; **SBP** – systolic blood pressure; ***SD*** – standard deviation; **SI** – stroke index; **SVRI** – systemic vascular resistance index; **TFC** – thoracic fluid content.

Tab. 2. Comparison of subgroups regarding the presence of dyspnoea at rest

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However, we noted no difference in the distribution of oedema, concentration of natriuretic peptides, or creatinine depending on resting symptoms. Moreover, in contrast to the before mentioned study, in which patients with NYHA IV had significantly higher SBP and HR, we did not observe differences in SBP, DBP, and HR[24]. Some studies[25,26] have revealed that women can be more symptomatic, but we did not observe any difference in the prevalence of resting dyspnoea between the sexes. Conversely, in accordance with previous reports that patients with de novo HF had generally poorer clinical states (dyspnoea at rest, hypoperfusion), we observed more de novo HF patients in the DR[+] subgroup[27–29].

The measures of heart function (LVEF, SI, and CI) were also comparable. We did not note any significant intergroup differences in NTproBNP, but it should be emphasised that previous studies have shown that high levels of natriuretic peptides indicate not only pure outcomes[26], but also the severity of HF[15,27]. Our observations also confirmed the limited value of chest X-rays in the diagnosis of hypervolaemia[3,30,31].

We found that there was difficulty in objectively diagnosing patients with different levels of dyspnoea using daily basic tests[32]. Differentiating the cause of dyspnoea in HF patients with comorbidities is particularly difficult, so the application of ICG, especially TFC, may support the assessment of chest volume load. Moreover, from our previous reports, we can recommend TFC as a useful marker for monitoring the effects of in-hospital treatment[15].

STUDY LIMITATIONS

The main limitation of our study is the small size of the study group. Another important limitation is the discrepancy between the reported prevalence of rest dyspnoea and NYHA class IV. It should also be noted that even a short delay between admission and data on symptoms and signs may bias its relation to subsequent diagnostic parameters. This applies especially to the time point of echocardiography in our study (median of 3 days after admission), implying that most of those examinations took place after clinical stabilisation. Additionally, chest X-ray descriptions were analysed based on everyday standard protocols without quantified grading of congestion. Another limitation to consider is that it was an observational study conducted at a single centre.

CONCLUSIONS

TFC assessed by ICG is a good marker for differentiating patients admitted to hospital due to acute decompensation of chronic heart failure with and without rest dyspnoea, and it is superior to chest X-rays, NTproBNP, and LVEF. This simple and non-invasive technique can be used in dyspnoeic patients in different clinical settings, including emergency departments.

Conflict of interest

The authors do not report any financial or personal connections with other persons or organisations which might negatively affect the content of this publication and/or claim authorship rights to this publication.

Author contributions

Original concept of study: AG, PK. Collection, recording and/or compilation of data: AG, PK. Analysis and interpretation of data: AG, PK. Writing of manuscript: AG. Critical review of manuscript: PK, GG. Final approval of manuscript: AG, PK, GG.

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